

QUANTEC

Quantum Timber Finishes

Chemwatch Hazard Alert Code: 3

Chemwatch: 84-5172

Version No: 2.1.1.1

Safety Data Sheet according to WHS and ADG requirements

Issue Date: 22/08/2017

Print Date: 22/08/2017

L.GHS.AUS.EN

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

Product Identifier

Product name	QUANTEC
Synonyms	Product Code: 4590
Other means of identification	Not Available

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

Relevant identified uses	For full details on application and properties consult the technical datasheet. A deeply penetrative, natural oiled finish for hardwoods, especially decking. Exterior timber that has not been previously painted, use to protect the timber without changing the colour.
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Details of the supplier of the safety data sheet

Registered company name	Quantum Timber Finishes	Mirotone
Address	147 Ordish Road, Dandenong Vic 3175 Australia	21 Marigold Street Revesby NSW 2212 Australia
Telephone	+61 2 9795 3700	+61 2 9795 3700
Fax	1300 132 202	+61 2 9771 3601
Website	www.qtf.com.au	www.mirotone.com, www.polycure.com.au
Email	Not Available	Not Available

Emergency telephone number

Association / Organisation	Not Available	Not Available
Emergency telephone numbers	Not Available	1800 039 008 (Aust)
Other emergency telephone numbers	Not Available	+61 3 9573 3112 (International)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

Label elements

Hazard pictogram(s)	
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SIGNAL WORD	DANGER
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Hazard statement(s)

H319	Causes serious eye irritation.
H317	May cause an allergic skin reaction.
H360	May damage fertility or the unborn child.

Continued...

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

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

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

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/attention.
P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.

Precautionary statement(s) Storage

P405	Store locked up.
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Precautionary statement(s) Disposal

P501	Dispose of contents/container in accordance with local regulations.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS**Substances**

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Not Available	10-30	resins
111-76-2	10-30	<u>ethylene glycol monobutyl ether</u>
12007-60-2	<2	<u>lithium tetraborate</u>
112-34-5	<2	<u>diethylene glycol monobutyl ether</u>
55406-53-6	<1	<u>3-iodo-2-propynyl butyl carbamate</u>
26530-20-1	<0.2	<u>2-octyl-4-isothiazolin-3-one</u>
92502-70-0	<0.2	<u>eucalyptus oil</u>
22464-99-9	<0.1	<u>zirconium 2-ethylhexanoate</u>
366-18-7	<0.1	<u>2,2'-bipyridyl</u>
	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES**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"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.

- ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.

Followed acute or short term repeated exposures to ethylene glycol monoalkyl ethers and their acetates:

- ▶ Hepatic metabolism produces ethylene glycol as a metabolite.
- ▶ Clinical presentation, following severe intoxication, resembles that of ethylene glycol exposures.
- ▶ Monitoring the urinary excretion of the alkoxyacetic acid metabolites may be a useful indication of exposure.

[Ellenhorn and Barceloux: Medical Toxicology]

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

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

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

In such an event consider:

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

Special hazards arising from the substrate or mixture

Fire Incompatibility	None known.
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves in the event of a fire. ▶ Prevent, by any means available, spillage from entering drains or water courses. ▶ Use fire fighting procedures suitable for surrounding area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ The material is not readily combustible under normal conditions. ▶ However, it will break down under fire conditions and the organic component may burn. ▶ Not considered to be a significant fire risk. ▶ Heat may cause expansion or decomposition with violent rupture of containers. ▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO). ▶ May emit acrid smoke. <p>Decomposes on heating and produces toxic fumes of:</p> <ul style="list-style-type: none"> · carbon dioxide (CO₂) · other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes. May emit corrosive fumes.</p>
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<ul style="list-style-type: none"> ▶ 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	<p>Chemical Class: ester and ethers For release onto land: recommended sorbents listed in order of priority.</p> <table border="1"> <thead> <tr> <th>SORBENT TYPE</th> <th>RANK</th> <th>APPLICATION</th> <th>COLLECTION</th> <th>LIMITATIONS</th> </tr> </thead> <tbody> <tr> <td colspan="5">LAND SPILL - SMALL</td> </tr> <tr> <td>cross-linked polymer - particulate</td> <td>1</td> <td>shovel</td> <td>shovel</td> <td>R, W, SS</td> </tr> <tr> <td>cross-linked polymer - pillow</td> <td>1</td> <td>throw</td> <td>pitchfork</td> <td>R, DGC, RT</td> </tr> <tr> <td>sorbent clay - particulate</td> <td>2</td> <td>shovel</td> <td>shovel</td> <td>R,I, P</td> </tr> <tr> <td>wood fiber - particulate</td> <td>3</td> <td>shovel</td> <td>shovel</td> <td>R, W, P, DGC</td> </tr> </tbody> </table>	SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS	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 - particulate	3	shovel	shovel	R, W, P, DGC
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wood fiber - pillow	3	throw	pitchfork	R, P, DGC, RT
treated wood fiber - pillow	3	throw	pitchfork	DGC, RT

LAND SPILL - MEDIUM

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

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

- ▶ Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite.

- ▶ The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite (Na₂S₂O₅) or sodium bisulfite (NaHSO₃), or 12% sodium sulfite (Na₂SO₃) and 8% hydrochloric acid (HCl).

- ▶ Glutathione has also been used to inactivate the isothiazolinones.

- ▶ Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal.

- ▶ If contamination of drains or waterways occurs, advise emergency services.

- ▶ After clean up operations, decontaminate and launder all protective clothing

- ▶ and equipment before storing and re-using.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ DO NOT allow material to contact humans, exposed food or food utensils. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ Keep containers securely sealed when not in use. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<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	None known

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	ethylene glycol monobutyl ether	2-Butoxyethanol	96.9 mg/m ³ / 20 ppm	242 mg/m ³ / 50 ppm	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3

Continued...

ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
lithium tetraborate	Lithium tetraborate	4.3 mg/m3	47 mg/m3	280 mg/m3
lithium tetraborate	Lithium tetraborate pentahydrate	6.6 mg/m3	72 mg/m3	430 mg/m3
diethylene glycol monobutyl ether	Butoxyethoxy)ethanol, 2-(2-; (Diethylene glycol monobutyl ether)	30 ppm	33 ppm	200 ppm
3-iodo-2-propynyl butyl carbamate	Butyl-3-iodo-2-propynylcarbamate	3.3 mg/m3	36 mg/m3	220 mg/m3

Ingredient	Original IDLH	Revised IDLH
resins	Not Available	Not Available
ethylene glycol monobutyl ether	700 ppm	700 [Unch] ppm
lithium tetraborate	Not Available	Not Available
diethylene glycol monobutyl ether	Not Available	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available	Not Available
2-octyl-4-isothiazolin-3-one	Not Available	Not Available
eucalyptus oil	Not Available	Not Available
zirconium 2-ethylhexanoate	500 mg/m3	25 mg/m3
2,2'-bipyridyl	Not Available	Not Available

MATERIAL DATA

Fragrance substance with is an established contact allergen in humans.
 Scientific Committee on Consumer Safety SCCS OPINION on Fragrance allergens in cosmetic products 2012
 CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas)
 (CEL = Chemwatch Exposure Limit)

Exposure controls

Appropriate engineering controls	<p>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.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>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.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection. An approved self contained breathing apparatus (SCBA) may be required in some situations.</p> <p>Provide adequate ventilation in warehouse or closed storage area. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.</p>																			
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air).</td> <td>0.25-0.5 m/s (50-100 f/min.)</td> </tr> <tr> <td>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)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min.)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table> <p>Within each range the appropriate value depends on:</p> <table border="1"> <thead> <tr> <th>Lower end of the range</th> <th>Upper end of the range</th> </tr> </thead> <tbody> <tr> <td>1: Room air currents minimal or favourable to capture</td> <td>1: Disturbing room air currents</td> </tr> <tr> <td>2: Contaminants of low toxicity or of nuisance value only.</td> <td>2: Contaminants of high toxicity</td> </tr> <tr> <td>3: Intermittent, low production.</td> <td>3: High production, heavy use</td> </tr> <tr> <td>4: Large hood or large air mass in motion</td> <td>4: Small hood-local control only</td> </tr> </tbody> </table> <p>Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.</p>	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.)	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
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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 																			

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	<ul style="list-style-type: none"> ▶ 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 moisturizer 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>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> <ul style="list-style-type: none"> ▶ Butyl rubber gloves ▶ Nitrile rubber gloves
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.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:

"Forsberg Clothing Performance Index".

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:
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Material	CPI
BUTYL	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
VITON	C
##propylene	glycol

* CPI - Chemwatch Performance Index

A: Best Selection

Respiratory protection

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

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

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

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	AK-AUS P2	-	AK-PAPR-AUS / Class 1 P2
up to 50 x ES	-	AK-AUS / Class 1 P2	-
up to 100 x ES	-	AK-2 P2	AK-PAPR-2 P2 ^

^ - Full-face

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

Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content. The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

Continued...

B: Satisfactory; may degrade after 4 hours continuous immersion
 C: Poor to Dangerous Choice for other than short term immersion

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Hazy, pale to dark brown, low viscosity liquid with a slight odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.95-1.05
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	>200
pH (as supplied)	8-10	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	20-180 @25C
Initial boiling point and boiling range (°C)	107 (initial)	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	75-83
Vapour pressure (kPa)	2.2	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	0.8	VOC g/L	183-202

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Unstable in the presence of incompatible materials. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

Inhaled	The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Isothiazolinones are moderately to highly toxic by oral administration. The major signs of toxicity were severe gastric irritation, lethargy, and ataxia
Skin Contact	Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Aqueous solutions of isothiazolinones may be irritating or even corrosive depending on concentration. Solutions containing more than 0.5% (5000 ppm active substance) may produce severe irritation of human skin whilst solutions containing more than 100 ppm may irritate the skin. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur. Solutions containing isothiazolinones may produce corrosion of the mucous membranes and cornea. Instillation of 0.1 ml of an aqueous solution containing 560 ppm isothiazolinone into rabbit eye did not produce irritation whereas concentrations, typically around 3% and 5.5 %, were severely irritating or corrosive to the eye.. Symptoms included clouding of the cornea, chemosis and swelling of the eyelids.

Chronic

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.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in impaired fertility on the basis of: - clear evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.

There is sufficient evidence to provide a strong presumption that human exposure to the material may result in developmental toxicity, generally on the basis of: - clear results in appropriate animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

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

The isothiazolinones are known contact sensitizers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.

The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.

Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :

- ▶ The strongest sensitizers are the chlorinated isothiazolinones.
- ▶ There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones.
- ▶ There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones.
- ▶ Although classified as sensitizers, the nonchlorinated isothiazolinones are considerably less potent sensitizers than are the chlorinated isothiazolinones.
- ▶ By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced.
- ▶ Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons.
- ▶ Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available.

* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196

Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in *Salmonella typhimurium* strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells *in vitro* and of cytogenetic effects and DNA-binding *in vivo*. The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.

A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.

Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses

On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.

Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600

QUANTEC	TOXICITY	IRRITATION
	Not Available	Not Available
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
	Inhalation (rat) LC50: 450 ppm/4hr ^[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg ^[2]	Skin (rabbit): 500 mg, open; mild
lithium tetraborate	TOXICITY	IRRITATION
	Not Available	Not Available
diethylene glycol monobutyl ether	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2700 mg/kg ^[2]	Eye (rabbit): 20 mg/24h moderate
	Oral (rat) LD50: 4500 mg/kg ^[2]	Eye (rabbit): 5 mg - SEVERE
3-iodo-2-propynyl butyl carbamate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Eye: Irritating
	Inhalation (rat) LC50: 0.680 mg/l/4h ^[2]	Skin: Slight irritant
	Oral (rat) LD50: 1056 mg/kg ^[2]	
2-octyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 690 mg/kgE ^[2]	Eye (rabbit): 0.5% non irritant
	Oral (rat) LD50: 550 mg/kgS ^[2]	Eye (rabbit): 45% conc CORROSIVE

QUANTEC

		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
eucalyptus oil	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 2480 mg/kg ^[2]	Skin (rabbit): 500 mg/24h - mod
	Oral (rat) LD50: 2480 mg/kg ^[2]	
zirconium 2-ethylhexanoate	TOXICITY	IRRITATION
	Not Available	Not Available
2,2'-bipyridyl	TOXICITY	IRRITATION
	Oral (rat) LD50: 100 mg/kgd ^[2]	Not Available

Legend:

1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2.* Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

QUANTEC	<p>Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A prohaptens is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems. A prohaptens is a chemical that itself is non- or low-sensitising but that is transformed into a hapten in the skin (bioactivation) usually via enzyme catalysis. It is not always possible to know whether a particular allergen that is not directly reactive acts as a prohaptens or as a prohaptens, or both, because air oxidation and bioactivation can often give the same product (geraniol is an example). Some chemicals might act by all three pathways.</p> <p>Prohaptens Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohaptens. In the case of prohaptens, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.</p> <p>The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohaptens can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or in vivo and in vitro studies of sensitisation potential and chemical reactivity.</p> <p>QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohaptens) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohaptens. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation</p>
ETHYLENE GLYCOL MONO BUTYL ETHER	<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> <p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.</p> <p>EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.</p> <p>Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE <i>in vitro</i> than those of rats.</p> <p>Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA <i>in vitro</i> and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA <i>in vitro</i>.</p> <p>Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in <i>S. typhimurium</i> strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. <i>In vitro</i> cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.</p> <p>Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was</p>

no significant hazard for human carcinogenicity

Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes).

Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m³ and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m³), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m³), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m³) indicate that the members of the category are not teratogenic.

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

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

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

Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility.

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

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

For ethylene glycol:

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

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

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

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

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

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

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

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

Renal Effects. Adverse renal effects after ethylene glycol ingestion in humans can be observed during the third stage of ethylene glycol toxicity 24-72 hours after acute exposure. The hallmark of renal toxicity is the presence of birefringent calcium oxalate monohydrate crystals deposited in renal tubules and their presence in urine after ingestion of relatively high amounts of ethylene glycol. Other signs of nephrotoxicity can include tubular cell degeneration and necrosis and tubular interstitial inflammation. If untreated, the degree of renal damage caused by high doses of ethylene glycol progresses and leads to haematuria, proteinuria, decreased renal function, oliguria, anuria, and ultimately renal failure. These changes in the kidney are linked to acute tubular necrosis but normal or near normal renal function can return with adequate supportive therapy.

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

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

Effects on cranial nerves appear late (generally 5-20 days post-ingestion), are relatively rare, and according to some investigators constitute a fourth, late cerebral phase in ethylene glycol intoxication. Clinical manifestations of the cranial neuropathy commonly involve lower motor neurons of the facial and bulbar nerves and are reversible over many months.

Reproductive Effects: Reproductive function after intermediate-duration oral exposure to ethylene glycol has been tested in three multi-generation studies (one in rats and two in mice) and several shorter studies (15-20 days in rats and mice). In these studies, effects on fertility, foetal viability, and male reproductive organs were observed in mice, while the only effect in rats was an increase in gestational duration.

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

Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.

Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available *in vivo* and *in vitro* laboratory studies

