

Mirotone TIMBRE PLUS INTERIOR

Mirotone (Mirotone (NZ))

Chemwatch Hazard Alert Code: 2

Chemwatch: 84-3561
Version No: 2.1.1.1
Safety Data Sheet according to HSNO Regulations

Issue Date: 18/08/2017
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L.GHS.NZLEN

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

Product Identifier

Product name	Mirotone TIMBRE PLUS INTERIOR
Synonyms	TIMBRE PLUS INT 4330, Product Code: 4330
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. Interior timber Ideal for wall panelling, furniture, doors, joinery and other interior woodwork.
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Details of the supplier of the safety data sheet

Registered company name	Mirotone (Mirotone (NZ))	Mirotone	Quantum Timber Finishes
Address	32 Cryers Road East Tamaki, Manukau Auckland 2163 New Zealand	21 Marigold Street Revesby NSW 2212 Australia	147 Ordish Road Dandenong VIC 3175 Australia
Telephone	0800 FINISH (0800 34 64 74)	+61 2 9795 3700	+61 2 9795 3700
Fax	0800 34 64 34	+61 2 9771 3601	1300 132 202
Website	www.mirotone.co.nz	www.mirotone.com, www.polycure.com.au	www.qtf.com.au
Email	Not Available	Not Available	Not Available

Emergency telephone number

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

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Considered a Hazardous Substance according to the criteria of the New Zealand Hazardous Substances New Organisms legislation. Not regulated for transport of Dangerous Goods.


CHEMWATCH HAZARD RATINGS

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

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

Classification [1]	Acute Toxicity (Oral) Category 5, Acute Toxicity (Inhalation) Category 5, Skin Corrosion/Irritation Category 3, Skin Sensitizer Category 1
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1E (inhalation), 6.1E (oral), 6.3B, 6.5B (contact)

Label elements

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

H303	May be harmful if swallowed.
H333	May be harmful if inhaled

Continued...

H316	Causes mild skin irritation
H317	May cause an allergic skin reaction.

Supplementary statement(s)

Not Applicable

CLP classification (additional)

Not Applicable

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P304+P312	IF INHALED: Call a POISON CENTER or doctor/physician if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

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
9043-30-5	<0.5	<u>isotridecyl alcohol, ethoxylated</u>
55406-53-6	<0.2	<u>3-iodo-2-propynyl butyl carbamate</u>
41556-26-7	<0.2	<u>bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate</u>
104810-47-1	<0.2	<u>Tinuvin 1130</u>
104810-48-2	<0.1	<u>Tinuvin 213</u>
82919-37-7	<0.1	<u>methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate</u>
Not Available	<0.1	isothiazolinone
22464-99-9	<0.1	<u>zirconium 2-ethylhexanoate</u>
7732-18-5	75.662	<u>water</u>

SECTION 4 FIRST AID MEASURES

NZ Poisons Centre 0800 POISON (0800 764 766) | NZ Emergency Services: 111

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 or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
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.

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) , nitrogen oxides (NO_x) , other pyrolysis products typical of burning organic material. <p>May emit poisonous fumes.</p>

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>Moderate hazard.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Stop leak if safe to do so. ▶ Contain spill with sand, earth or vermiculite. ▶ Collect recoverable product into labelled containers for recycling. ▶ Neutralise/decontaminate residue (see Section 13 for specific agent). ▶ Collect solid residues and seal in labelled drums for disposal. ▶ Wash area and prevent runoff into drains. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▶ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE**Precautions for safe handling**

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin
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	<ul style="list-style-type: none"> ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ 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. ▶ 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
New Zealand Workplace Exposure Standards (WES)	zirconium 2-ethylhexanoate	Zirconium and compounds, as Zr	5 mg/m3	Not Available	10 ppm	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
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
isotridecyl alcohol, ethoxylated	Not Available	Not Available
3-iodo-2-propynyl butyl carbamate	Not Available	Not Available
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	Not Available	Not Available
Tinuvin 1130	Not Available	Not Available
Tinuvin 213	Not Available	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	Not Available	Not Available
isothiazolinone	Not Available	Not Available
zirconium 2-ethylhexanoate	500 mg/m3	25 mg/m3
water	Not Available	Not Available

MATERIAL DATA

for polyethylene glycols:

For powdered forms:

The polyethylene glycols are extremely low in oral toxicity, are not significantly irritating to the eyes or skin, and are not absorbed through the skin in toxic amounts. vapour pressures are extremely low and inhalation exposure is limited to mists. Based on experimental data and human experience, these substances do not present significant hazards to health in the workplace.

For trimethyl benzene as mixed isomers (of unstated proportions)

Odour Threshold Value: 2.4 ppm (detection)

Use care in interpreting effects as a single isomer or other isomer mix. Trimethylbenzene is an eye, nose and respiratory irritant. High concentrations cause central nervous system depression.

Exposed workers show CNS changes, asthmatic bronchitis and blood dyscrasias at 60 ppm. The TLV-TWA is thought to be protective against the significant risk of CNS excitation, asthmatic bronchitis and blood dyscrasias associated with exposures above the limit.

Odour Safety Factor (OSF)

OSF=10 (1,2,4-TRIMETHYLBENZENE)

for dipropylene glycol monomethyl ether:

The TLV-TWA and STEL recommendations were thought to be sufficiently low to prevent objectionable irritation and provide a considerable safety factor against CNS impairment. In view of the large dose required to cause weight loss and narcosis in rabbits the skin notation is being reviewed.

Probable minimum concentration that may cause minor nasal irritation is about 35 ppm.

Probable minimum concentration that may cause tolerable eye, throat, and respiratory irritation is about 75 ppm.

Lowest concentration at which vapour is rated tolerable 80 ppm.

Based on these criteria it is possible that an occasional person may find the vapour of dipropylene glycol monomethyl ether intolerable at the recommended 100 ppm TLV.

Dermal absorption of the substance under specific experimental conditions led to narcotic effects and consequent deaths. However, only slight narcotic effects were seen after several hours exposure of rats to

aerosols which wet the fur of animals. Rabbits tolerated dermal application of 3.0 ml/kg per day without effects. A skin designation is thought to be unnecessary by the MAK committee, in contrast with others.

for propylene glycol monomethyl ether (PGME)

Odour Threshold: 10 ppm.

The TLV-TWA is protective against discomfort caused by odour, against eye and skin irritation, and chronic effects (including possible liver and kidney damage).

Individuals exposed to 100 ppm reported a transient unpleasant odour with slight eye irritation after about 1 or 2 hours. At 300 ppm, mild irritation of the eyes and nose developed within 5 minutes;

some individuals found the irritation hardly bearable after about an hour. A concentration of 750 ppm was highly irritating. Signs of central nervous system depression developed at 1000 ppm.

Neurological, clinical chemical and general medical examinations showed no other conspicuous toxicity.

Concentrations of the beta-isomer, 2-methoxy-1-propyl acetate are low in commercial grades of PGME and teratogenic effects associated with this isomer are expected to be absent.

Odour Safety Factor(OSF)

OSF=10 (propylene glycol monomethyl ether)

Exposed individuals are **NOT** reasonably expected to be warned, by smell, that the Exposure Standard is being exceeded.

