



MIROTONE

MIROTEC WB 8060 Clear T/Coat

Mirotone (NZ) Ltd

Chemwatch: 4731-35
Version No: 6.1.1.1
Safety Data Sheet according to HSNO Regulations

Chemwatch Hazard Alert Code: 2

Issue Date: 04/03/2014
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Initial Date: Not Available
L.GHS.NZL.EN

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

Product Identifier

Product name	MIROTEC WB 8060 Clear T/Coat
Synonyms	MIROTEC WB 8060 Clear T/Coat 8060-1, MIROTEC WB 8060 Clear T/Coat 8060-3, MIROTEC WB 8060 Clear T/Coat 8060-6, MIROTEC WB 8060 Clear T/Coat 8060-9, Product Code: 8060
Other means of identification	Not Available

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

Relevant identified uses	Clear coating for furniture.
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Details of the supplier of the safety data sheet

Registered company name	Mirotone (NZ) Ltd
Address	32 Cryers Road New Zealand
Telephone	0800 FINISH (0800 346 474)
Fax	0800 346 434
Website	mirotone.com
Email	information@mirotone.co.nz

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	111
Other emergency telephone numbers	Not Available

CHEMWATCH EMERGENCY RESPONSE

Primary Number	Alternative Number 1	Alternative Number 2
+800 2436 2255	+612 9186 1132	Not Available

Once connected and if the message is not in your preferred language then please dial 01

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

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

CHEMWATCH HAZARD RATINGS

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

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

Classification [1]	Skin Sensitizer Category 1, Acute Aquatic Hazard Category 2
Legend:	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI
Determined by Chemwatch using GHS/HSNO criteria	6.5B (contact), 9.1D

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Label elements

GHS label elements	
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SIGNAL WORD

WARNING

Hazard statement(s)

H317	May cause an allergic skin reaction.
H401	Toxic to aquatic life

Precautionary statement(s) Prevention

P280	Wear protective gloves/protective clothing/eye protection/face protection.
P261	Avoid breathing mist/vapours/spray.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P363	Wash contaminated clothing before reuse.
P302+P352	IF ON SKIN: Wash with plenty of soap and water.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.

Precautionary statement(s) Storage

Not Applicable

Precautionary statement(s) Disposal

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

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
Various	30-60	<u>acrylic resin</u>
Not Available	10-30	additive
Not Available	0-10	pigment
2634-33-5	0-10	<u>1,2-benzisothiazoline-3-one</u>
Not Available	NotSpec.	other ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

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

Description of first aid measures

Eye Contact	<p>If this product comes in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Wash out immediately with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If skin contact occurs:</p> <ul style="list-style-type: none"> ▶ Immediately remove all contaminated clothing, including footwear. ▶ Flush skin and hair with running water (and soap if available). ▶ Seek medical attention in event of irritation.
Inhalation	<ul style="list-style-type: none"> ▶ If fumes or combustion products are inhaled remove from contaminated area. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ If swallowed do NOT induce vomiting. ▶ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▶ Observe the patient carefully. ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▶ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Continued...

1,2-Benzisothiazoline-3-one (BIT) is rapidly metabolised in animals. Neither the substance nor its metabolites accumulate in the liver or adipose tissue. Excretion is mainly via the urine. The main metabolite is o-methylsulfanylbenzamide

Treat symptomatically.

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

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

For acute or short term repeated exposures to ethylene glycol:

- ▶ Early treatment of ingestion is important. Ensure emesis is satisfactory.
- ▶ Test and correct for metabolic acidosis and hypocalcaemia.
- ▶ Apply sustained diuresis when possible with hypertonic mannitol.
- ▶ Evaluate renal status and begin haemodialysis if indicated. [I.L.O.]
- ▶ Rapid absorption is an indication that emesis or lavage is effective only in the first few hours. Cathartics and charcoal are generally not effective.
- ▶ Correct acidosis, fluid/electrolyte balance and respiratory depression in the usual manner. Systemic acidosis (below 7.2) can be treated with intravenous sodium bicarbonate solution.
- ▶ Ethanol therapy prolongs the half-life of ethylene glycol and reduces the formation of toxic metabolites.
- ▶ Pyridoxine and thiamine are cofactors for ethylene glycol metabolism and should be given (50 to 100 mg respectively) intramuscularly, four times per day for 2 days.
- ▶ Magnesium is also a cofactor and should be replenished. The status of 4-methylpyrazole, in the treatment regime, is still uncertain. For clearance of the material and its metabolites, haemodialysis is much superior to peritoneal dialysis.

