

Mirotone GOLD PREMIUM

Mirotone (NZ) LTD

Chemwatch Hazard Alert Code: 2

Chemwatch: 84-6346

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Safety Data Sheet according to HSNO Regulations

L.GHS.NZL.EN

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

Product Identifier

Product name	Mirotone GOLD PREMIUM
Synonyms	Not Available
Other means of identification	Not Available

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

Relevant identified uses	For full details on application and properties consult the technical datasheet. A premium exterior, transparent, penetrating timber finish, to enhance the natural grain and hues of timber without concealing them. Excellent on broad surface areas and outdoor furniture.
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Details of the supplier of the safety data sheet

Registered company name	Mirotone (NZ) LTD
Address	32 Cryers Road East Tamaki, Auckland New Zealand
Telephone	+64 800 346 474
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Website	www.mirotone.co.nz
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Emergency telephone number


Association / Organisation	Not Available	CHEMWATCH EMERGENCY RESPONSE
Emergency telephone numbers	111 FIRE AND EMERGENCY	+64 800 700 112
Other emergency telephone numbers	0800 764 766 NATIONAL POISON CENTRE	+61 2 9186 1132

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Classification ^[1]	Acute Toxicity (Oral) 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 Regulation (EU) No 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.1E (oral), 6.3B, 6.5B (contact)

Label elements

Hazard pictogram(s)	
SIGNAL WORD	WARNING

Hazard statement(s)

Continued...

H303	May be harmful if swallowed.
H316	Causes mild skin irritation.
H317	May cause an allergic skin reaction.

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

P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P302+P352	IF ON SKIN: Wash with plenty of water and soap.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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
111-76-2	10-30	<u>ethylene glycol monobutyl ether</u>
55406-53-6	<0.5	<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	<0.2	<u>Tinuvin 213</u>
82919-37-7	<0.2	<u>methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate</u>
26530-20-1	<0.1	<u>2-octyl-4-isothiazolin-3-one</u>
13586-82-8	<0.1	<u>cobalt 2-ethylhexanoate</u>
22464-99-9	<0.05	<u>zirconium 2-ethylhexanoate</u>
104-76-7	<0.02	<u>2-ethylhexanol</u>
149-57-5	<0.01	<u>2-ethylhexanoic acid</u>
Not Available	balance	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES**Description of first aid measures**

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes, aerosols or combustion products are inhaled remove from contaminated area. ▶ Other measures are usually unnecessary.

Ingestion

- ▶ 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.

Advice for firefighters**Fire Fighting**

- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves in the event of a fire.
- ▶ Prevent, by any means available, spillage from entering drains or water courses.
- ▶ Use fire fighting procedures suitable for surrounding area.
- ▶ **DO NOT** approach containers suspected to be hot.
- ▶ Cool fire exposed containers with water spray from a protected location.
- ▶ If safe to do so, remove containers from path of fire.
- ▶ Equipment should be thoroughly decontaminated after use.

Fire/Explosion Hazard

- ▶ The 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.

Decomposes on heating and produces toxic fumes of:
carbon dioxide (CO₂)
nitrogen oxides (NO_x)
other pyrolysis products typical of burning organic material.

SECTION 6 ACCIDENTAL RELEASE MEASURES**Personal precautions, protective equipment and emergency procedures**

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up**Minor Spills**

- ▶ Clean up all spills immediately.
- ▶ Avoid breathing vapours and contact with skin and eyes.
- ▶ Control personal contact with the substance, by using protective equipment.
- ▶ Contain and absorb spill with sand, earth, inert material or vermiculite.
- ▶ Wipe up.
- ▶ Place in a suitable, labelled container for waste disposal.

Major Spills

Chemical Class: bases
For release onto land: recommended sorbents listed in order of priority.

SORBENT TYPE	RANK	APPLICATION	COLLECTION	LIMITATIONS
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LAND SPILL - SMALL

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

LAND SPILL - MEDIUM

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

Legend

DGC: Not effective where ground cover is dense

R; Not reusable

I: Not incinerable

P: Effectiveness reduced when rainy

RT:Not effective where terrain is rugged

SS: Not for use within environmentally sensitive sites

W: Effectiveness reduced when windy

Reference: Sorbents for Liquid Hazardous Substance Cleanup and Control;

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

Moderate hazard.

- ▶ Clear area of personnel and move upwind.
- ▶ Alert Fire Brigade and tell them location and nature of hazard.
- ▶ Wear breathing apparatus plus protective gloves.
- ▶ Prevent, by any means available, spillage from entering drains or water course.
- ▶ Stop leak if safe to do so.
- ▶ Contain spill with sand, earth or vermiculite.
- ▶ Collect recoverable product into labelled containers for recycling.
- ▶ Neutralise/decontaminate residue (see Section 13 for specific agent).
- ▶ Collect solid residues and seal in labelled drums for disposal.
- ▶ Wash area and prevent runoff into drains.
- ▶ 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

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

	<ul style="list-style-type: none"> ▶ 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)	ethylene glycol monobutyl ether	2-Butoxyethanol (Butyl glycol ether)	25 ppm / 121 mg/m ³	Not Available	Not Available	(skin) - Skin absorption
New Zealand Workplace Exposure Standards (WES)	zirconium 2-ethylhexanoate	Zirconium and compounds, as Zr	5 mg/m ³	10 mg/m ³	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
ethylene glycol monobutyl ether	Butoxyethanol, 2-; (Glycol ether EB)	60 ppm	120 ppm	700 ppm
3-iodo-2-propynyl butyl carbamate	Butyl-3-iodo-2-propynylcarbamate	3.3 mg/m ³	36 mg/m ³	220 mg/m ³
2-ethylhexanol	Ethyl-1-hexanol, 2-	Not Available	Not Available	Not Available
2-ethylhexanoic acid	Ethyl hexanoic acid, 2-; (Butyl ethyl acetic acid)	15 mg/m ³	140 mg/m ³	590 mg/m ³

Ingredient	Original IDLH	Revised IDLH
ethylene glycol monobutyl ether	700 ppm	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
2-octyl-4-isothiazolin-3-one	Not Available	Not Available
cobalt 2-ethylhexanoate	Not Available	Not Available
zirconium 2-ethylhexanoate	25 mg/m ³	Not Available
2-ethylhexanol	Not Available	Not Available
2-ethylhexanoic acid	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.

bitumen (asphalt) fumes [8052-42-4]

Continued...