	<p>provide consistently negative genotoxicity results for ethylene glycol.</p> <p>NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS</p>
LITHIUM TETRABORATE	<p>No data of toxicological significance identified in literature search.</p>
DIETHYLENE GLYCOL MONOBUTYL ETHER	<p>For diethylene glycol monoalkyl ethers and their acetates: This category includes diethylene glycol ethyl ether (DGEE), diethylene glycol propyl ether (DGPE) diethylene glycol butyl ether (DGBE) and diethylene glycol hexyl ether (DGHE) and their acetates.</p> <p>Acute toxicity: There are adequate oral, inhalation and/or dermal toxicity studies on the category members. Oral LD50 values in rats for all category members are all > 3000 mg/kg bw, with values generally decreasing with increasing molecular weight. Four to eight hour acute inhalation toxicity studies were conducted for all category members except DGPE in rats at the highest vapour concentrations achievable. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 2000 mg/kg bw (DGHE) to 15000 mg/kg bw (DGEEA). Signs of acute toxicity in rodents are consistent with non-specific CNS depression typical of organic solvents in general. All category members are slightly irritating to skin and slightly to moderately irritating to eyes (with the exception of DGHE, which is highly irritating to eyes). Sensitisation tests with DGEE, DGEEA, DGPE, DGBE and DGBEA in animals and/or humans were negative.</p> <p>Repeat dose toxicity: Valid oral studies conducted using DGEE, DGPE, DGBEA, DGHE and the supporting chemical DGBE ranged in duration from 30 days to 2 years. Effects predominantly included kidney and liver toxicity, absolute and/or relative changes in organ weights, and some changes in haematological parameters. All effects were seen at doses greater than 800-1000 mg/kg bw/day from oral or dermal studies; no systemic effects were observed in inhalation studies with less than continuous exposure regimens.</p> <p>Mutagenicity: DGEE, DGEEA, DGBE, DGBEA and DGHE generally tested negative for mutagenicity in <i>S. typhimurium</i> strains TA98, TA100, TA1535, TA1537 and TA1538 and DGBEA tested negative in <i>E. coli</i> WP2uvrA, with and without metabolic activation. <i>In vitro</i> cytogenetic and sister chromatid exchange assays with DGBE and DGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus or cytogenetic tests with DGEE, DGBE and DGHE in rats and mice were negative, indicating that these diethylene glycol ethers are not likely to be genotoxic.</p> <p>Reproductive and developmental toxicity: Reliable reproductive toxicity studies on DGEE, DGBE and DGHE show no effect on fertility at the highest oral doses tested (4,400 mg/kg/day for DGEE in the mouse and 1,000 mg/kg/day for DGBE and DGHE in the rat). The dermal NOAEL for reproductive toxicity in rats administered DGBE also was the highest dose tested (2,000 mg/kg/day). Although decreased sperm motility was noted in F1 mice treated with 4,400 mg/kg/day DGEE in drinking water for 14 weeks, sperm concentrations and morphology, histopathology of the testes and fertility were not affected. Results of the majority of adequate repeated dose toxicity studies in which reproductive organs were examined indicate that DGPE and DGBEA do not cause toxicity to reproductive organs (including the testes). Test material-related testicular toxicity was not noted in the majority of the studies with DGEE or DGEEA. Results of the developmental toxicity studies conducted with DGEE, DGBE and DGHE are almost exclusively negative. In these studies, effects on the foetus are generally not observed (even at concentrations that produced maternal toxicity). Exposure to 102 ppm (560 mg/m³) DGEE by inhalation (maximal achievable vapour concentration) or 1385 mg/kg/day DGEE by the dermal route during gestation did not cause maternal or developmental toxicity in the rat. Maternal toxicity and teratogenesis were not observed in rabbits receiving up to 1000 mg/kg/day DGBE by the dermal route during gestation; however a transient decrease in body weight was observed, which reversed by Day 21. In the mouse, the only concentration of DGEE tested (3500 mg/kg/day by gavage) caused maternal, but no foetal toxicity. Also, whereas oral administration of 2050 mg/kg/day DGBE (gavage) to the mouse and 1000 mg/kg/day DGHE (dietary) caused maternal toxicity, these doses had no effect on the developing foetus</p>
3-iodo-2-propynyl BUTYL CARBAMATE	<p>for 3-iodo-2-propynyl butyl carbamate (IPBC):</p> <p>Acute toxicity: Acceptable acute toxicity studies with IPBC indicate low toxicity except eye irritation. In a primary eye irritation study in rabbit, IPBC technical was severely irritating to the eyes of white rabbits, with corneal opacity and corneal vascularization reported in unwashed eyes by day 21 post-treatment. The technical grade of IPBC was slightly irritating to the skin of white rabbits. In a dermal sensitization study in Guinea pigs IPBC technical, at a concentration of 0.32%, produced no evidence of sensitization in male and female Guinea pigs.</p> <p>Subchronic toxicity: In a subchronic oral toxicity study, male and female Sprague-Dawley rats received IPBC technical by gavage for 13 weeks at doses of 0, 20, 50, and 125 mg/kg/day. At the 125 mg/kg/day dose level, body weight gain was decreased by 19% in male rats for weeks 1-13 of the study, and by 12% in female rats over the same period. Absolute liver weight was increased by 20% in male rats at the 125 mg/kg/day dose, and by 31% in female rats at this dose level. Liver to body weight ratio was significantly increased by approximately 31% in both male and female rats at the 125 mg/kg/day dose level, while kidney to body weight ratio in female rats was increased 18% at the 125 mg/kg/day dose level. The systemic NOEL was considered to be 20 mg/kg/day, while the systemic LEL was considered to be 50 mg/kg/day, based on increased liver to body weight ratio.</p> <p>In a subchronic dermal toxicity study, male and female Sprague-Dawley rats (10/sex/dose) received dermal doses of 50, 200, and 500 mg/kg/day IPBC technical grade (97.5%) to the shaved skin for five days a week, six hours per day. At the 500 mg/kg/day dose, decreased body weight (4-6%) and weight gain (11%) were observed in male rats, but not in female rats. In female rats, significant increases in haemoglobin, haematocrit, and eosinophils were observed at the 500 mg/kg/day dose level. Reticulocytes as a percentage of red cells were decreased in the 50 and 200 mg/kg/day dose groups but not at the 500 mg/kg/day dose level. Females in this study showed inhibition of plasma cholinesterase at 500 mg/kg/day test article, which may have been the result of either direct liver toxicity or inhibition of cholinesterase itself. Based upon the results of this study, the systemic NOEL is 200 mg/kg/day, the systemic LEL is 500 mg/kg/day for male and female rats.</p> <p>Carcinogenicity: In a 2-year chronic toxicity/carcinogenicity study, technical grade IPBC (98.68% ai) was administered to male and female Sprague Dawley rats (50/sex/group) at dose levels of 0, 20, 40, and 80 mg/kg/day. There were no statistically significant increases in tumor incidences in male rats. The incidence of mammary gland fibroadenoma and combined fibroadenoma/carcinoma in female rats was significantly increased at the 20 mg/kg/day dose level but there was no dose-related trend.</p> <p>Developmental and reproductive toxicity: The developmental toxicity of IPBC was assessed in pregnant Sprague-Dawley rats on gestation days six through 15 by oral administration of the test chemical at doses of 0, 20, 50, and 125 mg/kg/day. Maternal toxicity as reduced body weight gain during dosing was observed at the 125 mg/kg/day dose level. Developmental toxicity consisted of an increased incidence of skeletal abnormalities at the 125 mg/kg/day dose level. The maternal toxicity NOEL was determined to be 50 mg/kg/day, and the maternal toxicity LEL was determined to be 125 mg/kg/day, based on reduced body weight gain. The developmental toxicity NOEL was determined to be 50 mg/kg/day, and the developmental toxicity LEL was determined to be 125 mg/kg/day, based on incompletely ossified frontal skull bones and pelvic girdles.</p> <p>A 2-generation reproductive toxicity study was conducted in male and female Sprague-Dawley rats. IPBC technical was administered over two generations at doses of 0, 120, 300, and 750 ppm (0, 6, 15, and 37.5 mg/kg/day). Reduced body weight and food consumption was observed for P1 and F1 males during the pre-mating period at the 37.5 mg/kg/day dose. A decreased mean live birth index was reported for P1 and F1 generations without an effect on viability and development of pups. No adverse effects on reproductive indices or mating performance were observed at any dose level. The parental toxicity NOEL was determined to be 15 mg/kg/day, and the parental toxicity LEL was determined to be 37.5 mg/kg/day, based on decreased body weight and food consumption during pre-mating for P1 and F1 males, and decreased mean live birth index for the P1 and F1 generations. The reproductive toxicity NOEL was determined to be 37.5 mg/kg/day, and the reproductive toxicity LEL was determined to be >37.5 mg/kg/day.</p> <p>Mutagenicity: In a mutagenicity study, IPBC technical was tested for the ability to cause mutations in <i>Salmonella typhimurium</i> strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100. In the five strains used, IPBC was found to be non-mutagenic in the presence or absence of metabolic activation at the concentrations tested, 1-1000 jig/plate. In a micronucleus assay in mice, IPBC at doses of 200, 600, and 2000 mg/kg did not induce any significant increase of the PCE containing micronuclei from the treated mice when compared to that of the vehicle control mice. In two independent unscheduled DNA synthesis (UDS) assays in primary rat hepatocytes, eight doses of IPBC ranging from 3.0 to 13.5 ug/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 ug/ml were cytotoxic, supporting the conclusion that IPBC induced cytotoxicity but no genotoxicity in this assay.</p> <p>Metabolism: Based on the metabolite identification data, a scheme for metabolism of IPBC was proposed. According to this scheme, IPBC undergoes reductive dehalogenation followed by dealkylation to form the URM-9 and URM-10 metabolites. In addition, de-carboxylation following reductive dehalogenation yields carbon dioxide. Various other metabolites formed from dehalogenation are glucuronidated and constitute minor metabolites of IPBC..</p>
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	<p>ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day</p>
EUCALYPTUS OIL	<p>For monoterpenes: The chemical category designated terpenoid hydrocarbons includes three simple C10 isomeric monocyclic terpene hydrocarbons (<i>α</i>-limonene, <i>d</i>-limonene, and terpinolene) two simple C10 acyclic terpene hydrocarbons (<i>β</i>-myrcene and dihydromyrcene) and mixtures composed primarily of <i>d</i>-limonene, <i>d</i>-limonene (dipentene), terpinolene, myrcene, and <i>α</i> and <i>β</i>-pinene Monoterpene hydrocarbons are mainly released by coniferous woodland such as pine trees, cedars, redwood and firs. To a lesser extent, they are also</p>

produced and released by deciduous plants. They are common components of traditional foods occurring in essentially all fruits and vegetables.