[Ellenhorn and Barceloux: Medical Toxicology]

It has been suggested that there is a need for establishing a new biological exposure limit before a workshift that is clearly below 100 mmol ethoxy-acetic acids per mole creatinine in morning urine of people occupationally exposed to ethylene glycol ethers. This arises from the finding that an increase in urinary stones may be associated with such exposures.

Laitinen J., et al: *Occupational & Environmental Medicine* 1996; 53, 595-600

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

- ▶ Water spray or fog.
- ▶ Foam.
- ▶ Dry chemical powder.
- ▶ BCF (where regulations permit).
- ▶ Carbon dioxide.

Special hazards arising from the substrate or mixture

- | | |
|-----------------------------|--|
| Fire Incompatibility | ▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result |
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ Wear full body protective clothing with breathing apparatus. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ Avoid spraying water onto liquid pools. ▶ 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.
Fire/Explosion Hazard	<ul style="list-style-type: none"> ▶ Combustible. ▶ Slight fire hazard when exposed to heat or flame. ▶ Heating may cause expansion or decomposition leading to violent rupture of containers. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). ▶ May emit acid smoke. ▶ Mists containing combustible materials may be explosive. <p>Combustion products include; carbon dioxide (CO₂) silicon dioxide (SiO₂) other pyrolysis products typical of burning organic material May emit poisonous fumes. May emit corrosive fumes.</p>

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	<ul style="list-style-type: none"> ▶ Remove all ignition sources. ▶ 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	<ul style="list-style-type: none"> ▶ Absorb or contain isothiazolinone liquid spills with sand, earth, inert material or vermiculite. ▶ The absorbent (and surface soil to a depth sufficient to remove all of the biocide) should be shovelled into a drum and treated with an 11% solution of sodium metabisulfite (Na₂S₂O₅) or sodium bisulfite (NaHSO₃), or 12% sodium sulfite (Na₂SO₃) and 8% hydrochloric acid (HCl). ▶ Glutathione has also been used to inactivate the isothiazolinones. ▶ Use 20 volumes of decontaminating solution for each volume of biocide, and let containers stand for at least 30 minutes to deactivate microbicide before disposal. ▶ If contamination of drains or waterways occurs, advise emergency services. ▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ 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. ▶ 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.
Other information	<ul style="list-style-type: none"> ▶ Store in original containers. ▶ Keep containers securely sealed. ▶ No smoking, naked lights or ignition sources. ▶ Store in a cool, dry, well-ventilated area. ▶ Store away from incompatible materials and foodstuff containers. ▶ Protect containers against physical damage and check regularly for leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Metal can or drum ▶ Packaging as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Avoid reaction with oxidising agents, bases and strong reducing agents.

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
MIROTEC WB 8060 Clear T/Coat	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
acrylic resin	Not Available	Not Available
additive	Not Available	Not Available
pigment	Not Available	Not Available
1,2-benzisothiazoline-3-one	Not Available	Not Available
other ingredients determined not to be hazardous	Not Available	Not Available

MATERIAL DATA

1,2-Benzisothiazoline-3-one (BIT) produces sensitising effects and causes skin irritation at concentrations of 0.05%. Solutions containing the substance should contain levels considerably lower than 0.05%.

CEL TWA: 0.1 mg/m³; STEL 0.3 mg/m³ total isothiazolinones (Rohm and Haas)

(CEL = Chemwatch Exposure Limit)

For amorphous crystalline silica (precipitated silicic acid):

Amorphous crystalline silica shows little potential for producing adverse effects on the lung and exposure standards should reflect a particulate of low intrinsic toxicity. Mixtures of amorphous silicas/ diatomaceous earth and crystalline silica should be monitored as if they comprise only the crystalline forms.

The dusts from precipitated silica and silica gel produce little adverse effect on pulmonary functions and are not known to produce significant disease or toxic effect.

IARC has classified silica, amorphous as Group 3: **NOT** classifiable as to its carcinogenicity to humans.

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

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)

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p>
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General exhaust is adequate under normal operating conditions. Local exhaust ventilation may be required in specific circumstances. If risk of overexposure exists, wear approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

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

Within each range the appropriate value depends on:

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

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

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.

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.

- ▶ Butyl rubber gloves
- ▶ Nitrile rubber gloves

Body protection

See Other protection below

Other protection

- ▶ Overalls.
- ▶ P.V.C. apron.
- ▶ Barrier cream.
- ▶ Skin cleansing cream.
- ▶ Eye wash unit.

Thermal hazards

Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Respiratory protection

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

Continued...