TLV* TWA: 0.5 mg/m³ A4 asphalt (petroleum, bitumen) fume, as benzene soluble aerosol

ES* TWA: 5 mg/m³ as fumes

OES* TWA: 5 mg/m³; STEL: 10 mg/m³ as fumes

Based on surveys of asphalt workers in oil refineries and in the roofing industry the TLV-TWA is thought to reduce the risk of possible carcinogenicity

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.

for exposure to ammonia gas/ vapours:

Odour Threshold Value: Various reported as 0.019 ppm and 55 ppm; AIHA Value 16.7 ppm (detection)

NOTE: Detector tubes for ammonia, measuring in excess of 1 ppm, are commercially available.

The TLV-TWA is thought to be protective against irritation of the eyes and respiratory tract and minimise discomfort among workers that are not inured to its effects and systemic damage. Acclimatised persons are able to tolerate prolonged exposures of up to 100 ppm without symptoms. Marked irritation has been seen in persons exposed to ammonia concentrations between 50 and 100 ppm only when the exposures involved sudden concentration peaks which do not permit short-term acclimatisation. The detoxification capacity of the liver is significant since the amount of ammonia formed endogenously in the intestines markedly exceeds that from external sources.

Human exposure effects, at vapour concentrations of about:

Concentration Possible Effects

(ppm)

5	minimal irritation
9-50	nasal dryness, olfactory fatigue and moderate irritation
125-137	definite nose, throat and chest irritation
140	slight eye irritation
150	laryngeal spasm
500	30 minute exposures may produce cyclic hypernea, increased blood pressure and pulse rate, and upper respiratory tract irritation which may persist for 24 hours
700	immediate eye irritation
1,500-10,000	dyspnea, convulsive coughing, chest pain, respiratory spasm, pink frothy sputum, rapid asphyxia and delayed pulmonary oedema which may be fatal. Other effects include runny nose, swelling of the lips, restlessness, headache, salivation, nausea, vomiting, glottal oedema, pharyngitis, tracheitis, and speech difficulties. Bronchopneumonia, asphyxiation due to spasms, inflammation, and oedema of the larynx, may be fatal. Residual effects include hoarseness, productive cough, and decreased respiratory function
>2,500	severe eye irritation, with swelling of the eyelids, lachrymation, blepharospasm, palpebral oedema, increased intraocular pressure, oval semi-dilated, fixed pupils, corneal ulceration (often severe) and temporary blindness. Depending on duration of exposure, there may be destruction of the epithelium, corneal and lenticular opacification, and iritis accompanied by hypopyon or haemorrhage and possible loss of pigment from the posterior layer of the iris. Less severe damage is often resolved. In the case of severe damage, symptoms may be delayed; late complications including persistent oedema, vascularisation and corneal scarring, permanent opacity, acute angle glaucoma, staphyloma, cataract, and atrophy of the retina, iris, and symblepharon. Long-term exposure to sub-acute concentrations or single exposures to high concentrations may produce chronic airway dysfunction, alveolar disease, bronchiolitis, bronchiectasis, emphysema and anxiety neuroses

Odour Safety Factor(OSF)

OSF=3.8 (AMMONIA)

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

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

Exposure controls

Appropriate engineering controls

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

The basic types of engineering controls are:

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

Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air

contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.

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

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

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min) for extraction of solvents generated in a tank 2 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

- ▶ 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

- ▶ Wear chemical protective gloves, e.g. PVC.
- ▶ Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.

The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.

Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:

- frequency and duration of contact,
- chemical resistance of glove material,

- glove thickness and
 - dexterity
- Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).
- When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended.
 - Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.
 - Contaminated gloves should be replaced.
- As defined in ASTM F-739-96 in any application, gloves are rated as:
- Excellent when breakthrough time > 480 min
 - Good when breakthrough time > 20 min
 - Fair when breakthrough time < 20 min
 - Poor when glove material degrades
- For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.
- Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.
- Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:
- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
 - Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential
- 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.

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.

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:

Mirotone GOLD PREMIUM

Material	CPI
BUTYL	C
HYPALON	C
NAT+NEOPR+NITRILE	C
NATURAL RUBBER	C
NATURAL+NEOPRENE	C
NEOPRENE	C
NEOPRENE/NATURAL	C
NITRILE	C
NITRILE+PVC	C
PE/EVAL/PE	C
PVA	C
PVC	C
SARANEX-23	C
VITON	C

* CPI - Chemwatch Performance Index

Respiratory protection

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

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

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

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

^ - Full-face

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

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

Continued...

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.

▶ Because of these limitations, only restricted use of cartridge respirators is considered appropriate.

▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Brown non-flammable low viscosity liquid with a characteristic odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	0.95-1.05
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	8-10	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	80-490 @25C
Initial boiling point and boiling range (°C)	105 (initial)	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	Not Applicable	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Not Applicable	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	73-81
Vapour pressure (kPa)	2.3	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	0.8	VOC g/L	137-152

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	Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.
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Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Open cuts, abraded or irritated skin should not be exposed to this material
Eye	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.</p> <p>There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects. Exposure to the material may cause concerns for human fertility, on the basis that similar materials provide some evidence of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects.</p> <p>On the basis, primarily, of animal experiments, concern has been expressed by at least one classification body that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment.</p> <p>Studies with some ethylene glycol ethers and their esters indicate reproductive changes, testicular atrophy, infertility and kidney function changes. The metabolic acetic acid derivatives of the glycol ethers (alkoxyacetic acids), not the ether itself, have been found to be the proximal reproductive toxin in animals. The potency of these metabolites decrease significantly as the chain length of the ether increases. Consequently glycol ethers with longer substituents (e.g diethylene glycols, triethylene glycols) have not generally been associated with reproductive effects. One of the most sensitive indicators of toxic effects observed from many of the glycol ethers is an increase in the erythrocytic osmotic fragility in rats. This appears to be related to the development of haemoglobinuria (blood in the urine) at higher exposure levels or as a result of chronic exposure. Ethylene glycol ethers and acetates are mainly metabolised to alkoxyacetic acids but there is also a minor pathway through ethylene glycol to oxalic acid. The main pathway of ethylene glycol ethers is associated with significant clinical or experimental health effects, but the minor pathway is also interesting because formation of urinary stones depends principally upon urinary concentration of oxalate and calcium. In one study (1) the tendency to form urinary stones was 2.4 times higher amongst silk-screen printers exposed to ethylene glycol ethers, than among office workers. (1) Laitinen J., et al: Occupational Environmental Medicine 1996, 53 595-600</p>