Members of this chemical category are of very low acute toxicity

Studies of terpene hydrocarbons indicate that they are rapidly absorbed, distributed, metabolised and excreted. The principal metabolic pathway involves side chain oxidation to yield monocyclic terpene alcohols and carboxylic acids. These metabolites are mainly conjugated with glucuronic acid and excreted in the urine, or to a lesser extent in the feces. A secondary pathway involves epoxidation of either the exocyclic or endocyclic double bond yielding an epoxide that is subsequently detoxicated via formation of the corresponding diol or conjugation with glutathione. Although some species- and sex-specific differences exist, studies for *d*-limonene and *beta*-myrcene indicate that the monoterpene hydrocarbons in this chemical category will participate in common pathways of absorption, distribution, metabolism and excretion.

Genotoxicity: Based on the results of this *in vivo* genotoxicity assay and the numerous *in vitro* genotoxicity assays, it is unlikely that any of these materials would exhibit a significant genotoxic potential *in vivo*.

Carcinogenicity: Under the conditions of 2-year gavage studies, conducted by NTP, there was clear evidence of carcinogenic activity of *d*-limonene for male F344/N rats as shown by increased incidences in tubular cell hyperplasia, adenomas, and adenocarcinomas of the kidney. There was no evidence of carcinogenic activity of *d*-limonene for female rats receiving 300 or 600 mg/kg bw/d. It has been demonstrated that renal lesions, which were observed in the NTP study, resulted from the accumulation of aggregates of *alpha*-2 microglobulin (a low molecular-weight protein synthesised in the liver) and limonene-1,2-epoxide in the P2 segment of the renal proximal tubule. While humans produce low molecular weight serum proteins, which are reabsorbed by the kidney, there is no evidence that a similar *alpha*-2 microglobulin is produced.

The kidney changes seen in male rats administered limonene have been well characterized, and are known to be specific to the male rat and of no significance in human risk assessment.

Reproductive toxicity: Substances within this chemical category exhibit low reproductive toxicity potential. This is based on the results of three reproductive toxicity assays, using sweet orange peel oil predominantly composed of *d*-limonene and *beta*-myrcene.

Developmental toxicity: Given the results of six developmental toxicity assays using limonene, sweet orange oil and *beta*-myrcene, it may be concluded that the substances within this chemical category exhibit low developmental toxicity potential

Fragrance allergens act as haptens, i.e. low molecular weight chemicals that are immunogenic only when attached to a carrier protein. However, not all sensitising fragrance chemicals are directly reactive, but require previous activation. A **prehapten** is a chemical that itself is non- or low-sensitising, but that is transformed into a hapten outside the skin by simple chemical transformation (air oxidation, photoactivation) and without the requirement of specific enzymatic systems.

In the case of prehapten, it is possible to prevent activation outside the body to a certain extent by different measures, e.g. prevention of air exposure during handling and storage of the ingredients and the final product, and by the addition of suitable antioxidants. When antioxidants are used, care should be taken that they will not be activated themselves and thereby form new sensitisers.

Prehapten

Most terpenes with oxidisable allylic positions can be expected to autoxidise on air exposure due to their inherent properties. Depending on the stability of the oxidation products that are formed, a difference in the sensitisation potency of the oxidised terpenes can be seen

Autoxidation is a free radical chain reaction in which hydrogen atom abstraction in combination with addition of oxygen forms peroxy radicals. The reaction shows selectivity for positions where stable radicals can be formed. So far, all fragrance substances that have been investigated with regard to the influence of autoxidation on the allergenic potential, including identification of formed oxidation products, have oxidisable allylic positions that are able to form hydroperoxides and/or hydrogen peroxide as primary oxidation products upon air exposure. Once the hydroperoxides have been formed outside the skin they form specific antigens and act as skin sensitisers. Secondary oxidation products such as aldehydes and epoxides can also be allergenic, thus further increasing the sensitisation potency of the autoxidation mixture. The process of photoactivation may also play a role, but further research is required to establish whether this activation route is currently underestimated in importance due to insufficient knowledge of the true haptens in this context.

It should be noted that activation of substances via air oxidation results in various haptens that might be the same or cross-reacting with other haptens (allergens). The main allergens after air oxidation of linalool and linalyl acetate are the hydroperoxides. If linalyl acetate is chemically hydrolysed outside the skin it can thereafter be oxidised to the same haptens as seen for linalool. A corresponding example is citronellol and citronellyl acetate. In clinical studies, concomitant reactions to oxidised linalool and oxidised linalyl acetate have been observed. Whether these reactions depend on cross-reactivity or are due to exposure to both fragrance substances cannot be elucidated as both have an allergenic effect themselves. Linalool and linalyl acetate are the main components of lavender oil. They autoxidise on air exposure also when present in the essential oil, and form the same oxidation products found in previous studies of the pure synthetic terpenes. Experimental sensitisation studies showed that air exposure of lavender oil increased the sensitisation potency. Patch test results in dermatitis patients showed a connection between positive reactions to oxidised linalool, linalyl acetate and lavender oil.

Prohapten

Compounds that are bioactivated in the skin and thereby form haptens are referred to as prohapten.

In the case of prohapten, the possibility to become activated is inherent to the molecule and activation cannot be avoided by extrinsic measures. Activation processes increase the risk for cross-reactivity between fragrance substances. Crossreactivity has been shown for certain alcohols and their corresponding aldehydes, i.e. between geraniol and geranial (citral) and between cinnamyl alcohol and cinnamal.

The human skin expresses enzyme systems that are able to metabolise xenobiotics, modifying their chemical structure to increase hydrophilicity and allow elimination from the body. Xenobiotic metabolism can be divided into two phases: phase I and phase II. Phase I transformations are known as activation or functionalisation reactions, which normally introduce or unmask hydrophilic functional groups. If the metabolites are sufficiently polar at this point they will be eliminated. However, many phase I products have to undergo subsequent phase II transformations, i.e. conjugation to make them sufficiently water soluble to be eliminated. Although the purpose of xenobiotic metabolism is detoxification, it can also convert relatively harmless compounds into reactive species. Cutaneous enzymes that catalyse phase I transformations include the cytochrome P450 mixed-function oxidase system, alcohol and aldehyde dehydrogenases, monoamine oxidases, flavin-containing monooxygenases and hydrolytic enzymes. Acyltransferases, glutathione S-transferases, UDP-glucuronosyltransferases and sulfotransferases are examples of phase II enzymes that have been shown to be present in human skin. These enzymes are known to catalyse both activating and deactivating biotransformations, but the influence of the reactions on the allergenic activity of skin sensitisers has not been studied in detail. Skin sensitising prohapten can be recognised and grouped into chemical classes based on knowledge of xenobiotic bioactivation reactions, clinical observations and/or *in vivo* and *in vitro* studies of sensitisation potential and chemical reactivity.