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:

MIROTEC WB 8060 Clear T/Coat

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

* CPI - Chemwatch Performance Index

A: Best Selection

B: Satisfactory; may degrade after 4 hours continuous immersion

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

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

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

Z88 or national equivalent)

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

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

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

^ - Full-face

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

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

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Colourless milky liquid with a characteristic odour; miscible with water.		
Physical state	Liquid	Relative density (Water = 1)	1.05-1.07
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	100	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>100 (CC)	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)	65-70 (by wt)
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water (g/L)	Miscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

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	<p>The material is not thought to produce either adverse health effects or irritation of the respiratory tract following inhalation (as classified by EC Directives using animal models). Nevertheless, adverse systemic effects have been produced following exposure of animals by at least one other route and good hygiene practice requires that exposure be kept to a minimum and that suitable control measures be used in an occupational setting.</p> <p>Ethylene glycol monobutyl ether (2-butoxyethanol) and its metabolite butoxyacetic acid are haemolytic agents, causing red blood cell destruction.</p> <p>On the basis of industrial experience and volunteer short-term exposure humans are shown to be less susceptible than experimental animals to exposure. In 8-hour exposures at concentrations of 200 or 100 ppm no objective effects were seen other than raised urinary excretion of the metabolite butoxyacetic acid. No increased osmotic fragility of the red blood cell is observed. Subjectively these concentrations were uncomfortable with mild eye, nose and throat irritation occurring. No clinical signs of adverse effects nor subjective complaints were produced when male volunteers were exposed for 2 hours to 20 ppm during light physical exercise. Other studies have established that the most sensitive indicators of toxic effect observed from many of the glycol ethers is an increase in erythrocyte osmotic fragility in rats. This appears to be related to the development of haemoglobinuria at higher exposure levels.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual.</p> <p>Rats given 900 mg/kg of 1,2-benzisothiazoline-3-one showed severe symptoms of intoxication including piloerection, pinched in sides, dehydration, hypothermia, and reduced activity.</p> <p>Isothiazolinones are moderately to highly toxic by oral administration. The major signs of toxicity were severe gastric irritation, lethargy, and ataxia</p> <p>Severe acute exposure to ethylene glycol monobutyl ether, by ingestion, may cause kidney damage, haemoglobinuria, (blood in urine) and is potentially fatal.</p>
Skin Contact	<p>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.</p> <p>Solutions of 0.5% strength 1,2-benzisothiazoline-3-one (BIT) are irritating to the skin. Allergic effects also begin at 0.05% and have been confirmed in a series of case and patch test studies. When the substance was applied to human volunteers under an occlusive patch the maximum tolerated doses was 0.05%.</p> <p>Five hours after application of 0.1% (1000 ppm) one person showed moderate erythema with papule development which was interpreted as a reaction to the sticking plaster; in four persons there was mild reddening of the skin. The reaction had ameliorated in several persons after 72 hours. A second application produced various severe dermal reactions (erythema and papules) in 8 persons. A third application to several of the group produced erythema.</p> <p>Provocation tests with BIT showed the material to be sensitising. Of 20 metal workers with dermatitis, 4 were shown to have been sensitised to BIT in cutting oils. Cases of contact eczema in workers producing polyacrylate emulsions for paints and wax polish, in which BIT was the preservative, have been described. Epicutaneous challenge tests to BIT were positive. Similar findings have been described in the paper-manufacturing industry, in the rubber industry, in the control laboratory of a chemical plant and among workers producing ceramic moulds in which BIT was added to the mould oil</p> <p>Aqueous solutions of isothiazolinones may be irritating or even corrosive depending on concentration. Solutions containing more than 0.5% (5000 ppm active substance) may produce severe irritation of human skin whilst solutions containing more than 100 ppm may irritate the skin.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Ethylene glycol monobutyl ether (2-butoxyethanol) penetrates the skin easily and toxic effects via this route may be more likely than by inhalation. Percutaneous uptake rate in the guinea pig was estimated to be 0.25 umole/min/cm².</p>
Eye	<p>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.</p> <p>Some nonionic surfactants may produce a localised anaesthetic effect on the cornea; this may effectively eliminate the warning discomfort produced by other substances and lead to corneal injury. Irritant effects range from minimal to severe dependent on the nature of the surfactant, its concentration and the duration of contact. Pain and corneal damage represent the most severe manifestation of irritation.</p> <p>Solutions containing isothiazolinones may produce corrosion of the mucous membranes and cornea. Instillation of 0.1 ml of an aqueous solution containing 560 ppm isothiazolinone into rabbit eye did not produce irritation whereas concentrations, typically around 3% and 5.5 %, were severely irritating or corrosive to the eye.. Symptoms included clouding of the cornea, chemosis and swelling of the eyelids.</p> <p>When instilled in rabbit eyes ethylene glycol monobutyl ether produced pain, conjunctival irritation, and transient corneal injury.</p>
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>In a teratogenic study in rats concentrations of up to 40 mg/kg 1,2-benzisothiazoline-3-one (BIT) were neither embryotoxic nor teratogenic. The material is not mutagenic. In a 2-year carcinogenicity study with rats, BIT did not produce excess tumours. The results derived from this test are questionable because no dose series was administered and because there were too few animals.</p> <p>A 90-day study with beagle dogs receiving oral doses showed reduced food consumption and body weight gain as well as mild anaemia, increases in the weights of liver and in male animals, brain and spleen weights.</p> <p>The no-observed-effect-level (NOEL) was given as 165 mg/kg (ie 0.5 BIT in the diet). A 90-day study with rats receiving dietary BIT showed reduced liver and pituitary weights in males. The NOEL was less than 0.1 %.</p> <p>The isothiazolinones are known contact sensitizers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.</p> <p>The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.</p> <p>Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :</p> <ul style="list-style-type: none"> ▶ The strongest sensitizers are the chlorinated isothiazolinones. ▶ There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones. ▶ There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones. ▶ Although classified as sensitizers, the nonchlorinated isothiazolinones are considerably less potent sensitizers than are the chlorinated isothiazolinones. ▶ By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced. ▶ Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons. ▶ Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available. <p>* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196</p> <p>Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in <i>Salmonella typhimurium</i> strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells <i>in vitro</i> and of cytogenetic effects and DNA-binding <i>in vivo</i>. The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.</p> <p>A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.</p>