Mirotone GOLD PREMIUM	TOXICITY	IRRITATION
	Not Available	Not Available
ethylene glycol monobutyl ether	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye (rabbit): 100 mg SEVERE
	Inhalation (rat) LC50: 449.48655 mg/l/4H ^[2]	Eye (rabbit): 100 mg/24h-moderate
	Oral (rat) LD50: 250 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 500 mg, open; mild
	Skin: adverse effect observed (irritating) ^[1]	
	Skin: no adverse effect observed (not irritating) ^[1]	
3-iodo-2-propynyl butyl carbamate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2]	Eye: adverse effect observed (irreversible damage) ^[1]
	Inhalation (rat) LC50: 0.680 mg/l/4h* ^[2]	Eye: Irritating
	Oral (rat) LD50: 1056 mg/kg ^[2]	Skin: no adverse effect observed (not irritating) ^[1]
	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
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (rat) LD50: >5000 mg/kg ^[1]	Not Available
Tinuvin 213	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Oral (rat) LD50: >5000 mg/kg ^[1]	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
2-octyl-4-isothiazolin-3-one	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: 690 mg/kg ^[2]	Eye (rabbit): 0.5% non irritant
	Oral (rat) LD50: 550 mg/kg ^[2]	Eye (rabbit): 45% conc CORROSIVE
		Eye (rabbit): 5% conc moderate
		Eye(rabbit):100 mg SEVERE
		Eye: adverse effect observed (irreversible damage) ^[1]
		Skin (rabbit): 45% conc SEVERE
		Skin (rabbit): 500 mg/24 hours
		Skin: adverse effect observed (corrosive) ^[1] Skin: adverse effect observed (irritating) ^[1]
cobalt 2-ethylhexanoate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Inhalation (rat) LC50: >2.5 mg/l/1H ^[2] Oral (rat) LD50: 1220 mg/kg ^[2]	Eye: adverse effect observed (irritating) ^[1] Skin: no adverse effect observed (not irritating) ^[1]
zirconium 2-ethylhexanoate	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1] Inhalation (rat) LC50: >2.2 mg/l/1H ^[2] Oral (rat) LD50: 2043 mg/kg ^[1]	Not Available
2-ethylhexanol	TOXICITY	IRRITATION
	dermal (rat) LD50: >3000 mg/kg ^[2] Oral (rat) LD50: 1516-2774 mg/kg ^[2]	Eye (rabbit): 20 mg/24h - moderate Eye (rabbit): 4.17 mg - SEVERE
		Eye: adverse effect observed (irritating) ^[1]
		Skin (rabbit): 415 mg (open)-mild Skin (rabbit): 500 mg/24h-moderate
		Skin: adverse effect observed (irritating) ^[1]
2-ethylhexanoic acid	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[2] Inhalation (rat) LC50: >3.54 mg/l/6H ^[2] Oral (rat) LD50: 1600 mg/kg ^[2]	Eye (rabbit): 4.5 mg SEVERE Skin (rabbit): 10 mg/24h mild Skin (rabbit): 450 mg open mild
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	

ETHYLENE GLYCOL MONOBUTYL ETHER

For ethylene glycol:

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

dehydrogenase to form glycolaldehyde, which is rapidly converted to glycolic acid and glyoxal by aldehyde oxidase and aldehyde dehydrogenase. These metabolites are oxidised to glyoxylate; glyoxylate may be further metabolised to formic

acid, oxalic acid, and glycine. Breakdown of both glycine and formic acid can generate CO₂, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO₂, ethylene glycol is eliminated in the urine as both the parent compound and glycolic acid. Elimination of ethylene glycol from the plasma in both humans and laboratory animals is rapid after oral exposure; elimination half-lives are in the range of 1-4 hours in most species tested.

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

Cardiovascular Effects. Cardiovascular system involvement in humans occurs at the same time as respiratory system involvement, during the second phase of oral ethylene glycol poisoning, which is 12- 24 hours after acute exposure. The symptoms of cardiac involvement include tachycardia, ventricular gallop and cardiac enlargement. Ingestion of ethylene glycol may also cause hypertension or hypotension, which may progress to cardiogenic shock. Myocarditis has been observed at autopsy in cases of people who died following acute ingestion of ethylene glycol. As in the case of respiratory effects, cardiovascular involvement occurs with ingestion of relatively high doses of ethylene glycol. Nevertheless, circulatory disturbances are a rare occurrence, having been reported in only 8 of 36 severely poisoned cases. Therefore, it appears that acute exposure to high levels of ethylene glycol can cause serious cardiovascular effects in humans. The effects of a long-term, low-dose exposure are unknown.