Odour Safety Factor (OSF) is determined to fall into either Class C, D or E.

The Odour Safety Factor (OSF) is defined as:

OSF= Exposure Standard (TWA) ppm/ Odour Threshold Value (OTV) ppm

Classification into classes follows:

Class OSF Description

A 550 Over 90% of exposed individuals are aware by smell that the Exposure Standard (TLV-TWA for example) is being reached, even when distracted by working activities

B 26-550 As "A" for 50-90% of persons being distracted

C 1-26 As "A" for less than 50% of persons being distracted

D 0.18-1 10-50% of persons aware of being tested perceive by smell that the Exposure Standard is being reached

E <0.18 As "D" for less than 10% of persons aware of being tested

For ethylene glycol monobutyl ether (2-butoxyethanol)

Odour Threshold Value: 0.10 ppm (detection), 0.35 ppm (recognition)

Although rats appear to be more susceptible than other animals anaemia is not uncommon amongst humans following exposure. The TLV reflects the need to maintain exposures below levels found to cause blood changes in experimental animals. It is concluded that this limit will reduce the significant risk of irritation, haematologic effects and other systemic effects observed in humans and animals exposed to higher vapour concentrations. The toxic effects typical of some other glycol ethers (pancytopenia, testis atrophy and teratogenic effects) are not found with this substance.

Odour Safety Factor (OSF)

OSF=2E2 (2-BUTOXYETHANOL)

For diethylene glycol monobutyl ether:

CEL TWA: 15.5 ppm, 100 mg/m³

(CEL = Chemwatch Exposure Limit)

In studies involving the inhalation toxicity of diethylene glycol monobutyl ether, exposure for 6 hours daily at 100 mg/m³ had no effect. This concentration is in the range of the saturated vapour concentration.

Local damage was produced following inhalation of concentrations higher than the saturated vapour concentrations, that is, during inhalation of the aerosol (350 mg/m³). Since the only potential effects of inhalation are restricted to local discomfort (in the aerosol concentration range) the substance is classified in category I for the limitation of exposure peaks.

Teratogenicity studies have not revealed prenatal toxic effects at high oral doses and this ether is classified in pregnancy risk group C.

Exposure controls

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

The basic types of engineering controls are:

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

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

Employers may need to use multiple types of controls to prevent employee overexposure.

Local exhaust ventilation usually required. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection.

Supplied-air type respirator may be required in special circumstances. Correct fit is essential to ensure adequate protection.

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


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

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square

	of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.
Personal protection	
Eye and face protection	<ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ Chemical goggles. ▶ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
Skin protection	See Hand protection below
Hands/feet protection	<ul style="list-style-type: none"> ▶ Wear chemical protective gloves, e.g. PVC. ▶ Wear safety footwear or safety gumboots, e.g. Rubber <p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed 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>
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
NEOPRENE	C
NITRILE	C
PE/EVAL/PE	C

Respiratory protection

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

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

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

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

^ - Full-face

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PVA	C
PVC	C
SARANEX-23	C
VITON	C
##ethylene glycol monobutyl	ether
##propylene glycol monomethyl ether - alpha	isomer

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.

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Note that all of the monopropylene glycol ethers may exist in two isomeric forms, alpha or beta. The alpha form, which is thermodynamically favored during synthesis, consists of a secondary alcohol configuration. The beta form consists of a primary alcohol. The two isomeric forms are shown above. The di- and tripropylene glycol ethers may form up to 4 and 8 isomeric forms, respectively. Even so, all isomers exhibit either the "alpha" or "beta" configuration, existing as secondary or primary alcohols, respectively. The distribution of isomeric forms for the di- and tripropylene glycols, as with the mono-PGEs, also results in predominantly the alpha form (i.e., a secondary alcohol). It should be noted that only the alpha isomer and isomeric mixtures (consisting predominantly of the alpha isomer) are produced commercially; the purified beta isomer is not produced at this time. Milky to coloured low viscosity non-flammable liquid with a slight odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.97-1.07
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8-9	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	430-490 @25C
Initial boiling point and boiling range (°C)	101 (initial)	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Applicable	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)	79-88
Vapour pressure (kPa)	2.3 @25C	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	0.6	VOC g/L	21-24

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	Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo. Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
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Ingestion	Accidental ingestion of the material may be damaging to the health of the individual. Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.
Skin Contact	The material is not thought to produce adverse health effects or skin irritation following contact (as classified by EC Directives using animal models). Nevertheless, good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	Although the liquid is not thought to be an irritant (as classified by EC Directives), direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
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. Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

Mirotone TIMBRE PLUS INTERIOR	TOXICITY	IRRITATION
	Not Available	Not Available
isotridecyl alcohol, ethoxylated	TOXICITY	IRRITATION
	Not Available	Not Available
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
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	TOXICITY	IRRITATION
	Oral (rat) LD50: 3100 mg/kg ^[2]	Not Available
Tinuvin 1130	TOXICITY	IRRITATION
	Not Available	Not Available
Tinuvin 213	TOXICITY	IRRITATION
	Not Available	Skin (guinea pig): Strong sensit. Skin (rabbit): non-irritant
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	TOXICITY	IRRITATION
	Not Available	Not Available
zirconium 2-ethylhexanoate	TOXICITY	IRRITATION
	Not Available	Not Available
water	TOXICITY	IRRITATION
	Not Available	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

Mirotone TIMBRE PLUS INTERIOR	<p>for propylene glycol ethers (PGEs): Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM). Testing of a wide variety of propylene glycol ethers Testing of a wide variety of propylene glycol ethers has shown that propylene glycol-based ethers are less toxic than some ethers of the ethylene series. The common toxicities associated with the lower molecular weight homologues of the ethylene series, such as adverse effects on reproductive organs, the developing embryo and fetus, blood (haemolytic effects), or thymus, are not seen with the commercial-grade propylene glycol ethers. In the ethylene series, metabolism of the terminal hydroxyl group produces an alkoxyacetic acid. The reproductive and developmental toxicities of the lower molecular weight homologues in the ethylene series are due specifically to the formation of methoxyacetic and ethoxyacetic acids. Longer chain length homologues in the ethylene series are not associated with the reproductive toxicity but can cause haemolysis in sensitive species, also through formation of an alkoxyacetic acid. The predominant alpha isomer of all the PGEs (thermodynamically favored during manufacture of PGEs) is a secondary alcohol incapable of forming an alkoxypropionic acid. In contrast beta-isomers are able to form the alkoxypropionic acids and these are linked to teratogenic effects (and possibly haemolytic effects). This alpha isomer comprises greater than 95% of the isomeric mixture in the commercial product. Because the alpha isomer cannot form an alkoxypropionic acid, this is the most likely reason for the lack of toxicity shown by the PGEs as distinct from the lower molecular weight ethylene glycol ethers. More importantly, however, very extensive empirical test data show that this class of commercial-grade glycol ether presents a low toxicity hazard. PGEs, whether mono, di- or tripropylene glycol-based (and no matter what the alcohol group), show a very similar pattern of low to non-detectable toxicity of any type at doses or exposure levels greatly exceeding those showing pronounced effects from the ethylene series. One of the primary metabolites of the propylene glycol ethers is propylene glycol, which is of low toxicity and completely metabolised in the body. As a class, the propylene glycol ethers are rapidly absorbed and distributed throughout the body when introduced by inhalation or oral exposure. Dermal absorption is somewhat slower but subsequent distribution is rapid. Most excretion for PGEs is via the urine and expired air. A small portion is excreted in the faeces. As a group PGEs exhibits low acute toxicity by the oral, dermal, and inhalation routes. Rat oral LD50s range from >3,000 mg/kg (PnB) to >5,000 mg/kg (DPMA). Dermal LD50s are all > 2,000 mg/kg (PnB, & DPnB; where no deaths occurred), and ranging up to >15,000 mg/kg (TPM). Inhalation LC50 values were higher than 5,000 mg/m³ for DPMA (4-hour exposure), and TPM (1-hour exposure). For DPnB the 4-hour LC50 is >2,040 mg/m³. For PnB, the 4-hour LC50 was >651 ppm (>3,412 mg/m³), representing the highest practically attainable vapor level. No deaths occurred at these concentrations. PnB and TPM are moderately irritating to eyes while the remaining category members are only slightly irritating to nonirritating. PnB is moderately irritating to skin while the</p>
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remaining category members are slightly to non-irritating
None are skin sensitizers.