MIROTEC WB 8060 Clear T/Coat

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

The synthetic, amorphous silicas are believed to represent a very greatly reduced silicosis hazard compared to crystalline silicas and are considered to be nuisance dusts.

When heated to high temperature and a long time, amorphous silica can produce crystalline silica on cooling. Inhalation of dusts containing crystalline silicas may lead to silicosis, a disabling pulmonary fibrosis that may take years to develop. Discrepancies between various studies showing that fibrosis associated with chronic exposure to amorphous silica and those that do not may be explained by assuming that diatomaceous earth (a non-synthetic silica commonly used in industry) is either weakly fibrogenic or nonfibrogenic and that fibrosis is due to contamination by crystalline silica content

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

MIROTEC WB 8060 Clear T/Coat	TOXICITY	IRRITATION
	Not Available	Not Available
acrylic resin	TOXICITY	IRRITATION
	Not Available	Not Available
1,2-benzisothiazoline-3-one	TOXICITY	IRRITATION
	Oral (rat) LD50: 670 mg/kg ^[2]	*MAK Documentation Nil reported

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

MIROTEC WB 8060 Clear T/Coat	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>No significant acute toxicological data identified in literature search.</p> <p>Acute toxicity data show that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that this chemical is a severe eye irritant. Irritation to the skin from acute data show only mild skin irritation, but repeated dermal application indicated a more significant skin irritation response.</p> <p>The neurotoxicity observed in the rat acute oral toxicity study (piloerection and upward curvature of the spine at 300 mg/kg and above; decreased activity, prostration, decreased abdominal muscle tone, reduced righting reflex, and decreased rate and depth of breathing at 900 mg/kg) and the acute dermal toxicity study (upward curvature of the spine was observed in increased incidence, but this was absent after day 5 post-dose at a dose of 2000 mg/kg) were felt to be at exposures in excess of those expected from the use pattern of this pesticide and that such effects would not be observed at estimated exposure doses.</p> <p>Subchronic oral toxicity studies showed systemic effects after repeated oral administration including decreased body weight, increased incidence of forestomach hyperplasia, and non-glandular stomach lesions in rats. In dogs, the effects occurred at lower doses than in rats, and included alterations in blood chemistry (decreased plasma albumin, total protein, and alanine aminotransferase) and increased absolute liver weight.</p> <p>Developmental toxicity studies were conducted in rats with maternal effects including decreased body weight gain, decreased food consumption, and clinical toxicity signs (audible breathing, haircoat staining of the anogenital region, dry brown material around the nasal area) as well as increased mortality. Developmental effects consisted of increases in skeletal abnormalities (extra sites of ossification of skull bones, unossified sternbrae) but not external or visceral abnormalities.</p> <p>Reproductive toxicity: In a two-generation reproduction study, parental toxicity was observed at 500 ppm and was characterized by lesions in the stomach. In pups, toxic effects were reported at 1000 ppm and consisted of preputial separation in males and impaired growth and survival in both sexes. The reproduction study did not show evidence of increased susceptibility of offspring.</p> <p>For ethylene glycol monoalkyl ethers and their acetates (EGMAEs):</p> <p>Typical members of this category are ethylene glycol propylene ether (EGPE), ethylene glycol butyl ether (EGBE) and ethylene glycol hexyl ether (EGHE) and their acetates.</p> <p>EGMAEs are substrates for alcohol dehydrogenase isozyme ADH-3, which catalyzes the conversion of their terminal alcohols to aldehydes (which are transient metabolites). Further, rapid conversion of the aldehydes by aldehyde dehydrogenase produces alkoxyacetic acids, which are the predominant urinary metabolites of mono substituted glycol ethers.</p> <p>Acute Toxicity: Oral LD50 values in rats for all category members range from 739 (EGHE) to 3089 mg/kg bw (EGPE), with values increasing with decreasing molecular weight. Four to six hour acute inhalation toxicity studies were conducted for these chemicals in rats at the highest vapour concentrations practically achievable. Values range from LC0 > 85 ppm (508 mg/m³) for EGHE, LC50 > 400ppm (2620 mg/m³) for EGBEA to LC50 > 2132 ppm (9061 mg/m³) for EGPE. No lethality was observed for any of these materials under these conditions. Dermal LD50 values in rabbits range from 435 mg/kg bw (EGBE) to 1500 mg/kg bw (EGBEA). Overall these category members can be considered to be of low to moderate acute toxicity. All category members cause reversible irritation to skin and eyes, with EGBEA less irritating and EGHE more irritating than the other category members. EGPE and EGBE are not sensitizers 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 S.</p>