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

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

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

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

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

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

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

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

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

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

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

	NOTE: Changes in kidney, liver, spleen and lungs are observed in animals exposed to high concentrations of this substance by all routes. ** ASCC (NZ) SDS
3-iodo-2-propynyl BUTYL CARBAMATE	<p>for 3-iodo-2-propynyl butyl carbamate (IPBC):</p> <p>Acute toxicity: Acceptable acute toxicity studies with IPBC indicate low toxicity except eye irritation. In a primary eye irritation study in rabbit. IPBC technical was severely irritating to the eyes of white rabbits, with corneal opacity and corneal vascularization reported in unwashed eyes by day 21 post-treatment. The technical grade of IPBC was slightly irritating to the skin of white rabbits. In a dermal sensitization study in Guinea pigs IPBC technical, at a concentration of 0.32%, produced no evidence of sensitization in male and female Guinea pigs.</p> <p>Subchronic toxicity: In a subchronic oral toxicity study, male and female Sprague-Dawley rats received IPBC technical by gavage for 13 weeks at doses of 0, 20, 50, and 125 mg/kg/day. At the 125 mg/kg/day dose level, body weight gain was decreased by 19% in male rats for weeks 1-13 of the study, and by 12% in female rats over the same period. Absolute liver weight was increased by 20% in male rats at the 125 mg/kg/day dose, and by 31% in female rats at this dose level. Liver to body weight ratio was significantly increased by approximately 31% in both male and female rats at the 125 mg/kg/day dose level, while kidney to body weight ratio in female rats was increased 18% at the 125 mg/kg/day dose level. The systemic NOEL was considered to be 20 mg/kg/day, while the systemic LEL was considered to be 50 mg/kg/day, based on increased liver to body weight ratio.</p> <p>In a subchronic dermal toxicity study, male and female Sprague-Dawley rats (10/sex/dose) received dermal doses of 50, 200, and 500 mg/kg/day IPBC technical grade (97.5%) to the shaved skin for five days a week, six hours per day. At the 500 mg/kg/day dose, decreased body weight (4-6%) and weight gain (11%) were observed in male rats, but not in female rats. In female rats, significant increases in haemoglobin, haematocrit, and eosinophils were observed at the 500 mg/kg/day dose level. Reticulocytes as a percentage of red cells were decreased in the 50 and 200 mg/kg/day dose groups but not at the 500 mg/kg/day dose level. Females in this study showed inhibition of plasma cholinesterase at 500 mg/kg/day test article, which may have been the result of either direct liver toxicity or inhibition of cholinesterase itself. Based upon the results of this study, the systemic NOEL is 200 mg/kg/day, the systemic LEL is 500 mg/kg/day for male and female rats.</p> <p>Carcinogenicity: In a 2-year chronic toxicity/carcinogenicity study, technical grade IPBC (98.68% ai) was administered to male and female Sprague Dawley rats (50/sex/group) at dose levels of 0, 20, 40, and 80 mg/kg/day. There were no statistically significant increases in tumor incidences in male rats. The incidence of mammary gland fibroadenoma and combined fibroadenoma/carcinoma in female rats was significantly increased at the 20 mg/kg/day dose level but there was no dose-related trend.</p> <p>Developmental and reproductive toxicity: The developmental toxicity of IPBC was assessed in pregnant Sprague-Dawley rats on gestation days six through 15 by oral administration of the test chemical at doses of 0, 20, 50, and 125 mg/kg/day. Maternal toxicity as reduced body weight gain during dosing was observed at the 125 mg/kg/day dose level. Developmental toxicity consisted of an increased incidence of skeletal abnormalities at the 125 mg/kg/day dose level. The maternal toxicity NOEL was determined to be 50 mg/kg/day, and the maternal toxicity LEL was determined to be 125 mg/kg/day, based on reduced body weight gain. The developmental toxicity NOEL was determined to be 50 mg/kg/day, and the developmental toxicity LEL was determined to be 125 mg/kg/day, based on incompletely ossified frontal skull bones and pelvic girdles.</p> <p>A 2-generation reproductive toxicity study was conducted in male and female Sprague-Dawley rats. IPBC technical was administered over two generations at doses of 0, 120, 300, and 750 ppm (0, 6, 15, and 37.5 mg/kg/day). Reduced body weight and food consumption was observed for P1 and F1 males during the pre-mating period at the 37.5 mg/kg/day dose. A decreased mean live birth index was reported for P1 and F1 generations without an effect on viability and development of pups. No adverse effects on reproductive indices or mating performance were observed at any dose level. The parental toxicity NOEL was determined to be 15 mg/kg/day, and the parental toxicity LEL was determined to be 37.5 mg/kg/day, based on decreased body weight and food consumption during pre-mating for P1 and F1 males, and decreased mean live birth index for the P1 and F1 generations. The reproductive toxicity NOEL was determined to be 37.5 mg/kg/day, and the reproductive toxicity LEL was determined to be >37.5 mg/kg/day.</p> <p>Mutagenicity: In a mutagenicity study, IPBC technical was tested for the ability to cause mutations in <i>Salmonella typhimurium</i> strains TA 1535, TA 1537, TA 1538, TA 98, and TA 100. In the five strains used, IPBC was found to be non-mutagenic in the presence or absence of metabolic activation at the concentrations tested, 1-1000 jig/plate. In a micronucleus assay in mice, IPBC at doses of 200, 600, and 2000 mg/kg did not induce any significant increase of the PCE containing micronuclei from the treated mice when compared to that of the vehicle control mice. In two independent unscheduled DNA synthesis (UDS) assays in primary rat hepatocytes, eight doses of IPBC ranging from 3.0 to 13.5 ug/ml did not cause an appreciable increase in mean net nuclear grain counts. Doses >13.5 ug/ml were cytotoxic, supporting the conclusion that IPBC induced cytotoxicity but no genotoxicity in this assay.</p> <p>Metabolism: Based on the metabolite identification data, a scheme for metabolism of IPBC was proposed. According to this scheme, IPBC undergoes reductive dehalogenation followed by dealkylation to form the URM-9 and URM-10 metabolites. In addition, de-carboxylation following reductive dehalogenation yields carbon dioxide. Various other metabolites formed from dehalogenation are glucuronidated and constitute minor metabolites of IPBC..</p>
TINUVIN 213	Inhalation (rat) LC50: > 5.8 mg/l Aerosol Eye (rabbit): non-irritant Ames Test: Non Mutagenic
2-OCTYL-4-ISOTHIAZOLIN-3-ONE	ROHM & HAAS Data ADI: 0.03 mg/kg/day NOEL: 60 mg/kg/day
ZIRCONIUM 2-ETHYLHEXANOATE	<p>For aliphatic fatty acids (and salts)</p> <p>Acute oral (gavage) toxicity:</p> <p>The acute oral LD50 values in rats for both were greater than >2000 mg/kg bw Clinical signs were generally associated with poor condition following administration of high doses (salivation, diarrhoea, staining, piloerection and lethargy). There were no adverse effects on body weight in any study. In some studies, excess test substance and/or irritation in the gastrointestinal tract was observed at necropsy.</p> <p>Skin and eye irritation potential, with a few stated exceptions, is chain length dependent and decreases with increasing chain length</p>

According to several OECD test regimes the animal skin irritation studies indicate that the C6-10 aliphatic acids are severely irritating or corrosive, while the C12 aliphatic acid is irritating, and the C14-22 aliphatic acids generally are not irritating or mildly irritating.

Human skin irritation studies using more realistic exposures (30-minute, 1-hour or 24-hours) indicate that the aliphatic acids have sufficient, good or very good skin compatibility.

Animal eye irritation studies indicate that among the aliphatic acids, the C8-12 aliphatic acids are irritating to the eye while the C14-22 aliphatic acids are not irritating.

Eye irritation potential of the ammonium salts does not follow chain length dependence; the C18 ammonium salts are corrosive to the eyes.

Dermal absorption:

The in vitro penetration of C10, C12, C14, C16 and C18 fatty acids (as sodium salt solutions) through rat skin decreases with increasing chain length. At 86.73 ug C16/cm² and 91.84 ug C18/cm², about 0.23% and less than 0.1% of the C16 and C18 soap solutions is absorbed after 24 h exposure, respectively.

Sensitisation:

No sensitisation data were located.

Repeat dose toxicity:

Repeated dose oral (gavage or diet) exposure to aliphatic acids did not result in systemic toxicity with NOAELs greater than the limit dose of 1000 mg/kg bw. .

Mutagenicity

Aliphatic acids do not appear to be mutagenic or clastogenic in vitro or in vivo

Carcinogenicity

No data were located for carcinogenicity of aliphatic fatty acids.

Reproductive toxicity

No effects on fertility or on reproductive organs, or developmental effects were observed in studies on aliphatic acids and the NOAELs correspond to the maximum dose tested. The weight of evidence supports the lack of reproductive and developmental toxicity potential of the aliphatic acids category.