In repeated dose studies ranging in duration from 2 to 13 weeks, few adverse effects were found even at high exposure levels and effects that did occur were mild in nature. By the oral route of administration, NOAELs of 350 mg/kg-d (PnB – 13 wk) and 450 mg/kg-d (DPnB – 13 wk) were observed for liver and kidney weight increases (without accompanying histopathology). LOAELs for these two chemicals were 1000 mg/kg-d (highest dose tested).

Dermal repeated-dose toxicity tests have been performed for many PGEs. For PnB, no effects were seen in a 13-wk study at doses as high as 1,000 mg/kg-d. A dose of 273 mg/kg-d constituted a LOAEL (increased organ weights without histopathology) in a 13-week dermal study for DPnB. For TPM, increased kidney weights (no histopathology) and transiently decreased body weights were found at a dose of 2,895 mg/kg-d in a 90-day study in rabbits. By inhalation, no effects were observed in 2-week studies in rats at the highest tested concentrations of 3244 mg/m³ (600 ppm) for PnB and 2,010 mg/m³ (260 ppm) for DPnB. TPM caused increased liver weights without histopathology by inhalation in a 2-week study at a LOAEL of 360 mg/m³ (43 ppm). In this study, the highest tested TPM concentration, 1010 mg/m³ (120 ppm), also caused increased liver weights without accompanying histopathology. Although no repeated-dose studies are available for the oral route for TPM, or for any route for DPMA, it is anticipated that these chemicals would behave similarly to other category members.

One and two-generation reproductive toxicity testing has been conducted in mice, rats, and rabbits via the oral or inhalation routes of exposure on PM and PMA. In an inhalation rat study using PM, the NOAEL for parental toxicity is 300 ppm (1106 mg/m³) with decreases in body and organ weights occurring at the LOAEL of 1000 ppm (3686 mg/m³). For offspring toxicity the NOAEL is 1000 ppm (3686 mg/m³), with decreased body weights occurring at 3000 ppm (11058 mg/m³). For PMA, the NOAEL for parental and offspring toxicity is 1000 mg/kg/d. In a two generation gavage study in rats. No adverse effects were found on reproductive organs, fertility rates, or other indices commonly monitored in such studies. In addition, there is no evidence from histopathological data from repeated-dose studies for the category members that would indicate that these chemicals would pose a reproductive hazard to human health.

In developmental toxicity studies many PGEs have been tested by various routes of exposure and in various species at significant exposure levels and show no frank developmental effects. Due to the rapid hydrolysis of DPMA to DPM, DPMA would not be expected to show teratogenic effects. At high doses where maternal toxicity occurs (e.g., significant body weight loss), an increased incidence of some anomalies such as delayed skeletal ossification or increased 13th ribs, have been reported. Commercially available PGEs showed no teratogenicity.

The weight of the evidence indicates that propylene glycol ethers are not likely to be genotoxic. *In vitro*, negative results have been seen in a number of assays for PnB, DPnB, DPMA and TPM. Positive results were only seen in 3 out of 5 chromosome aberration assays in mammalian cells with DPnB. However, negative results were seen in a mouse micronucleus assay with DPnB and PM. Thus, there is no evidence to suggest these PGEs would be genotoxic *in vivo*. In a 2-year bioassay on PM, there were no statistically significant increases in tumors in rats and mice.

For trimethylbenzenes:

Absorption of 1,2,4-trimethylbenzene occurs after oral, inhalation, or dermal exposure. Occupationally, inhalation and dermal exposures are the most important routes of absorption although systemic intoxication from dermal absorption is not likely to occur due to the dermal irritation caused by the chemical prompting quick removal. Following oral administration of the chemical to rats, 62.6% of the dose was recovered as urinary metabolites indicating substantial absorption. 1,2,4-Trimethylbenzene is lipophilic and may accumulate in fat and fatty tissues. In the blood stream, approximately 85% of the chemical is bound to red blood cells. Metabolism occurs by side-chain oxidation to form alcohols and carboxylic acids which are then conjugated with glucuronic acid, glycine, or sulfates for urinary excretion. After a single oral dose to rats of 1200 mg/kg, urinary metabolites consisted of approximately 43.2% glycine, 6.6% glucuronic, and 12.9% sulfuric acid conjugates. The two principle metabolites excreted by rabbits after oral administration of 438 mg/kg/day for 5 days were 2,4-dimethylbenzoic acid and 3,4-dimethylhippuric acid. The major routes of excretion of 1,2,4-trimethylbenzene are exhalation of parent compound and elimination of urinary metabolites. Half-times for urinary metabolites were reported as 9.5 hours for glycine, 22.9 hours for glucuronide, and 37.6 hours for sulfuric acid conjugates.

Acute Toxicity Direct contact with liquid 1,2,4-trimethylbenzene is irritating to the skin and breathing the vapor is irritating to the respiratory tract causing pneumonitis. Breathing high concentrations of the chemical vapor causes headache, fatigue, and drowsiness. In humans liquid 1,2,4-trimethylbenzene is irritating to the skin and inhalation of vapor causes chemical pneumonitis. High concentrations of vapor (5000-9000 ppm) cause headache, fatigue, and drowsiness. The concentration of 5000 ppm is roughly equivalent to a total of 221 mg/kg assuming a 30 minute exposure period (see end note 1). 2. Animals - Mice exposed to 8130-9140 ppm 1,2,4-trimethylbenzene (no duration given) had loss of righting response and loss of reflexes. Direct dermal contact with the chemical (no species given) causes vasodilation, erythema, and irritation (U.S. EPA). Seven of 10 rats died after an oral dose of 2.5 mL of a mixture of trimethylbenzenes in olive oil (average dose approximately 4.4 g/kg). Rats and mice were exposed by inhalation to a coal tar distillate containing about 70% 1,3,5- and 1,2,4-trimethylbenzene; no pathological changes were noted in either species after exposure to 1800-2000 ppm for up to 48 continuous hours, or in rats after 14 exposures of 8 hours each at the same exposure levels. No effects were reported for rats exposed to a mixture of trimethylbenzenes at 1700 ppm for 10 to 21 days.