typhimurium strains TA97, TA98, TA100, TA1535 and TA1537 and EGHE tested negative in strains TA98, TA100, TA1535, TA1537 and TA1538. *In vitro* cytogenetic and sister chromatid exchange assays with EGBE and EGHE in Chinese Hamster Ovary Cells with and without metabolic activation and *in vivo* micronucleus tests with EGBE in rats and mice were negative, indicating that these glycol ethers are not genotoxic.

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

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).

For silica amorphous:

When experimental animals inhale synthetic amorphous silica (SAS) dust, it dissolves in the lung fluid and is rapidly eliminated. If swallowed, the vast majority of SAS is excreted in the faeces and there is little accumulation in the body. Following absorption across the gut, SAS is eliminated via urine without modification in animals and humans. SAS is not expected to be broken down (metabolised) in mammals.

After ingestion, there is limited accumulation of SAS in body tissues and rapid elimination occurs. Intestinal absorption has not been calculated, but appears to be insignificant in animals and humans. SASs injected subcutaneously are subjected to rapid dissolution and removal. There is no indication of metabolism of SAS in animals or humans based on chemical structure and available data. In contrast to crystalline silica, SAS is soluble in physiological media and the soluble chemical species that are formed are eliminated via the urinary tract without modification.

Both the mammalian and environmental toxicology of SASs are significantly influenced by the physical and chemical properties, particularly those of solubility and particle size. SAS has no acute intrinsic toxicity by inhalation. Adverse effects, including suffocation, that have been reported were caused by the presence of high numbers of respirable particles generated to meet the required test atmosphere. These results are not representative of exposure to commercial SASs and should not be used for human risk assessment. Though repeated exposure of the skin may cause dryness and cracking, SAS is not a skin or eye irritant, and it is not a sensitiser.

Repeated-dose and chronic toxicity studies confirm the absence of toxicity when SAS is swallowed or upon skin contact.

Long-term inhalation of SAS caused some adverse effects in animals (increases in lung inflammation, cell injury and lung collagen content), all of which subsided after exposure.

Numerous repeated-dose, subchronic and chronic inhalation toxicity studies have been conducted with SAS in a number of species, at airborne concentrations ranging from 0.5 mg/m³ to 150 mg/m³. Lowest-observed adverse effect levels (LOAELs) were typically in the range of 1 to 50 mg/m³.

When available, the no-observed adverse effect levels (NOAELs) were between 0.5 and 10 mg/m³. The difference in values may be explained by different particle size, and therefore the number of particles administered per unit dose. In general, as particle size decreases so does the NOAEL/LOAEL.

Neither inhalation nor oral administration caused neoplasms (tumours). SAS is not mutagenic *in vitro*. No genotoxicity was detected in *in vivo* assays.

SAS does not impair development of the foetus. Fertility was not specifically studied, but the reproductive organs in long-term studies were not affected.