Given the large number of substances in this category, their closely related chemical structure, expected trends in physical chemical properties, and similarity of toxicokinetic properties, both mammalian and aquatic endpoints were filled using read-across to the closest structural analogue, and selecting the most conservative supporting substance effect level.

Structure-activity relationships are not evident for the mammalian toxicity endpoints. That is, the low mammalian toxicity of this category of substances limits the ability to discern structural effects on biological activity. Regardless, the closest structural analogue with the most conservative effect value was selected for read across. Irritation is observed for chain lengths up to a "cut-off" at or near 12 carbons).

Metabolism:

The aliphatic acids share a common degradation pathway in which they are metabolized to acetyl-CoA or other key metabolites in all living systems. Common biological pathways result in structurally similar breakdown products, and are, together with the physico-chemical properties, responsible for similar environmental behavior and essentially identical hazard profiles with regard to human health.

Differences in metabolism or biodegradability of even and odd numbered carbon chain compounds or saturated/unsaturated compounds are not expected; even- and odd-numbered carbon chain compounds, and the saturated and unsaturated compounds are naturally occurring and are expected to be metabolized and biodegraded in the same manner.

The acid and alkali salt forms of the homologous aliphatic acid are expected to have many similar physicochemical and toxicological properties when they become bioavailable; therefore, data read across is used for those instances where data are available for the acid form but not the salt, and vice versa. In the gastrointestinal tract, acids and bases are absorbed in the undissociated (non-ionised) form by simple diffusion or by facilitated diffusion. It is expected that both the acids and the salts will be present in (or converted to) the acid form in the stomach. This means that for both aliphatic acid or aliphatic acid salt, the same compounds eventually enter the small intestine, where equilibrium, as a result of increased pH, will shift towards dissociation (ionised form).

Hence, the situation will be similar for compounds originating from acids and therefore no differences in uptake are anticipated

Note that the saturation or unsaturation level is not a factor in the toxicity of these substances and is not a critical component of the read across process..

Toxicokinetics:

The turnover of the [¹⁴C] surfactants in the rat showed that there was no significant difference in the rate or route of excretion of ¹⁴C given by intraperitoneal or subcutaneous administration. The main route of excretion was as ¹⁴CO₂ in the expired air at 6 h after administration. The remaining material was incorporated in the body. Longer fatty acid chains are more readily incorporated than shorter chains. At ca. 1.55 and 1.64 mg/kg bw, 71% of the C16:0 and 56% of the C18:0 was incorporated and 21% and 38% was excreted as ¹⁴CO₂, respectively.

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

The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling the epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.

For alkyl alcohols C6-13:

This group of products are very similar in terms of physicochemical and toxicological properties. Interpolation of data can be used to assess the alkyl alcohols for which data is not available.

Acute toxicity: All of these alcohols have a low order of toxicity in rats via the oral route. The LD50 for C6-branched and linear alcohols were >3700 mg/kg; LD50s for the C6-8, C7-9, C8-10, C9-11 and C11-14 branched alkyl alcohols were all >2000 mg/kg.

These alcohols have a low order of toxicity via the dermal route. Dermal LD50s were greater than 2600 mg/kg.

Subchronic toxicity: Repeat dose studies indicate these alcohols have a low order of subchronic toxicity by both the oral and dermal route. Further they demonstrate that these alcohols display a consistent degree of subchronic toxicity by these routes

Developmental toxicity: Studies demonstrate that the alcohols are not selective developmental toxicants by either the oral or inhalation route of exposure. Inhalation of alkyl alcohols C6-13 is a primary concern during industrial use, particularly for lower molecular weight alcohols.

Collectively the weight of evidence demonstrates that these alcohols have a low order of maternal toxicity and do not induce signs of developmental toxicity until maternal toxicity is observed. The NOAELs for inhalation reflect the maximum achievable vapour concentration.

Reproductive toxicity: Developmental toxicity studies for several of these alcohols, conducted by the oral route, produce consistent results and demonstrate that these substances do not affect reproductive parameters. Although a slight increase in resorptions was observed in several studies, this occurred only in the highest dose group and in the presence of overt maternal toxicity.

Genotoxicity: The weight of evidence from existing data supports the conclusion that these materials are not genotoxic. Further data to support this assessment comes from a series of alkyl acetates C6-13. Alkyl acetates are produced from alkyl alcohols and undergo metabolism by esterases to produce acetic acid and the corresponding alkyl alcohol. There is no evidence for genotoxicity with these compounds in a variety of strains of *S. typhimurium* in the presence or absence of metabolic activation. C6, C6-8, C7-9 and C11-14 alkyl acetates produced negative results in the Ames test.

Based on data for structurally similar substances these alcohols are not expected to be clastogenic. Alkyl acetates can also be used to predict clastogenic potential of alkyl alcohols. Although there is evidence of cytotoxicity at extremely high doses, no clastogenic activity was seen in a homologous family of alkyl acetates.

Metabolism: Alkyl alcohols are broken down, in the body, by mitochondrial beta-oxidation or by cytochrome P450 omega and alpha-oxidation. The alcohol undergoes various oxidative steps to yield other alcohols, ketones, aldehydes, carboxylic acids and carbon dioxide. Data for monohydric, aliphatic alcohols show a systematic variation according to molecular weight in a manner similar to other homologous series. The body handles aliphatic hydrocarbons in a similar manner via oxidative conversion to alcohols, ketones, and eventual elimination as carbon dioxide and carboxylic acids. The undegraded alcohols can be conjugated either directly or as a metabolite with glucuronic acid, sulfuric acid or glycine and are readily excreted. Intermediate aldehydes may be reactive and bind with DNA and/or proteins.

The Branched Chain Saturated Alcohol (BCSA) group of fragrance ingredients was evaluated for safety.

The 15 materials tested have a low order of acute toxicity.

Following repeated application, seven BCSA tested were of low systemic toxicity

In humans, no evidence of skin irritation was found at concentrations of 2-10%. Undiluted, 11 materials evaluated caused moderate to severe eye irritation. As current end product use levels are between 0.001% and 1.7%, eye irritation is not a concern. The materials have no or low sensitizing potential.

For individuals who are already sensitized, an elicitation reaction is possible. Due to lack of UVA/UVB light-absorbing structures, and review of phototoxic/photoallergy data, the BCSA are not expected to elicit phototoxicity or photoallergy. Studies performed on eight BCSA and three metabolites show no in vivo or in vitro genotoxicity. A valid carcinogenicity study showed that 2-ethyl-1-hexanol is a weak inducer of liver tumours in female mice, however, the relevance of this effect and mode of action to humans is still a matter of debate.