Neurotoxicity 1,2,4-Trimethylbenzene depresses the central nervous system. Exposure to solvent mixtures containing the chemical causes headache, fatigue, nervousness, and drowsiness. Occupationally, workers exposed to a solvent containing 50% 1,2,4-trimethylbenzene had nervousness, headaches, drowsiness, and vertigo (U.S. EPA). Headache, fatigue, and drowsiness were reported for workers exposed (no dose given) to paint thinner containing 80% 1,2,4- and 1,3,5-trimethylbenzenes.

Results of the developmental toxicity study indicate that the C9 fraction caused adverse neurological effects at the highest dose (1500 ppm) tested.

Subchronic/Chronic Toxicity Long-term exposure to solvents containing 1,2,4-trimethylbenzene may cause nervousness, tension, and bronchitis. Painters that worked for several years with a solvent containing 50% 1,2,4- and 30% 1,3,5-trimethylbenzene showed nervousness, tension and anxiety, asthmatic bronchitis, anemia, and alterations in blood clotting; haematological effects may have been due to trace amounts of benzene.

Rats given 1,2,4-trimethylbenzene orally at doses of 0.5 or 2.0 g/kg/day, 5 days/week for 4 weeks. All rats exposed to the high dose died and 1 rat in the low dose died (no times given); no other effects were reported. Rats exposed by inhalation to 1700 ppm of a trimethylbenzene isomeric mixture for 4 months had decreased weight gain, lymphopenia and neutrophilia.

Genotoxicity: Results of mutagenicity testing, indicate that the C9 fraction does not induce gene mutations in prokaryotes (Salmonella typhimurium/mammalian microsome assay); or in mammalian cells in culture (in Chinese hamster ovary cells with and without activation). The C9 fraction does not induce chromosome mutations in Chinese hamster ovary cells with and without activation; does not induce chromosome aberrations in the bone marrow of Sprague-Dawley rats exposed by inhalation (6 hours/day for 5 days); and does not induce sister chromatid exchange in Chinese hamster ovary cells with and without activation.

Developmental/Reproductive Toxicity: A three-generation reproductive study on the C9 fraction was conducted. CD rats (30/sex/group) were exposed by inhalation to the C9 fraction at concentrations of 0, 100, 500, or 1500 ppm (0, 100, 500, or 1500 mg/kg/day) for 6 hours/day, 5 days/week. There was evidence of parental and reproductive toxicity at all dose levels. Indicators of parental toxicity included reduced body weights, increased salivation, hunched posture, aggressive behavior, and death. Indicators of adverse reproductive system effects included reduced litter size and reduced pup body weight. The LOEL was 100 ppm; a no-observed-effect level was not established. Developmental toxicity, including possible developmental neurotoxicity, was evident in rats in a 3-generation reproductive study.

No effects on fecundity or fertility occurred in rats treated dermally with up to 0.3 mL/rat/day of a mixture of trimethylbenzenes, 4-6 hours/day, 5 days/week over one generation.

For C9 aromatics (typically trimethylbenzenes - TMBs)

Acute Toxicity

Acute toxicity studies (oral, dermal and inhalation routes of exposure) have been conducted in rats using various solvent products containing predominantly mixed C9 aromatic hydrocarbons (CAS RN 64742-95-6). Inhalation LC50's range from 6,000 to 10,000 mg/m³ for C9 aromatic naphtha and 18,000 to 24,000 mg/m³ for 1,2,4 and 1,3,5-TMB, respectively. A rat oral LD50 reported for 1,2,4-TMB is 5 grams/kg bw and a rat dermal LD50 for the C9 aromatic naphtha is >4 mL/kg bw. These data indicate that C9 aromatic solvents show that LD50/LC50 values are greater than limit doses for acute toxicity studies established under OECD test guidelines.

Irritation and Sensitization

Several irritation studies, including skin, eye, and lung/respiratory system, have been conducted on members of the category. The results indicate that C9 aromatic hydrocarbon solvents are mildly to moderately irritating to the skin, minimally irritating to the eye, and have the potential to irritate the respiratory tract and cause depression of respiratory rates in mice. Respiratory irritation is a key endpoint in the current occupational exposure limits established for C9 aromatic hydrocarbon solvents and trimethylbenzenes. No evidence of skin sensitization was identified.

Repeated Dose Toxicity

Inhalation: The results from a subchronic (3 month) neurotoxicity study and a one-year chronic study (6 hr/day, 5 days/week) indicate that effects from inhalation exposure to C9 Aromatic Hydrocarbon Solvents on systemic toxicity are slight. A battery of neurotoxicity and neurobehavioral endpoints were evaluated in the 3-month inhalation study on C9 aromatic naphtha tested at concentrations of 0, 101, 452, or 1320 ppm (0, 500, 2,220, or 6,500 mg/m³). In this study, other than a transient weight reduction in the high exposure group (not statistically significant at termination of exposures), no effects were reported on neuropathology or neurobehavioral parameters. The NOAEL for systemic and/or neurotoxicity was 6,500 mg/m³, the highest concentration tested. In an inhalation study of a

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commercial blend, rats were exposed to C9 aromatic naphtha concentrations of 0, 96, 198, or 373 ppm (0, 470, 970, 1830 mg/m³) for 6 hr/day, 5 days/week, for 12 months. Liver and kidney weights were increased in the high exposure group but no accompanying histopathology was observed in these organs.

The NOAEL was considered to be the high exposure level of 373 ppm, or 1830 mg/m³. In two subchronic rat inhalation studies, both of three months duration, rats were exposed to the individual TMB isomers (1,2,4- and 1,3,5-) to nominal concentrations of 0, 25, 100, or 250 ppm (0, 123, 492, or 1230 mg/m³).

Respiratory irritation was observed at 492 (100 ppm) and 1230 mg/m³ (250 ppm) and no systemic toxicity was observed in either study. For both pure isomers, the NOELs are 25 ppm or 123 mg/m³ for respiratory irritation and 250 ppm or 1230 mg/m³ for systemic effects.

Oral: The C9 aromatic naphtha has not been tested via the oral route of exposure. Individual TMB isomers have been evaluated in a series of repeated-dose oral studies ranging from 14 days to 3 months over a wide range of doses. The effects observed in these studies included increased liver and kidney weights, changes in blood chemistry, increased salivation, and decreased weight gain at higher doses. Organ weight changes appeared to be adaptive as they were not accompanied by histopathological effects. Blood changes appeared sporadic and without pattern. One study reported hyaline droplet nephropathy in male rats at the highest dose (1000 mg/kg bw-day), an effect that is often associated with alpha-2mu-globulin-induced nephropathy and not considered relevant to humans. The doses at which effects were detected were 100 mg/kg-bw day or above (an exception was the pilot 14 day oral study - LOAEL 150 mg/kg bw-day - but the follow up three month study had a LOAEL of 600 mg/kg-bw-day with a NOAEL of 200 mg/kg bw-day). Since effects generally were not severe and could be considered adaptive or spurious, oral exposure does not appear to pose a high toxicity hazard for pure trimethylbenzene isomers.