In humans, SAS is essentially non-toxic by mouth, skin or eyes, and by inhalation. Epidemiology studies show little evidence of adverse health effects due to SAS. Repeated exposure (without personal protection) may cause mechanical irritation of the eye and drying/cracking of the skin.

There is no evidence of cancer or other long-term respiratory health effects (for example, silicosis) in workers employed in the manufacture of SAS.

Respiratory symptoms in SAS workers have been shown to correlate with smoking but not with SAS exposure, while serial pulmonary function values and chest radiographs are not adversely affected by long-term exposure to SAS.

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 diarrhoea 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

	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>Cancer: No studies were located regarding cancer effects in humans or animals after dermal exposure to ethylene glycol.</p> <p>Genotoxic Effects: Studies in humans have not addressed the genotoxic effects of ethylene glycol. However, available <i>in vivo</i> and <i>in vitro</i> laboratory studies provide consistently negative genotoxicity results for ethylene glycol.</p>
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	No significant acute toxicological data identified in literature search.
ACRYLIC RESIN	CAUTION: The chronic health effects of acrylic monomers are under review.
	Use good occupational work practices to avoid personal contact.

	<p>The following information refers to contact allergens as a group and may not be specific to this product.</p> <p>Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.</p> <p>Acute toxicity data show that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that this chemical is a severe eye irritant. Irritation to the skin from acute data show only mild skin irritation, but repeated dermal application indicated a more significant skin irritation response.</p> <p>The neurotoxicity observed in the rat acute oral toxicity study (piloerection and upward curvature of the spine at 300 mg/kg and above; decreased activity, prostration, decreased abdominal muscle tone, reduced righting reflex, and decreased rate and depth of breathing at 900 mg/kg) and the acute dermal toxicity study (upward curvature of the spine was observed in increased incidence, but this was absent after day 5 post-dose at a dose of 2000 mg/kg) were felt to be at exposures in excess of those expected from the use pattern of this pesticide and that such effects would not be observed at estimated exposure doses.</p> <p>Subchronic oral toxicity studies showed systemic effects after repeated oral administration including decreased body weight, increased incidence of forestomach hyperplasia, and non-glandular stomach lesions in rats. In dogs, the effects occurred at lower doses than in rats, and included alterations in blood chemistry (decreased plasma albumin, total protein, and alanine aminotransferase) and increased absolute liver weight.</p> <p>Developmental toxicity studies were conducted in rats with maternal effects including decreased body weight gain, decreased food consumption, and clinical toxicity signs (audible breathing, haircoat staining of the anogenital region, dry brown material around the nasal area) as well as increased mortality. Developmental effects consisted of increases in skeletal abnormalities (extra sites of ossification of skull bones, unossified sternbrae) but not external or visceral abnormalities.</p> <p>Reproductive toxicity: In a two-generation reproduction study, parental toxicity was observed at 500 ppm and was characterized by lesions in the stomach. In pups, toxic effects were reported at 1000 ppm and consisted of preputial separation in males and impaired growth and survival in both sexes. The reproduction study did not show evidence of increased susceptibility of offspring.</p>
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Acute Toxicity	☒	Carcinogenicity	☒
Skin Irritation/Corrosion	☒	Reproductivity	☒
Serious Eye Damage/Irritation	☒	STOT - Single Exposure	☒
Respiratory or Skin sensitisation	✓	STOT - Repeated Exposure	☒
Mutagenicity	☒	Aspiration Hazard	☒

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

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

Ingredient	Endpoint	Test Duration (hr)	Species	Value	Source
1,2-benzisothiazoline-3-one	EC50	48	Crustacea	0.062mg/L	4
1,2-benzisothiazoline-3-one	EC50	48	Crustacea	4.4mg/L	4
1,2-benzisothiazoline-3-one	LC50	96	Fish	1.6mg/L	4

Legend:

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

for 1,2-benzisothiazoline-3-one (BIT):

Environmental fate:

Based on environmental fate data, BIT binds moderately with soil and may potentially move with the soil during rainfall events and reach surface waters. Although, BIT has been shown to be hydrolytically stable with a half life of > 30 days, it breaks down fairly quickly in aerobic soils. BIT shows moderate to strong binding to soils, with adsorption Kd values estimated to be between 1.24 and 9.56. However, it breaks down aerobically on the surface soils. Since it has a moderate binding potential to soils, it is not likely to migrate into the ground and there is low potential for ground water contamination. Furthermore, with a Kow value of 20 at 25 deg C, BIT is unlikely to bioaccumulate in aquatic organisms.