QSAR prediction: The relationships between molecular structure and reactivity that form the basis for structural alerts are based on well established principles of mechanistic organic chemistry. Examples of structural alerts are aliphatic aldehydes (alerting to the possibility of sensitisation via a Schiff base reaction with protein amino groups), and alpha,beta-unsaturated carbonyl groups, C=C-CO- (alerting to the possibility of sensitisation via Michael addition of protein thiol groups). Prediction of the sensitisation potential of compounds that can act via abiotic or metabolic activation (pre- or prohapten) is more complex compared to that of compounds that act as direct haptens without any activation. The autoxidation patterns can differ due to differences in the stability of the intermediates formed, e.g. it has been shown that autoxidation of the structural isomers linalool and geraniol results in different major haptens/allergens. Moreover, the complexity of the prediction increases further for those compounds that can act both as pre- and prohapten. In such cases, the impact on the sensitisation potency depends on the degree of abiotic activation (e.g. autoxidation) in relation to the metabolic activation.

ZIRCONIUM 2-ETHYLHEXANOATE

Fatty acid salts are of low acute toxicity. Their skin and eye irritation potential is chain length dependent and decreases with increasing chain length - they are poorly absorbed through the skin nor are they skin sensitisers. The available repeated dose toxicity data demonstrate the low toxicity of the fatty acids and their salts. Also, they are not considered to be mutagenic, genotoxic or carcinogenic, and are not reproductive or developmental toxicants. Accidental ingestion of fatty acid salt containing detergent products is not expected to result in any significant adverse health effects. This assessment is based on toxicological data demonstrating the low acute oral toxicity of fatty acid salts and the fact that not a single fatality has been reported in the UK following accidental ingestion of detergents containing fatty acid salts. Also in a report published by the German Federal Institute for Health Protection of Consumers and Veterinary Medicine, detergent products were not mentioned as dangerous products with a high incidence of poisoning. The estimated total human exposure to fatty acid salts, from the different exposure scenarios for the handling and use of detergent products containing fatty acid salts, showed a margin of exposure (MOE) of 258,620. This extremely large MOE is large enough to be reassuring with regard to the relatively small variability of the hazard data on which it is based. Also, in the UK, the recommended dietary fatty acid intake by the Department of Health is about 100 g of fatty acids per day or 1.7 g (1700 mg) of fatty acids per kilogram body weight per day. This exposure is several orders of magnitude above that resulting from exposure to fatty acid salts in household cleaning products. Based on the available data, the use of fatty acid salts in household detergent and cleaning products does not raise any safety concerns with regard to consumer