Mutagenicity

In vitro genotoxicity testing of a variety of C9 aromatics has been conducted in both bacterial and mammalian cells. In vitro point mutation tests were conducted with *Salmonella typhimurium* and *Escherichia coli* bacterial strains, as well as with cultured mammalian cells such as the Chinese hamster cell ovary cells (HGPRT assay) with and without metabolic activation. In addition, several types of in vitro chromosomal aberration tests have been performed (chromosome aberration frequency in Chinese hamster ovary and lung cells, sister chromatid exchange in CHO cells). Results were negative both with and without metabolic activation for all category members. For the supporting chemical 1,2,3-TMB, a single in vitro chromosome aberration test was weakly positive. In vivo bone marrow cytogenetics test, rats were exposed to C9 aromatic naphtha at concentrations of 0, 153, 471, or 1540 ppm (0, 750, 2,310, or 7,560 mg/m³) 6 hr/day, for 5 days. No evidence of in vivo somatic cell genotoxicity was detected. Based on the cumulative results of these assays, genetic toxicity is unlikely for substances in the C9 Aromatic Hydrocarbon Solvents Category

Reproductive and Developmental Toxicity

Results from the three-generation reproduction inhalation study in rats indicate limited effects from C9 aromatic naphtha. In each of three generations (F0, F1 and F2), rats were exposed to High Flash Aromatic Naphtha (CAS RN 64742-95-6) via whole body inhalation at target concentrations of 0, 100, 500, or 1500 ppm (actual mean concentrations throughout the full study period were 0, 103, 495, or 1480 ppm, equivalent to 0, 505, 2430, or 7265 mg/m³, respectively). In each generation, both sexes were exposed for 10 weeks prior to and two weeks during mating for 6 hrs/day, 5 days/wks. Female rats in the F0, F1, and F2 generation were then exposed during gestation days 0-20 and lactation days 5-21 for 6 hrs/day, 7 days/wk. The age at exposure initiation differed among generations; F0 rats were exposed starting at 9 weeks of age, F1 exposure began at 5-7 weeks, and F2 exposure began at postnatal day (PND) 22. In the F0 and F1 parental generations, 30 rats/sex/group were exposed and mated. However, in the F2 generation, 40/sex/group were initially exposed due to concerns for toxicity, and 30/sex/group were randomly selected for mating, except that all survivors were used at 1480 ppm. F3 litters were not exposed directly and were sacrificed on lactation day 21.

Systemic Effects on Parental Generations:

The F0 males showed statistically and biologically significantly decreased mean body weight by ~15% at 1480 ppm when compared with controls. Seven females died or were sacrificed in extremis at 1480 ppm. The F0 female rats in the 495 ppm exposed group had a 13% decrease in body weight gain when adjusted for initial body weight when compared to controls. The F1 parents at 1480 ppm had statistically significantly decreased mean body weights (by ~13% (females) and 22% (males)), and locomotor activity. F1 parents at 1480 ppm had increased ataxia and mortality (six females). Most F2 parents (70/80) exposed to 1480 ppm died within the first week. The remaining animals survived throughout the rest of the exposure period. At week 4 and continuing through the study, F2 parents at 1480 ppm had statistically significant mean body weights much lower than controls (~33% for males; ~28% for females); body weights at 495 ppm were also reduced significantly (by 13% in males and 15% in females). The male rats in the 495 ppm exposed group had a 12% decrease in body weight gain when adjusted for initial body weight when compared to controls. Based on reduced body weight observed, the overall systemic toxicity LOAEC is 495 ppm (2430 mg/m³).

Reproductive Toxicity-Effects on Parental Generations: There were no pathological changes noted in the reproductive organs of any animal of the F0, F1, or F2 generation. No effects were reported on sperm morphology, gestational period, number of implantation sites, or post-implantation loss in any generation. Also, there were no statistically or biologically significant differences in any of the reproductive parameters, including: number of mated females, copulatory index, copulatory interval, number of females delivering a litter, number of females delivering a live litter, or male fertility in the F0 or in the F2 generation. Male fertility was statistically significantly reduced at 1480 ppm in the F1 rats. However, male fertility was not affected in the F0 or in the F2 generations; therefore, the biological significance of this change is unknown and may or may not be attributed to the test substance. No reproductive effects were observed in the F0 or F1 dams exposed to 1480 ppm (7265 mg/m³). Due to excessive mortality at the highest concentration (1480 ppm, only six dams available) in the F2 generation, a complete evaluation is precluded. However, no clear signs of reproductive toxicity were observed in the F2 generation. Therefore, the reproductive NOAEC is considered 495 ppm (2430 mg/m³), which excludes analysis of the highest concentration due to excessive mortality.

Developmental Toxicity - Effects on Pups: Because of significant maternal toxicity (including mortality) in dams in all generations at the highest concentration (1480 ppm), effects in offspring at 1480 ppm are not reported here. No significant effects were observed in the F1 and F2 generation offspring at 103 or 495 ppm. However, in F3 offspring, body weights and body weight gain were reduced by ~10-11% compared with controls at 495 ppm for approximately a week (PND 14 through 21). Maternal body weight was also depressed by ~12% throughout the gestational period compared with controls. The overall developmental LOAEC from this study is 495 ppm (2430 mg/m³) based on the body weights reductions observed in the F3 offspring.

Conclusion: No effects on reproductive parameters were observed at any exposure concentration, although a confident assessment of the group exposed at the highest concentration was not possible. A potential developmental effect (reduction in mean pup weight and weight gain) was observed at a concentration that was also associated with maternal toxicity.

Polyethers, for example, ethoxylated surfactants and polyethylene glycols, are highly susceptible towards air oxidation as the ether oxygens will stabilize intermediary radicals involved. Investigations of a chemically well-defined alcohol (pentaethylene glycol mono-n-dodecyl ether) ethoxylate, showed that polyethers form complex mixtures of oxidation products when exposed to air.

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

On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

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

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

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

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

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On the basis of the lower irritancy, nonionic surfactants are often preferred to ionic surfactants in topical products. However, their susceptibility towards autoxidation also increases the irritation. Because of their irritating effect, it is difficult to diagnose ACD to these compounds by patch testing.

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

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

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

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

>20 EO is not classified (CESIO 2000)

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

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

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

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

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

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

For high boiling ethylene glycol ethers (typically triethylene- and tetraethylene glycol ethers):

Skin absorption: Available skin absorption data for triethylene glycol ether (TGBE), triethylene glycol methyl ether (TGME), and triethylene glycol ethylene ether (TGEE) suggest that the rate of absorption in skin of these three glycol ethers is 22 to 34 micrograms/cm²/hr, with the methyl ether having the highest permeation constant and the butyl ether having the lowest. The rates of absorption of TGBE, TGEE and TGME are at least 100-fold less than EGME, EGEE, and EGBE, their ethylene glycol monoalkyl ether counterparts, which have absorption rates that range from 214 to 2890 micrograms/cm²/hr. Therefore, an increase in either the chain length of the alkyl substituent or the number of ethylene glycol moieties appears to lead to a decreased rate of percutaneous absorption. However, since the ratio of the change in values of the ethylene glycol to the diethylene glycol series is larger than that of the diethylene glycol to triethylene glycol series, the effect of the length of the chain and number of ethylene glycol moieties on absorption diminishes with an increased number of ethylene glycol moieties. Therefore, although tetraethylene glycol methyl ether (TetraME) and tetraethylene glycol butyl ether (TetraBE) are expected to be less permeable to skin than TGME and TGBE, the differences in permeation between these molecules may only be slight.