Ecotoxicity:

Based on acute toxicity information, 1,2-benzisothiazoline-3-one displays low to moderate toxicity to birds and mammals. It is moderately toxic to freshwater fish and invertebrates, slightly toxic to

marine/estuarine fish, and highly toxic to marine/estuarine invertebrates.

Fish LC50 (96 h): bluegill sunfish 2.7 - 5.1 ppm; rainbow trout 0.77 - 1.4 ppm -

Toxic to fish.

Daphnia magna LC50 (48 h): 1.05 - 7.7 ppm -

Brown Shrimp LC50 (96 h): 38 ppm

Algae EC50 (72 h): 0.37 mg/L -

Very toxic to Algae.

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*.

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

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

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

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

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

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.

For silica amorphous:

Amorphous silica is chemically and biologically inert. It is not biodegradable. Due to its insolubility in water there is a separation at every filtration and sedimentation process.]

Crystalline and/or amorphous silicas are ubiquitous on the earth in soils and sediments, and in living organisms (e.g. diatoms), but only the dissolved form is bioavailable. On a global scale, the level of man-made synthetic amorphous silicas (SAS) represents up to 2.4% of the dissolved silica naturally present in the aquatic environment. The rate of SAS released into the environment during the product life cycle is negligible in comparison with the natural flux of silica in the environment

Untreated SASs have a relatively low water solubility of 1.91 to 2.51 mmol/l (114 - 151 mg/l) and an extremely low vapour pressure (e.g. < 10-3 Pa at 20° C for Aerosil R972). On the basis of these properties it is expected that SAS released into the environment will be distributed mainly into soil/sediment, slightly into water, and probably not at all into air.

With surface-treated SASs, the addition of organosilicon compounds increases the hydrophobicity. Consequently, the water solubility is lower than that of untreated silica. The vapour pressure remains extremely low. Due to the presence of organic substances such as surfactants, salts, acids and alkalis in the environment, it is expected that surface-treated silica will be wetted and then adsorbed onto soils or sediments.

SAS is regarded as an inert substance and is not expected to undergo any transformation in the atmospheric or terrestrial compartment, apart from dissolution by water.

Biodegradability in sewage treatment plant or in surface water is not applicable to inorganic substances like SAS. Therefore the biodegradation endpoint has limited relevance for SAS. In surface modified SASs, the most common treating agents are organosilicon compounds and these generally represent less than 5% of the material. Biodegradation in sewage treatment plant or in surface water is not expected. Some biodegradation in soil may occur by analogy with the behaviour of linear polydimethylsiloxane in this compartment

Ecotoxicity:

Based on available data, SAS is not toxic to environmental organisms (apart from physical desiccation in insects). SAS presents a low risk for adverse effects to the environment.

When hydrophilic SASs (Aerosil 200 and Ultrasil VN3; purity 100% and 98%, respectively), were tested for their acute toxicity to fish and crustaceans, the LC50 and EC50 values were higher than 10,000 mg/l and 1,000 mg/l, respectively.

The zebra fish (*Brachydanio rerio*) test was performed with SAS in suspension, due to the insolubility of the SAS. No mortality was observed for the fish after 96 hours of exposure at 1,000 mg/l and 10,000 mg/l. The test media remained turbid throughout the test, indicating that the limit of solubility of the product was exceeded.

With the water flea (*Daphnia magna*), SAS suspensions exceeding the limit of solubility were tested.; some immobilisation was observed. However, no significant immobilisation was observed when a solution filtered through microfibre glass filter was tested. The observed effects were likely caused by physical hampering of the *Daphnia* due to the presence of undissolved particles.

A surface-treated SAS (Aerosil R974; 99.9% pure) was tested on fish and crustaceans. The LC50 to fish and EC50 to *Daphnia* were found to be higher than 10,000 mg/l and 1,000 mg/l, respectively

The EC50 to algae was found to be higher than 10,000 mg/l filtered suspension The actual dissolved concentrations were not determined. There was no inhibition of the biomass or of the growth rate with the 10,000 mg/l filtered suspension.

The antibacterial effect of pressed and unpressed high purity SAS (Aerosil, unspecified) (0.2 g silica + 0.15 ml bacteria strain suspension) kept at 22 C has been investigated (SAS is sometimes pressed to remove air before transportation). The following micro-organisms were studied: *Escherichia coli*, *Proteus* sp., *Pseudomonas aeruginosa*, *Aerobacter aerogenes*,

Micrococcus pyrogenes aureus, *Streptococcus faecalis*, *Streptococcus pyrogenes humans*, *Corynebacterium diphtheria*, *Candida albicans* and *Bacillus subtilis*. The SAS was contaminated either by hand contact, by saliva droplets or by contact with the atmosphere. Rodshaped gram-negative organisms (*Escherichia coli*, *Bacterium proteus*, *Pseudomonas aeruginosa*

and *Aerobacter aerogenes*) died between 6 hours and 3 days in contact with unpressed SAS. Gram-positive micro-organisms were somewhat more resistant. In addition, the tests demonstrated that survival of bacteria was shorter in unpressed than in pressed SAS.