2,2'-BIPYRIDYL	Exposure to the material for prolonged periods may cause physical defects in the developing embryo (teratogenesis).
QUANTEC & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & EUCALYPTUS OIL & 2,2'-BIPYRIDYL	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>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.</p>
QUANTEC & EUCALYPTUS OIL	<p>Adverse reactions to fragrances in perfumes and in fragranced cosmetic products include allergic contact dermatitis, irritant contact dermatitis, photosensitivity, immediate contact reactions (contact urticaria), and pigmented contact dermatitis. Airborne and conjugal contact dermatitis occur. Intolerance to perfumes, by inhalation, may occur if the perfume contains a sensitising principal. Symptoms may vary from general illness, coughing, phlegm, wheezing, chest-tightness, headache, exertional dyspnoea, acute respiratory illness, hayfever, and other respiratory diseases (including asthma). Perfumes can induce hyper-reactivity of the respiratory tract without producing an IgE-mediated allergy or demonstrable respiratory obstruction. This was shown by placebo-controlled challenges of nine patients to "perfume mix". The same patients were also subject to perfume provocation, with or without a carbon filter mask, to ascertain whether breathing through a filter with active carbon would prevent symptoms. The patients breathed through the mouth, during the provocations, as a nose clamp was used to prevent nasal inhalation. The patient's earlier symptoms were verified; breathing through the carbon filter had no protective effect. The symptoms were not transmitted via the olfactory nerve but they may have been induced by trigeminal reflex via the respiratory tract or by the eyes.</p> <p>Cases of occupational asthma induced by perfume substances such as isoamyl acetate, limonene, cinnamaldehyde and benzaldehyde, tend to give persistent symptoms even though the exposure is below occupational exposure limits.</p> <p>Inhalation intolerance has also been produced in animals. The emissions of five fragrance products, for one hour, produced various combinations of sensory irritation, pulmonary irritation, decreases in expiratory airflow velocity as well as alterations of the functional observational battery indicative of neurotoxicity in mice. Neurotoxicity was found to be more severe after mice were repeatedly exposed to the fragrance products, being four brands of cologne and one brand of toilet water.</p> <p>Contact allergy to fragrances is relatively common, affecting 1 to 3% of the general population, based on limited testing with eight common fragrance allergens and about 16 % of patients patch tested for suspected allergic contact dermatitis.</p> <p>Contact allergy to fragrance ingredients occurs when an individual has been exposed, on the skin, to a sufficient degree of fragrance contact allergens. Contact allergy is a life-long, specifically altered reactivity in the immune system. This means that once contact allergy is developed, cells in the immune system will be present which can recognise and react towards the allergen. As a consequence, symptoms, i.e. allergic contact dermatitis, may occur upon re-exposure to the fragrance allergen(s) in question. Allergic contact dermatitis is an inflammatory skin disease characterised by erythema, swelling and vesicles in the acute phase. If exposure continues it may develop into a chronic condition with scaling and painful fissures of the skin. Allergic contact dermatitis to fragrance ingredients is most often caused by cosmetic products and usually involves the face and/or hands. It may affect fitness for work and the quality of life of the individual. Fragrance contact allergy has long been recognised as a frequent and potentially disabling problem. Prevention is possible as it is an environmental disease and if the environment is modified (e.g. by reduced use concentrations of allergens), the disease frequency and severity will decrease.</p> <p>Fragrance contact allergy is mostly non-occupational and related to the personal use of cosmetic products. Allergic contact dermatitis can be severe and widespread, with a significant impairment of quality of life and potential consequences for fitness for work. Thus, prevention of contact sensitisation to fragrances, both in terms of primary prevention (avoiding sensitisation) and secondary prevention (avoiding relapses of allergic contact dermatitis in those already sensitised), is an important objective of public health risk management measure.</p> <p>Hands: Contact sensitisation may be the primary cause of hand eczema, or may be a complication of irritant or atopic hand eczema. The number of positive patch tests has been reported to correlate with the duration of hand eczema, indicating that long-standing hand eczema may often be complicated by sensitisation. Fragrance allergy may be a relevant problem in patients with hand eczema; perfumes are present in consumer products to which their hands are exposed. A significant relationship between hand eczema and fragrance contact allergy has been found in some studies based on patients investigated for contact allergy. However, hand eczema is a multi-factorial disease and the clinical significance of fragrance contact allergy in (severe) chronic hand eczema may not be clear.</p> <p>Axillae Bilateral axillary (underarm) dermatitis may be caused by perfume in deodorants and, if the reaction is severe, it may spread down the arms and to other areas of the body. In individuals who consulted a dermatologist, a history of such first-time symptoms was significantly related to the later diagnosis of perfume allergy.</p> <p>Face Facial eczema is an important manifestation of fragrance allergy from the use of cosmetic products (16). In men, after-shave products can cause an eczematous eruption of the beard area and the adjacent part of the neck and men using wet shaving as opposed to dry have been shown to have an increased risk of being fragrance allergic.</p> <p>Irritant reactions (including contact urticaria): Irritant effects of some individual fragrance ingredients, e.g. citral are known. Irritant contact dermatitis from perfumes is believed to be common, but there are no existing investigations to substantiate this. Many more people complain about intolerance or rashes to perfumes/perfumed products than are shown to be allergic by testing. This may be due to irritant effects or inadequate diagnostic procedures. Fragrances may cause a dose-related contact urticaria of the non-immunological type (irritant contact urticaria). Cinnamal, cinnamic alcohol, and Myroxylon pereirae are well recognised causes of contact urticaria, but others, including menthol, vanillin and benzaldehyde have also been reported. The reactions to Myroxylon pereirae may be due to cinnamates. A relationship to delayed contact hypersensitivity was suggested, but no significant difference was found between a fragrance-allergic group and a control group in the frequency of immediate reactions to fragrance ingredients in keeping with a nonimmunological basis for the reactions seen.</p> <p>Pigmentary anomalies: The term "pigmented cosmetic dermatitis" was introduced in 1973 for what had previously been known as melanosis faciei feminae when the mechanism (type IV allergy) and causative allergens were clarified. It refers to increased pigmentation, usually on the face/neck, often following sub-clinical contact dermatitis. Many cosmetic ingredients were patch tested at non-irritant concentrations and statistical evaluation showed that a number of fragrance ingredients were associated: jasmine absolute, ylang-ylang oil, cananga oil, benzyl salicylate, hydroxycitronellal, sandalwood oil, geraniol, geranium oil.</p> <p>Photo-reactions Musk ambrette produced a considerable number of allergic photocontact reactions (in which UV-light is required) in the 1970s and was later banned from use in the EU. Nowadays, photoallergic contact dermatitis is uncommon. Furocoumarins (psoralens) in some plant-derived fragrance ingredients caused phototoxic reactions with erythema followed by hyperpigmentation resulting in Berloque dermatitis. There are now limits for the amount of furocoumarins in fragrance products. Phototoxic reactions still occur but are rare.</p> <p>General/respiratory: Fragrances are volatile and therefore, in addition to skin exposure, a perfume also exposes the eyes and naso-respiratory tract. It is estimated that 2-4% of the adult population is affected by respiratory or eye symptoms by such an exposure. It is known that exposure to fragrances may exacerbate pre-existing asthma. Asthma-like symptoms can be provoked by sensory mechanisms. In an epidemiological investigation, a significant association was found between respiratory complaints related to fragrances and contact allergy to fragrance ingredients, in addition to hand eczema, which were independent risk factors in a multivariate analysis.</p>
QUANTEC & ZIRCONIUM 2-ETHYLHEXANOATE	No significant acute toxicological data identified in literature search.
ETHYLENE GLYCOL MONOBUTYL ETHER & DIETHYLENE GLYCOL MONOBUTYL ETHER	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
LITHIUM TETRABORATE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & EUCALYPTUS OIL & 2,2'-BIPYRIDYL	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

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irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

Legend: ✗ – Data available but does not fill the criteria for classification
✓ – Data available to make classification
⊖ – Data Not Available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

QUANTEC	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol monobutyl ether	LC50	96	Fish	1250mg/L	4
	EC50	48	Crustacea	>1000mg/L	4
	NOEC	96	Crustacea	1000mg/L	4
lithium tetraborate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
diethylene glycol monobutyl ether	LC50	96	Fish	1300mg/L	4
	EC50	48	Crustacea	>100mg/L	1
	EC50	96	Algae or other aquatic plants	>100mg/L	1
	NOEC	96	Algae or other aquatic plants	>=100mg/L	1
3-iodo-2-propynyl butyl carbamate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.067mg/L	4
	EC50	48	Crustacea	0.04mg/L	5
2-octyl-4-isothiazolin-3-one	NOEC	48	Crustacea	<0.01mg/L	4
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.047mg/L	4
	EC50	48	Crustacea	0.18mg/L	4
eucalyptus oil	BCF	1608	Fish	0.05mg/L	4
	NOEC	48	Crustacea	<=0.08mg/L	4
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.28mg/L	2
zirconium 2-ethylhexanoate	EC50	48	Crustacea	0.307mg/L	2
	EC50	96	Algae or other aquatic plants	>74mg/L	2
	NOEC	48	Algae or other aquatic plants	0.247mg/L	2
	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
LC50	96	Fish	>100mg/L	2	
EC50	48	Crustacea	>0.17mg/L	2	
EC50	72	Algae or other aquatic plants	>0.042mg/L	2	
NOEC	72	Algae or other aquatic plants	0.004mg/L	2	
2,2'-bipyridyl	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

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

For ethylene glycol monoalkyl ethers and their acetates:

Members of this category include ethylene glycol propyl ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE)

Environmental fate:

The ethers, like other simple glycol ethers possess no functional groups that are readily subject to hydrolysis in the presence of waters. The acetates possess an ester group that hydrolyses in neutral ambient water under abiotic conditions.

Level III fugacity modeling indicates that category members, when released to air and water, will partition predominately to water and, to a lesser extent, to air and soil. Estimates of soil and sediment partition coefficients (Kocs ranging from 1- 10) suggest that category members would exhibit high soil mobility. Estimated bioconcentration factors (log BCF) range from 0.463 to 0.732. Biodegradation studies indicate that all category members are readily biodegradable. The physical chemistry and environmental fate properties indicate that category members will not persist or bioconcentrate in the environment.