Metabolism: The main metabolic pathway for metabolism of ethylene glycol monoalkyl ethers (EGME, EGEE, and EGBE) is oxidation via alcohol and aldehyde dehydrogenases (ALD/ADH) that leads to the formation of an alkoxy acids. Alkoxy acids are the only toxicologically significant metabolites of glycol ethers that have been detected *in vivo*. The principal metabolite of TGME is believed to be 2-[2-(2-methoxyethoxy)ethoxy] acetic acid. Although ethylene glycol, a known kidney toxicant, has been identified as an impurity or a minor metabolite of glycol ethers in animal studies it does not appear to contribute to the toxicity of glycol ethers.

The metabolites of category members are not likely to be metabolized to any large extent to toxic molecules such as ethylene glycol or the mono alkoxy acids because metabolic breakdown of the ether linkages also has to occur

Acute toxicity: Category members generally display low acute toxicity by the oral, inhalation and dermal routes of exposure. Signs of toxicity in animals receiving lethal oral doses of TGBE included loss of righting reflex and flaccid muscle tone, coma, and heavy breathing. Animals administered lethal oral doses of TGEE exhibited lethargy, ataxia, blood in the urogenital area and piloerection before death.

Irritation: The data indicate that the glycol ethers may cause mild to moderate skin irritation. TGEE and TGBE are highly irritating to the eyes. Other category members show low eye irritation.

Repeat dose toxicity: Results of these studies suggest that repeated exposure to moderate to high doses of the glycol ethers in this category is required to produce systemic toxicity

In a 21-day dermal study, TGME, TGEE, and TGBE were administered to rabbits at 1,000 mg/kg/day. Erythema and oedema were observed. In addition, testicular degeneration (scored as trace in severity) was observed in one rabbit given TGEE and one rabbit given TGME. Testicular effects included spermatid giant cells, focal tubular hypospermatogenesis, and increased cytoplasmic vacuolisation. Due to a high incidence of similar spontaneous changes in normal New Zealand White rabbits, the testicular effects were considered not to be related to treatment. Thus, the NOAELs for TGME, TGEE and TGBE were established at 1000 mg/kg/day. Findings from this report were considered unremarkable.

A 2-week dermal study was conducted in rats administered TGME at doses of 1,000, 2,500, and 4,000 mg/kg/day. In this study, significantly-increased red blood cells at 4,000 mg/kg/day and significantly-increased urea concentrations in the urine at 2,500 mg/kg/day were observed. A few of the rats given 2,500 or 4,000 mg/kg/day had watery caecal contents and/or

haemolysed blood in the stomach. These gross pathologic observations were not associated with any histologic abnormalities in these tissues or alterations in haematologic and clinical chemistry parameters. A few males and females treated with either 1,000 or 2,500 mg/kg/day had a few small scabs or crusts at the test site. These alterations were slight in degree and did not adversely affect the rats

In a 13-week drinking water study, TGME was administered to rats at doses of 400, 1,200, and 4,000 mg/kg/day. Statistically-significant changes in relative liver weight were observed at 1,200 mg/kg/day and higher. Histopathological effects included hepatocellular cytoplasmic vacuolisation (minimal to mild in most animals) and hypertrophy (minimal to mild) in males at all doses and hepatocellular hypertrophy (minimal to mild) in high dose females. These effects were statistically significant at 4,000 mg/kg/day. Cholangiofibrosis was observed in 7/15 high-dose males; this effect was observed in a small number of bile ducts and was of mild severity. Significant, small decreases in total test session motor activity were observed in the high-dose animals, but no other neurological effects were observed. The changes in motor activity were secondary to systemic toxicity

Mutagenicity: Mutagenicity studies have been conducted for several category members. All *in vitro* and *in vivo* studies were negative at concentrations up to 5,000 micrograms/plate and 5,000 mg/kg, respectively, indicating that the category members are not genotoxic at the concentrations used in these studies. The uniformly negative outcomes of various mutagenicity studies performed on category members lessen the concern for carcinogenicity.

Reproductive toxicity: Although mating studies with either the category members or surrogates have not been performed, several of the repeated dose toxicity tests with the surrogates have included examination of reproductive organs. A lower molecular weight glycol ether, ethylene glycol methyl ether (EGME), has been shown to be a testicular toxicant. In addition, results of repeated dose toxicity tests with TGME clearly show testicular toxicity at an oral dose

	<p>of 4,000 mg/kg/day four times greater than the limit dose of 1,000 mg/kg/day recommended for repeat dose studies. It should be noted that TGME is 350 times less potent for testicular effects than EGME. TGBE is not associated with testicular toxicity, TetraME is not likely to be metabolised by any large extent to 2-MAA (the toxic metabolite of EGME), and a mixture containing predominantly methylated glycol ethers in the C5-C11 range does not produce testicular toxicity (even when administered intravenously at 1,000 mg/kg/day).</p> <p>Developmental toxicity: The bulk of the evidence shows that effects on the foetus are not noted in treatments with 1,000 mg/kg/day during gestation. At 1,250 to 1,650 mg/kg/day TGME (in the rat) and 1,500 mg/kg/day (in the rabbit), the developmental effects observed included skeletal variants and decreased body weight gain.</p>
<p>3-iodo-2-propynyl BUTYL CARBAMATE</p>	<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 µg/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 µg/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 µg/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>
<p>TINUVIN 213</p>	<p>Inhalation (rat) LC50: > 5.8 mg/l Aerosol Eye (rabbit): non-irritant Ames Test: Non Mutagenic</p>
<p>ZIRCONIUM 2-ETHYLHEXANOATE</p>	<p>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 sensitizers. 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</p>
<p>Mirotone TIMBRE PLUS INTERIOR & BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & TINUVIN 1130 & TINUVIN 213 & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE</p>	<p>The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p>
<p>Mirotone TIMBRE PLUS INTERIOR & ISOTRIDECYL ALCOHOL, ETHOXYLATED & TINUVIN 213 & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE & ZIRCONIUM 2-ETHYLHEXANOATE & WATER</p>	<p>No significant acute toxicological data identified in literature search.</p>

Mirotone TIMBRE PLUS INTERIOR

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

Mirotone TIMBRE PLUS INTERIOR	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
isotridecyl alcohol, ethoxylated	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
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
	NOEC	48	Crustacea	<0.01mg/L	4
bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	=0.34mg/L	1
Tinuvin 1130	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
Tinuvin 213	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
zirconium 2-ethylhexanoate	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
water	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

for propylene glycol ethers:

Environmental fate:

Most are liquids at room temperature and all are water-soluble.