Current opinion holds that there are no safety concerns regarding BCSA under the present levels of use and exposure.

The common characteristic structural elements of the alcohols with saturated branched chain are one hydroxyl group per molecule, a C4-C12 carbon chain with one or several methyl side chains. Two members of the group, 2-ethyl-1-butanol and 2-ethyl-1-hexanol, contain an ethyl side chain. One member contains a methoxy group. Metabolism studies are lacking for this compound, however, a methoxy group is enzymatically not readily cleaved and if it were so, another primary alcohol

group would be formed.

The Research Institute for Fragrance Materials (RIFM) Expert Panel

2-ETHYLHEXANOL

Mirotone GOLD PREMIUM & BIS(1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL)SEBACATE & TINUVIN 1130 & TINUVIN 213 & METHYL 1,2,2,6,6-

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

<p>PENTAMETHYL-4-PIPERIDYL SEBACATE & 2-OCTYL-4-ISOTHIAZOLIN-3-ONE & COBALT 2-ETHYLHEXANOATE</p>	<p>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 GOLD PREMIUM & TINUVIN 213 & METHYL 1,2,2,6,6-PENTAMETHYL-4-PIPERIDYL SEBACATE & COBALT 2-ETHYLHEXANOATE & ZIRCONIUM 2-ETHYLHEXANOATE</p>	<p>No significant acute toxicological data identified in literature search.</p>
<p>Mirotone GOLD PREMIUM & ETHYLENE GLYCOL MONOBUTYL ETHER</p>	<p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs): Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates. EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.</p> <p>Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m3) for EGHE, LC50 > 400ppm (2620 mg/m3) for EGBEA to LC50 > 2132 ppm (9061 mg/m3) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitisers in experimental animals or humans. Signs of acute toxicity in rats, mice and rabbits are consistent with haemolysis (with the exception of EGHE) and non-specific CNS depression typical of organic solvents in general. Alkoxyacetic acid metabolites, propoxyacetic acid (PAA) and butoxyacetic acid (BAA), are responsible for the red blood cell hemolysis. Signs of toxicity in humans deliberately ingesting cleaning fluids containing 9-22% EGBE are similar to those of rats, with the exception of haemolysis. Although decreased blood haemoglobin and/or haemoglobinuria were observed in some of the human cases, it is not clear if this was due to haemolysis or haemodilution as a result of administration of large volumes of fluid. Red blood cells of humans are many-fold more resistant to toxicity from EGPE and EGBE <i>in vitro</i> than those of rats.</p> <p>Repeat dose toxicity: The fact that the NOAEL for repeated dose toxicity of EGBE is less than that of EGPE is consistent with red blood cells being more sensitive to EGBE than EGPE. Blood from mice, rats, hamsters, rabbits and baboons were sensitive to the effects of BAA <i>in vitro</i> and displayed similar responses, which included erythrocyte swelling (increased haematocrit and mean corpuscular hemoglobin), followed by hemolysis. Blood from humans, pigs, dogs, cats, and guinea pigs was less sensitive to haemolysis by BAA <i>in vitro</i>.</p> <p>Mutagenicity: In the absence and presence of metabolic activation, EGBE tested negative for mutagenicity in Ames tests conducted in <i>S. typhimurium</i> strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. <i>In vitro</i> cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and <i>in vivo</i> micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.</p> <p>Carcinogenicity: In a 2-year inhalation chronic toxicity and carcinogenicity study with EGBE in rats and mice a significant increase in the incidence of liver haemangiosarcomas was seen in male mice and forestomach tumours in female mice. It was decided that based on the mode of action data available, there was no significant hazard for human carcinogenicity</p> <p>Reproductive and developmental toxicity. The results of reproductive and developmental toxicity studies indicate that the glycol ethers in this category are not selectively toxic to the reproductive system or developing fetus, developmental toxicity is secondary to maternal toxicity. The repeated dose toxicity studies in which reproductive organs were examined indicate that the members of this category are not associated with toxicity to reproductive organs (including the testes). Results of the developmental toxicity studies conducted via inhalation exposures during gestation periods on EGPE (rabbits -125, 250, 500 ppm or 531, 1062, or 2125 mg/m3 and rats - 100, 200, 300, 400 ppm or 425, 850, 1275, or 1700 mg/m3), EGBE (rat and rabbit - 25, 50, 100, 200 ppm or 121, 241, 483, or 966 mg/m3), and EGHE (rat and rabbit - 20.8, 41.4, 79.2 ppm or 124, 248, or 474 mg/m3) indicate that the members of the category are not teratogenic. The NOAELs for developmental toxicity are greater than 500 ppm or 2125 mg/m3 (rabbit-EGPE), 100 ppm or 425 mg/m3 (rat-EGPE), 50 ppm or 241 mg/m3 (rat EGBE) and 100 ppm or 483 mg/m3 (rabbit EGBE) and greater than 79.2 ppm or 474 mg/m3 (rat and rabbit-EGHE).</p> <p>Exposure of pregnant rats to ethylene glycol monobutyl ether (2-butoxyethanol) at 100 ppm or rabbits at 200 ppm during organogenesis resulted in maternal toxicity and embryotoxicity including a decreased number of viable implantations per litter. Slight foetotoxicity in the form of poorly ossified or unossified skeletal elements was also apparent in rats. Teratogenic effects were not observed in other species.</p> <p>At least one researcher has stated that the reproductive effects were less than that of other monoalkyl ethers of ethylene glycol.</p> <p>Chronic exposure may cause anaemia, macrocytosis, abnormally large red cells and abnormal red cell fragility. Exposure of male and female rats and mice for 14 weeks to 2 years produced a regenerative haemolytic anaemia and subsequent effects on the haemopoietic system in rats and mice. In addition, 2-butoxyethanol exposures caused increases in the incidence of neoplasms and nonneoplastic lesions (1). The occurrence of the anaemia was concentration-</p>