Ecotoxicity:

Glycol ether acetates do not hydrolyse rapidly into their corresponding glycol ethers in water under environmental conditions. The LC50 or EC50 values for EGHE are lower than those for EGPE and EGBE (which have shorter chain lengths and lower log Kow values). Overall, the LC50 values for the glycol ethers in aquatic species range from 94 to > 5000 mg/L. For EGHE, the 96-hour LC50 for *Brachydanio rerio* (zebra fish) is between 94 and mg/L, the 48-hour EC50 for *Daphnia magna* was 145 mg/L and the 72-hour EC50 values for biomass and growth rate of algae (*Scenedesmus subspicatus*) were 98 and 198 mg/L, respectively. LC50/EC50 values for EGPE and EGBE in aquatic species are 835 mg/l or greater.

Aquatic toxicity data for EGBEA show a 96-hour LC50 of 28.3 mg/L for rainbow trout (*Oncorhynchus mykiss*), a 48-hour LC50 of 37-143 mg/L for *Daphnia magna*, a 72-hour EC50 of greater than 500 mg/L for biomass or growth rate of algae (*Scenedesmus subspicatus* and *Pseudokirchneriella subcapitata*, respectively), and a 7-day EC10 of 30.4 mg/L and a NOEC of 16.4 mg/L for reproduction in *Ceriodaphnia dubia*.

For glycol ethers:

Environmental fate:

Ether groups are generally stable to hydrolysis in water under neutral conditions and ambient temperatures. OECD guideline studies indicate ready biodegradability for several glycol ethers although higher molecular weight species seem to biodegrade at a slower rate. No glycol ethers that have been tested demonstrate marked resistance to biodegradative processes. Upon release to the atmosphere by evaporation, high boiling glycol ethers are estimated to undergo photodegradation (atmospheric half lives = 2.4-2.5 hr). When released to water, glycol ethers undergo biodegradation (typically 47-92% after 8-21 days) and have a low potential for bioaccumulation (log Kow ranges from -1.73 to +0.51).

Ecotoxicity:

Aquatic toxicity data indicate that the tri- and tetra ethylene glycol ethers are "practically non-toxic" to aquatic species. No major differences are observed in the order of toxicity going from the methyl- to the butyl ethers.

Glycols exert a high oxygen demand for decomposition and once released to the environments cause the death of aquatic organisms if dissolved oxygen is depleted.

The isothiazolinones are very toxic to marine organisms (fish, *Daphnia magna* and algae)

The high water solubility and low log Kow values of several chlorinated and non-chlorinated indicate a low potential for bioaccumulation.

Studies of 5-chloro-2-methyl-4-isothiazolin-3-one (CMI) in bluegill sunfish (*Lepomis macrochirus*) show BCF values of 102, 114 and 67 at nominal concentrations of 0.02, 0.12 and 0.8 mg/l. The BCF for 2-methyl-4-isothiazolin-3-one (MI) was determined at 2.3 at a nominal concentration of 0.12 mg/l

Primary biodegradation of MI and CMI occurred with half-lives of less than 24 hours in aerobic and anoxic sediments, and within a period of less than one week the parent compounds were depleted to very low levels that could not be clearly distinguished from analytical artifacts. The ultimate aerobic biodegradability of both MI and CMI attained levels of > 55% within 29 days.

Furthermore, the proposed metabolites of MI and CMI are considered to have a low aquatic toxicity on the basis of QSAR estimates and the measured toxicity of the structurally related N-(n-octyl) malonamic acid.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
ethylene glycol monobutyl ether	LOW (Half-life = 56 days)	LOW (Half-life = 1.37 days)
diethylene glycol monobutyl ether	LOW	LOW
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH
2-octyl-4-isothiazolin-3-one	HIGH	HIGH
2,2'-bipyridyl	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
diethylene glycol monobutyl ether	LOW (BCF = 0.46)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)
2,2'-bipyridyl	LOW (BCF = 0.7)

Mobility in soil

Ingredient	Mobility
ethylene glycol monobutyl ether	HIGH (KOC = 1)
diethylene glycol monobutyl ether	LOW (KOC = 10)
3-iodo-2-propynyl butyl carbamate	LOW (KOC = 365.3)
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)
2,2'-bipyridyl	LOW (KOC = 248.7)

SECTION 13 DISPOSAL CONSIDERATIONS

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.
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SECTION 14 TRANSPORT INFORMATION**Labels Required**

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture****ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS**

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

LITHIUM TETRABORATE(12007-60-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

DIETHYLENE GLYCOL MONOBUTYL ETHER(112-34-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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3-IODO-2-PROPYNYL BUTYL CARBAMATE(55406-53-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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2-OCTYL-4-ISOTHIAZOLIN-3-ONE(26530-20-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Substances Information System - Consolidated Lists	Australia Inventory of Chemical Substances (AICS)
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EUCALYPTUS OIL(92502-70-0) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

ZIRCONIUM 2-ETHYLHEXANOATE(22464-99-9) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Exposure Standards	Australia Inventory of Chemical Substances (AICS)
Australia Hazardous Substances Information System - Consolidated Lists	

2,2'-BIPYRIDYL(366-18-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y

Canada - NDSL	N (lithium tetraborate; 3-iodo-2-propynyl butyl carbamate; diethylene glycol monobutyl ether; 2-octyl-4-isothiazolin-3-one; eucalyptus oil; zirconium 2-ethylhexanoate; 2,2'-bipyridyl; ethylene glycol monobutyl ether)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	N (lithium tetraborate; diethylene glycol monobutyl ether; eucalyptus oil; zirconium 2-ethylhexanoate; 2,2'-bipyridyl)
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing (see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

Ingredients with multiple cas numbers

Name	CAS No
lithium tetraborate	12007-60-2, 1303-94-2
eucalyptus oil	92502-70-0, 84625-32-1, 8000-48-4, 91771-68-5, 85203-56-1

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC – TWA: Permissible Concentration-Time Weighted Average
 PC – STEL: Permissible Concentration-Short Term Exposure Limit
 IARC: International Agency for Research on Cancer
 ACGIH: American Conference of Governmental Industrial Hygienists
 STEL: Short Term Exposure Limit
 TEEL: Temporary Emergency Exposure Limit,
 IDLH: Immediately Dangerous to Life or Health Concentrations
 OSF: Odour Safety Factor
 NOAEL :No Observed Adverse Effect Level
 LOAEL: Lowest Observed Adverse Effect Level
 TLV: Threshold Limit Value
 LOD: Limit Of Detection
 OTV: Odour Threshold Value
 BCF: BioConcentration Factors
 BEI: Biological Exposure Index

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