Typical propylene glycol ethers include propylene glycol n-butyl ether (PnB); dipropylene glycol n-butyl ether (DPnB); dipropylene glycol methyl ether acetate (DPMA); tripropylene glycol methyl ether (TPM)

Environmental fate: Log octanol-water partition coefficients (log Kow's) range from 0.309 for TPM to 1.523 for DPnB. Calculated BCFs range from 1.47 for DPnB to 3.16 for DPMA and TPM, indicating low bioaccumulation. Henry's Law Constants, which indicate propensity to partition from water to air, are low for all category members, ranging from 5.7 x 10⁻⁹ atm-m³/mole for TPM to 2.7 x 10⁻⁹ atm-m³/mole for PnB. Fugacity modeling indicates that most propylene glycol ethers are likely to partition roughly equally into the soil and water compartments in the environment with small to negligible amounts remaining in other environmental compartments (air, sediment, and aquatic biota). Propylene glycol ethers are unlikely to persist in the environment. Once in air, the half-life of the category members due to direct reactions with photochemically generated hydroxyl radicals, range from 2.0 hours for TPM to 4.6 hours for PnB. In water, most members of this family are "readily biodegradable" under aerobic conditions. (DPMA degraded within 28 days (and within the specified 10-day window) but only using pre-adapted or "acclimated" inoculum.). In soil, biodegradation is rapid for PM and PMA.

Ecotoxicity:

Acute aquatic toxicity testing indicates low toxicity for both ethers and acetates. For ethers, effect concentrations are > 500 mg/L. For acetates, effect concentrations are > 151 mg/L.

For 1,2,4-trimethylbenzene:

Continued...

Mirotone TIMBRE PLUS INTERIOR

Half-life (hr) air : 0.48-16
 Half-life (hr) H2O surface water : 0.24-672
 Half-life (hr) H2O ground : 336-1344
 Half-life (hr) soil : 168-672
 Henry's Pa m3 /mol: 385-627
 Bioaccumulation : not significant

1,2,4-Trimethylbenzene is a volatile organic compound (VOC) substance. As a VOC, 1,2,4-trimethylbenzene can contribute to the formation of photochemical smog in the presence of other VOCs.

Environmental fate:

Transport: 1,2,4-Trimethylbenzene volatilises rapidly from surface waters as predicted by a Henry's law constant of 5.18×10^{-3} (vapor pressure, 2.03 mm Hg). The volatilisation half-life from a model river is calculated to be 3.4 hours. The chemical also volatilises from soils, however, based on an estimated Koc of 472, moderate adsorption to soils and sediments may occur

Transformation/Persistence

Air - Degradation of 1,2,4-trimethylbenzene in the atmosphere occurs by reaction with hydroxyl radicals. Reaction also occurs with ozone but very slowly (half life, 8820 days). In the atmosphere, two estimates of the half-life are approximately 6 hours and, in the presence of hydroxyl radicals, 0.5 days.

Soil - Volatilisation is the major route of removal of 1,2,4-trimethylbenzene from soils; although, biodegradation may also occur. Due to the high volatility of the chemical it is unlikely to accumulate in soil or surface water to toxic concentrations.

Water - Because of 1,2,4-trimethylbenzene's water solubility and its vapor pressure of 2.03 mm Hg, the chemical will rapidly volatilise from surface waters. Biodegradation of 1,2,4-trimethylbenzene occurred with inoculums from both seawater and ground water. Various strains of *Pseudomonas* can biodegrade 1,2,4-trimethylbenzene.

Biota - The estimated bioconcentration factor (439) and high volatility of 1,2,4-trimethylbenzene indicates that bioaccumulation of the chemical will not be significant.

Ecotoxicity:

Fish LC50 (96 h): fathead minnow 7.72 mg/l

No stress was observed in *Oncorhynchus mykiss* (rainbow trout, fingerling) or *Petromyzon marinus* (sea lamprey, larvae) at 5 mg/L for 24 hours.

Daphnia magna EC50 (48 h): 3.61 mg/l

Cancer magister (dungeness crab) LC50 996 h): 5.1 mg/l

1,2,4-Trimethylbenzene has moderate acute toxicity to aquatic organisms; acute toxicity values fall within the range of greater than 1 mg/L and 100 mg/L. LC50 values for specific aquatic organisms range from approximately 5 to 8 mg/L, which is orders of magnitude greater than any measured concentration in seawater (0.002 - 0.54 microgram/L). The high concentrations required to induce toxicity in laboratory animals are not likely to be reached in the environment.

Within an aromatic series, acute toxicity increases with increasing alkyl substitution on the aromatic nucleus. For example, there is an increase in toxicity as alkylation of the naphthalene structure increases. The order of most toxic to least in a study using grass shrimp (*Palaemonetes pugio*) and brown shrimp (*Penaeus aztecus*) was dimethylnaphthalenes > methylnaphthalenes > naphthalenes.

Studies conclude that the toxicity of an oil appears to be a function of its di-aromatic and tri-aromatic hydrocarbons, which includes three-ring hydrocarbons such as phenanthrene.

The heavier (4-, 5-, and 6-ring) PAHs are more persistent than the lighter (2- and 3-ring) PAHs and tend to have greater carcinogenic and other chronic impact potential. PAHs in general are more frequently associated with chronic risks. These risks include cancer and often are the result of exposures to complex mixtures of chronic-risk aromatics (such as PAHs, alkyl PAHs, benzenes, and alkyl benzenes), rather than exposures to low levels of a single compound.

Anthracene is a phototoxic PAH. UV light greatly increases the toxicity of anthracene to bluegill sunfish. Benchmarks developed in the absence of UV light may be under-protective, and biological resources in strong sunlight are at more risk than those that are not.

For C9 aromatics (typically trimethylbenzene - TMBs)

Chemicals in this category possess properties indicating a hazard for the environment (acute toxicity for fish, invertebrates, and algae from 1 to 10 mg/L). Category members are readily biodegradable, except 1,3,5-trimethylbenzene (CAS RN 108-67-8). Category members are not expected to be bioaccumulative.

Environmental Fate:

In the air, category member constituents have the potential to rapidly degrade through indirect photolytic processes mediated primarily by hydroxyl radicals with calculated degradation half-lives ranging from 0.54 to 2.81 days (based on a 12-hour day and a hydroxyl radical concentration of 5×10^5). Aqueous photolysis and hydrolysis will not contribute to the transformation of category chemical constituents in aquatic environments because they are either poorly reactive or not susceptible to these reactions.

Results of the Mackay Level I environmental distribution model show that chemical constituents of C9 Aromatic Hydrocarbon Solvents Category members have the potential to partition to air (96.8 to 98.9%), with a negligible amount partitioning to water (0.2 to 0.6%) and soil (0.9 to 2.7%). In comparison, Level III modeling indicates that category members partition primarily to soil (66.3 to 79.6%) and water (17.8 to 25.0%) compartments rather than air (2.4 to 8.4%) when an equal emission rate (1000 kg/hr) is assumed to each of the air, water, and soil compartments. When release (1000 kg/hr) is modeled only to either the air, water, or soil compartment, constituents are indicated in the modeling to partition primarily (>94%) to the compartment to which they are emitted as advection and degradation influence constituent concentration in compartments to which constituents are not released. Solvent naphtha, (pet.), light aromatic (CAS RN 64742-95-6), 1,2,4-trimethylbenzene (CAS RN 95-63-6), and 1-ethyl-3-methylbenzene (CAS RN 620-14-4) were determined to be readily biodegradable based on the studies that used the TG OECD 301F (the latter substance is used to characterize the potential biodegradability of the category member, ethylmethylbenzene (CAS RN 25550-14-5)). These three substances exceed 60% biodegradation in 28 days and met the 10-day window criterion for ready biodegradation. In comparison 1,3,5-trimethylbenzene (CAS RN 108-67-8) was not readily biodegradable. It achieved 42% biodegradation after 28 days and 60% biodegradation after 39 days. The result for the multi-constituent substance (CAS RN 64742-95-6), a UVCB, characterizes the biodegradability of that substance as a whole, but it does not suggest that each constituent is equally biodegradable. As with all ready biodegradation test guidelines, the test system and study design used with these substances (OECD TG 301F) is not capable of distinguishing the relative contribution of the substances' constituents to the total biodegradation measured.