	dependent and more pronounced in rats and females. In this study it was proposed that 2-butoxyethanol at concentrations of 500 ppm and greater produced an acute disseminated thrombosis and bone infarction in male and female rats as a result of severe acute haemolysis and reduced deformability of erythrocytes or through anoxic damage to endothelial cells that compromise blood flow. In two-year studies, 2-butoxyethanol continued to affect circulating erythroid mass, inducing a responsive anaemia. Rats showed a marginal increase in the incidence of benign or malignant pheochromocytomas (combined) of the adrenal gland. In mice, 2-butoxyethanol exposure resulted in a concentration dependent increase in the incidence of squamous cell papilloma or carcinoma of the forestomach. It was hypothesised that exposure-induced irritation produced inflammatory and hyperplastic effects in the forestomach and that the neoplasia were associated with a continuation of the injury/ degeneration process. Exposure also produced a concentration -dependent increase in the incidence of haemangiosarcoma of the liver of male mice and hepatocellular carcinoma. 1: NTP Toxicology Program Technical report Series 484, March 2000.
ETHYLENE GLYCOL MONOBUTYL ETHER & 2-ETHYLHEXANOL & 2-ETHYLHEXANOIC ACID	The material may produce severe irritation to the eye causing pronounced inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
ETHYLENE GLYCOL MONOBUTYL ETHER & 2-ETHYLHEXANOIC ACID	The material may cause skin irritation after prolonged or repeated exposure and may produce a contact dermatitis (nonallergic). This form of dermatitis is often characterised by skin redness (erythema) and swelling epidermis. Histologically there may be intercellular oedema of the spongy layer (spongiosis) and intracellular oedema of the epidermis.
2-OCTYL-4-ISOTHIAZOLIN-3-ONE & 2-ETHYLHEXANOL & 2-ETHYLHEXANOIC ACID	Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergenic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.

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

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
Mirotone GOLD PREMIUM	Not Available	Not Available	Not Available	Not Available	Not Available
ethylene glycol monobutyl ether	LC50	96	Fish	1-700mg/L	2
	EC50	48	Crustacea	ca.1-800mg/L	2
	EC50	72	Algae or other aquatic plants	1-840mg/L	2
	NOEC	24	Crustacea	>1-mg/L	2
3-iodo-2-propynyl butyl carbamate	LC50	96	Fish	0.067mg/L	2
	EC50	48	Crustacea	0.04mg/L	5
	EC50	72	Algae or other aquatic plants	0.022mg/L	2
	EC10	72	Algae or other aquatic plants	0.0058mg/L	2
	NOEC	72	Algae or other aquatic plants	0.0046mg/L	2

Continued...

Mirotone GOLD PREMIUM

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
	LC50	96	Fish	2.8mg/L	2
	EC50	48	Crustacea	4mg/L	2
	EC50	72	Algae or other aquatic plants	>9mg/L	2
	EC0	48	Crustacea	1mg/L	2
	NOEC	72	Algae or other aquatic plants	0.11mg/L	2
Tinuvin 213	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	2.8mg/L	2
	EC50	48	Crustacea	4mg/L	2
	EC50	72	Algae or other aquatic plants	ca.9mg/L	2
	NOEC	504	Crustacea	0.23mg/L	2
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	
2-octyl-4-isothiazolin-3-one	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.047mg/L	4
	EC50	48	Crustacea	0.18mg/L	4
	EC50	96	Algae or other aquatic plants	0.146mg/L	3
	BCF	1608	Fish	0.05mg/L	4
NOEC	504	Crustacea	0.035mg/L	2	
cobalt 2-ethylhexanoate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.406mg/L	2
	EC50	48	Crustacea	0.002-0.618mg/L	2
	EC50	96	Algae or other aquatic plants	0.071-0.314mg/L	2
NOEC	96	Crustacea	0.001-0.2819mg/L	2	
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	
2-ethylhexanol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	<0.075mg/L	4
	EC50	48	Crustacea	39mg/L	2
	EC50	72	Algae or other aquatic plants	11.5mg/L	2
	EC10	72	Algae or other aquatic plants	=1.3mg/L	1
NOEC	48	Algae or other aquatic plants	=10mg/L	1	
2-ethylhexanoic acid	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	48.777mg/L	3
	EC50	48	Crustacea	85.4mg/L	2
	EC50	96	Algae or other aquatic plants	=41mg/L	1
NOEC	504	Crustacea	18mg/L	2	
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 ethylene glycol monoalkyl ethers and their acetates:

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

Environmental fate:

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

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

Ecotoxicity:

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

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

For glycol ethers:

Environmental fate:

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

Ecotoxicity:

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

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

In air ammonia is persistent whilst, in water, it biodegrades rapidly to nitrate, producing a high oxygen demand. Ammonia is strongly adsorbed to soil.

Ammonia is non-persistent in water (half-life 2 days) and is moderately toxic to fish under normal temperature and pH conditions. Ammonia is harmful to aquatic life at low concentrations but does not concentrate in the food chain. Ammonium ions may be toxic to fish at 0.3 mg/l

Drinking Water Standards:

0.5 mg/l (UK max.)

1.5 mg/l (WHO Levels)

Soil Guidelines: none available.

Air Quality Standards: none available.

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation
ethylene glycol monobutyl ether	LOW (BCF = 2.51)
3-iodo-2-propynyl butyl carbamate	LOW (LogKOW = 2.4542)
2-octyl-4-isothiazolin-3-one	LOW (LogKOW = 2.561)
2-ethylhexanol	LOW (BCF = 27)
2-ethylhexanoic acid	LOW (LogKOW = 2.64)

Mobility in soil

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

3-iodo-2-propynyl butyl carbamate	LOW (KOC = 365.3)
2-octyl-4-isothiazolin-3-one	LOW (KOC = 2120)
2-ethylhexanol	LOW (KOC = 26.01)
2-ethylhexanoic acid	LOW (KOC = 24.06)

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 hazardous substance is disposed in accordance with the Hazardous Substances (Disposal) Notice 2017

Disposal Requirements

Packages that have been in direct contact with the hazardous substance must be only disposed if the hazardous substance was appropriately removed and cleaned out from the package.

The package must be disposed according to the manufacturer's directions taking into account the material it is made of.

Packages which hazardous content have been appropriately treated and removed may be recycled.

The hazardous substance must only be disposed if it has been treated by a method that changed the characteristics or composition of the substance and it is no longer hazardous.

Only dispose to the environment if a tolerable exposure limit has been set for the substance.

Only deposit the hazardous substance into or onto a landfill or sewage facility or incinerator, where the hazardous substance can be handled and treated appropriately.

SECTION 14 TRANSPORT INFORMATION

Labels Required

Marine Pollutant	NO Not Applicable
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
HSR002670	Surface Coatings and Colourants (Subsidiary Hazard) Group Standard 2017

ETHYLENE GLYCOL MONOBUTYL ETHER(111-76-2) IS FOUND ON THE FOLLOWING REGULATORY LISTS

GESAMP/EHS Composite List - GESAMP Hazard Profiles	New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals
IMO IBC Code Chapter 17: Summary of minimum requirements	New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data
IMO MARPOL 73/78 (Annex II) - List of Other Liquid Substances	New Zealand Inventory of Chemicals (NZIoC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs	New Zealand Workplace Exposure Standards (WES)
International Air Transport Association (IATA) Dangerous Goods Regulations	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
International Maritime Dangerous Goods Requirements (IMDG Code)	

3-iodo-2-propynyl butyl carbamate(55406-53-6) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations	New Zealand Inventory of Chemicals (NZIoC)
International Maritime Dangerous Goods Requirements (IMDG Code)	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)
New Zealand Hazardous Substances and New Organisms (HSNO) Act - Classification of Chemicals - Classification Data	

BIS(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate(41556-26-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits
International Maritime Dangerous Goods Requirements (IMDG Code)	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 3 Segregation requirements for dangerous goods
New Zealand Inventory of Chemicals (NZIoC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

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

International Air Transport Association (IATA) Dangerous Goods Regulations	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits
International Maritime Dangerous Goods Requirements (IMDG Code)	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 3 Segregation requirements for dangerous goods
New Zealand Inventory of Chemicals (NZIoC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

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

International Air Transport Association (IATA) Dangerous Goods Regulations	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits
International Maritime Dangerous Goods Requirements (IMDG Code)	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 3 Segregation requirements for dangerous goods
New Zealand Inventory of Chemicals (NZIoC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

METHYL 1,2,2,6,6-pentamethyl-4-piperidyl sebacate(82919-37-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1 Quantity limits
International Maritime Dangerous Goods Requirements (IMDG Code)	New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 3 Segregation requirements for dangerous goods
New Zealand Inventory of Chemicals (NZIoC)	United Nations Recommendations on the Transport of Dangerous Goods Model Regulations (English)

2-octyl-4-isothiazolin-3-one(26530-20-1) IS FOUND ON THE FOLLOWING REGULATORY LISTS

International Air Transport Association (IATA) Dangerous Goods Regulations
 International Maritime Dangerous Goods Requirements (IMDG Code)
 New Zealand Hazardous Substances and New Organisms (HSNO) Act -
 Classification of Chemicals

New Zealand Hazardous Substances and New Organisms (HSNO) Act -
 Classification of Chemicals - Classification Data
 New Zealand Inventory of Chemicals (NZIoC)
 United Nations Recommendations on the Transport of Dangerous Goods
 Model Regulations (English)

COBALT 2-ETHYLHEXANOATE(13586-82-8) IS FOUND ON THE FOLLOWING REGULATORY LISTS

GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in
 Bulk
 International Agency for Research on Cancer (IARC) - Agents Classified
 by the IARC Monographs
 International Air Transport Association (IATA) Dangerous Goods Regulations

International Maritime Dangerous Goods Requirements (IMDG Code)
 New Zealand Inventory of Chemicals (NZIoC)
 New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1
 Quantity limits
 United Nations Recommendations on the Transport of Dangerous Goods
 Model Regulations (English)

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)

2-ETHYLHEXANOL(104-76-7) IS FOUND ON THE FOLLOWING REGULATORY LISTS

GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in
 Bulk
 IMO Provisional Categorization of Liquid Substances - List 2: Pollutant only
 mixtures containing at least 99% by weight of components already
 assessed by IMO
 IMO Provisional Categorization of Liquid Substances - List 3: (Trade-named)
 mixtures containing at least 99% by weight of components already
 assessed by IMO, presenting safety hazards
 International Air Transport Association (IATA) Dangerous Goods Regulations

International Maritime Dangerous Goods Requirements (IMDG Code)
 New Zealand Hazardous Substances and New Organisms (HSNO) Act -
 Classification of Chemicals
 New Zealand Hazardous Substances and New Organisms (HSNO) Act -
 Classification of Chemicals - Classification Data
 New Zealand Inventory of Chemicals (NZIoC)
 New Zealand Land Transport Rule: Dangerous Goods 2005 - Schedule 1
 Quantity limits
 United Nations Recommendations on the Transport of Dangerous Goods
 Model Regulations (English)

2-ETHYLHEXANOIC ACID(149-57-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

GESAMP/EHS Composite List - GESAMP Hazard Profiles
 IMO IBC Code Chapter 17: Summary of minimum requirements
 IMO MARPOL (Annex II) - List of Noxious Liquid Substances Carried in
 Bulk

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

Hazardous Substance Location

Subject to the Health and Safety at Work (Hazardous Substances) Regulations 2017.

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

Certified Handler

Subject to Part 4 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Class of substance	Quantities
Not Applicable	Not Applicable

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AICS	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Canada - DSL	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Canada - NDSL	No (3-iodo-2-propynyl butyl carbamate; bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; 2-octyl-4-isothiazolin-3-one; 2-ethylhexanol; Tinuvin 213; zirconium 2-ethylhexanoate; Tinuvin 1130; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; ethylene glycol monobutyl ether; cobalt 2-ethylhexanoate; 2-ethylhexanoic acid; Ingredients determined not to be hazardous) Non-disclosed ingredients
China - IECSC	No (Ingredients determined not to be hazardous) Non-disclosed ingredients

Europe - EINEC / ELINCS / NLP	No (Tinuvin 213; Tinuvin 1130; Ingredients determined not to be hazardous) Non-disclosed ingredients
Japan - ENCS	No (Tinuvin 213; Tinuvin 1130; Ingredients determined not to be hazardous) Non-disclosed ingredients
Korea - KECL	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
New Zealand - NZIoC	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Philippines - PICCS	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
USA - TSCA	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Taiwan - TCSI	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Mexico - INSQ	No (Tinuvin 213; zirconium 2-ethylhexanoate; Tinuvin 1130; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; Ingredients determined not to be hazardous) Non-disclosed ingredients
Vietnam - NCI	No (Ingredients determined not to be hazardous) Non-disclosed ingredients
Russia - ARIPS	No (bis(1,2,2,6,6-pentamethyl-4-piperidyl)sebacate; Tinuvin 213; Tinuvin 1130; methyl 1,2,2,6,6-pentamethyl-4-piperidyl sebacate; Ingredients determined not to be hazardous) Non-disclosed ingredients
Thailand - TECI	No (2-octyl-4-isothiazolin-3-one; zirconium 2-ethylhexanoate; Ingredients determined not to be hazardous) Non-disclosed ingredients
Legend:	<i>Yes = All declared ingredients are on the inventory No = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)</i>

SECTION 16 OTHER INFORMATION

Revision Date	24/08/2017
Initial Date	Not Available

Other information

Ingredients with multiple cas numbers

Name	CAS No
cobalt 2-ethylhexanoate	13586-82-8, 136-52-7
zirconium 2-ethylhexanoate	22464-99-9, 94581-21-2
2-ethylhexanol	104-76-7, 111675-57-1, 68526-83-0, 68526-84-1

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

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

Definitions and abbreviations

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

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