Based on Henry's Law constants (HLCs) representing a potential to volatilize from water that range from 590 to 1000 Pa-m³/mole, the potential to volatilize from surface waters for chemicals in the C9 Aromatic Hydrocarbon Solvents Category is expected to be high.

Based on the measured bioconcentration factors that range from 23 to 342 for 1,2,4-trimethylbenzene and 1,3,5-trimethylbenzene, the category members are not expected to be bioaccumulative.

Ecotoxicity

Acute toxicity values used to characterize this category for fish (LL50; LC50) and invertebrates (EL50; EC50) range from 3.5 to 9.2 mg/L, based on measured data. For algae, one study for a category member (CAS RN 64742-95-6) resulted in a 72-hr EC50 of 2.4 mg/L (biomass) and 2.7 mg/L (growth rate) based on measured concentrations.

The algal 72-hour NOEC (no observed effect concentration) for biomass and growth rate is 1.3 mg/L, based on mean measured concentrations. A 21-day *Daphnia magna* reproduction study with 1,3,5-trimethylbenzene (CAS RN 108-67-8) resulted in a NOEC value of 0.4 mg/L, based on a minimum measured value.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
3-iodo-2-propynyl butyl carbamate	HIGH	HIGH
water	LOW	LOW

Bioaccumulative potential

Ingredient	Bioaccumulation
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
water	LOW (LogKOW = -1.38)

Mobility in soil

Ingredient	Mobility
3-iodo-2-propynyl butyl carbamate	LOW (KOC = 365.3)
water	LOW (KOC = 14.3)

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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Ensure that the disposal of material is carried out in accordance with Hazardous Substances (Disposal) Regulations 2001.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (UN): 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

This substance is to be managed using the conditions specified in an applicable Group Standard

HSR Number	Group Standard
HSR002624	N.O.S. (Subsidiary Hazard) Group Standard 2006
HSR002535	Compressed Gas Mixtures (Subsidiary Hazard) Group Standard 2006
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2006
HSR002530	Cleaning Products (Subsidiary Hazard) Group Standard 2006
HSR002585	Fuel Additives (Subsidiary Hazard) Group Standard 2006
HSR002519	Aerosols (Subsidiary Hazard) Group Standard 2006
HSR002521	Animal Nutritional and Animal Care Products Group Standard 2006
HSR002606	Lubricants, Lubricant Additives, Coolants and Anti-freeze Agents (Subsidiary Hazard) Group Standard 2006
HSR002644	Polymers (Subsidiary Hazard) Group Standard 2006
HSR002647	Reagent Kits Group Standard 2006
HSR002612	Metal Industry Products (Subsidiary Hazard) Group Standard 2006
HSR002670	Surface Coatings and Colourants (Subsidiary Hazard) Group Standard 2006
HSR002503	Additives, Process Chemicals and Raw Materials (Subsidiary Hazard) Group Standard 2006
HSR002638	Photographic Chemicals (Subsidiary Hazard) Group Standard 2006
HSR002565	Embalming Products (Subsidiary Hazard) Group Standard 2006
HSR002578	Food Additives and Fragrance Materials (Subsidiary Hazard) Group Standard 2006
HSR002558	Dental Products (Subsidiary Hazard) Group Standard 2006

Continued...

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HSR002684	Water Treatment Chemicals (Subsidiary Hazard) Group Standard 2006
HSR002573	Fire Fighting Chemicals Group Standard 2006
HSR100425	Pharmaceutical Active Ingredients Group Standard 2010
HSR002600	Leather and Textile Products (Subsidiary Hazard) Group Standard 2006
HSR002571	Fertilisers (Subsidiary Hazard) Group Standard 2006
HSR002648	Refining Catalysts Group Standard 2006
HSR002653	Solvents (Subsidiary Hazard) Group Standard 2006
HSR002544	Construction Products (Subsidiary Hazard) Group Standard 2006
HSR002549	Corrosion Inhibitors (Subsidiary Hazard) Group Standard 2006
HSR002552	Cosmetic Products Group Standard 2006
HSR100757	Veterinary Medicine (Limited Pack Size, Finished Dose) Standard 2012
HSR100758	Veterinary Medicines (Non-dispersive Closed System Application) Group Standard 2012
HSR100628	Straight-chained Lepidopteran Sex Pheromone Group Standard 2012

ISOTRIDECYL ALCOHOL, ETHOXYLATED(9043-30-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

3-IODO-2-PROPYNYL BUTYL CARBAMATE(55406-53-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE(41556-26-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

TINUVIN 1130(104810-47-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

TINUVIN 213(104810-48-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE(82919-37-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals

New Zealand Inventory of Chemicals (NZIoC)

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

New Zealand Inventory of Chemicals (NZIoC)

New Zealand Workplace Exposure Standards (WES)

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

New Zealand Inventory of Chemicals (NZIoC)

Location Test Certificate

Subject to Regulation 55 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations, a location test certificate is required when quantity greater than or equal to those indicated below are present.

Hazard Class	Quantity beyond which controls apply for closed containers	Quantity beyond which controls apply when use occurring in open containers
Not Applicable	Not Applicable	Not Applicable

Approved Handler

Subject to Regulation 56 of the Hazardous Substances (Classes 1 to 5 Controls) Regulations and Regulation 9 of the Hazardous Substances (Classes 6, 8, and 9 Controls) Regulations, the substance must be under the personal control of an Approved Handler when present in a quantity greater than or equal to those indicated below.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (3-iodo-2-propynyl butyl carbamate; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; water; Tinuvin 213; isotridecyl alcohol, ethoxylated; zirconium 2-ethylhexanoate; Tinuvin 1130; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	N (Tinuvin 213; Tinuvin 1130)
Japan - ENCS	N (Tinuvin 213; isotridecyl alcohol, ethoxylated; zirconium 2-ethylhexanoate; Tinuvin 1130)
Korea - KECI	Y

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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
isotridecyl alcohol, ethoxylated	9043-30-5, 112481-34-2, 126730-81-2, 128088-01-7, 128088-02-8, 142901-64-2, 145054-32-6, 165943-41-9, 195395-82-5, 357177-18-5, 53858-87-0, 558452-92-9, 779349-84-7, 8076-51-5, 856005-68-0, 870083-47-9, 9063-90-5, 909706-17-8

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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TEL (+61 3) 9572 4700.