For silica:

The literature on the fate of silica in the environment concerns dissolved silica in the aquatic environment, irrespective of its origin (man-made or natural), or structure (crystalline or amorphous). Indeed, once released and dissolved into the environment no distinction can be made between the initial forms of silica. At normal environmental pH, dissolved silica exists exclusively as monosilicic acid [Si(OH)4]. At pH 9.4 the solubility of amorphous silica is about 120 mg SiO2/l. Quartz has a solubility of only 6 mg/l, but its rate of dissolution is so slow at ordinary temperature and pressure that the solubility of amorphous silica represents the upper limit of dissolved silica concentration in natural waters. Moreover, silicic acid is the bioavailable form for aquatic organisms and it plays an important role in the biogeochemical cycle of Si, particularly in the oceans.

In the oceans, the transfer of dissolved silica from the marine hydrosphere to the biosphere initiates the global biological silicon cycle. Marine organisms such as diatoms, silicoflagellates and radiolarians build up their skeletons by taking up silicic acid from seawater. After these organisms die, the biogenic silica accumulated in them partly dissolves. The portion of the biogenic silica that does not dissolve settles and ultimately reaches the sediment. The transformation of opal (amorphous biogenic silica) deposits in sediments through diagenetic processes allows silica to re-enter the geological cycle. Silica is labile between the water and sediment interface.

Ecotoxicity:

Fish LC50 (96 h): *Brachydanio rerio* >10000 mg/l; zebra fish >10000 mg/l

Daphnia magna EC50 (24 h): >1000 mg/l; LC50 924 h): >10000 mg/l

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

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.</p> <p>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 or consult manufacturer for recycling options. ▶ Consult State Land Waste Authority for disposal. ▶ Bury or incinerate residue at an approved site. ▶ Recycle containers if possible, or dispose of in an authorised landfill.

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

SECTION 14 TRANSPORT INFORMATION**Labels Required**

Marine Pollutant	NO
HAZCHEM	Not Applicable

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

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

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

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

Not Applicable

SECTION 15 REGULATORY INFORMATION**Safety, health and environmental regulations / legislation specific for the substance or mixture**

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

HSR Number	Group Standard
HSR002624	N.O.S. (Subsidiary Hazard) Group Standard 2006
HSR002535	Compressed Gas Mixtures (Subsidiary Hazard) Group Standard 2006
HSR002596	Laboratory Chemicals and Reagent Kits Group Standard 2006
HSR002530	Cleaning Products (Subsidiary Hazard) Group Standard 2006
HSR002585	Fuel Additives (Subsidiary Hazard) Group Standard 2006
HSR002519	Aerosols (Subsidiary Hazard) Group Standard 2006
HSR002521	Animal Nutritional and Animal Care Products Group Standard 2006

HSR002606	Lubricants, Lubricant Additives, Coolants and Anti-freeze Agents (Subsidiary Hazard) Group Standard 2006
HSR002644	Polymers (Subsidiary Hazard) Group Standard 2006
HSR002647	Reagent Kits Group Standard 2006
HSR002612	Metal Industry Products (Subsidiary Hazard) Group Standard 2006
HSR002670	Surface Coatings and Colourants (Subsidiary Hazard) Group Standard 2006
HSR002503	Additives, Process Chemicals and Raw Materials (Subsidiary Hazard) Group Standard 2006
HSR002638	Photographic Chemicals (Subsidiary Hazard) Group Standard 2006
HSR002565	Embalming Products (Subsidiary Hazard) Group Standard 2006
HSR002578	Food Additives and Fragrance Materials (Subsidiary Hazard) Group Standard 2006
HSR002558	Dental Products (Subsidiary Hazard) Group Standard 2006
HSR002684	Water Treatment Chemicals (Subsidiary Hazard) Group Standard 2006
HSR002573	Fire Fighting Chemicals Group Standard 2006
HSR100425	Pharmaceutical Active Ingredients Group Standard 2010
HSR002600	Leather and Textile Products (Subsidiary Hazard) Group Standard 2006
HSR002598	Leather and Textile products (Corrosive) Group Standard 2006
HSR002571	Fertilisers (Subsidiary Hazard) Group Standard 2006
HSR002648	Refining Catalysts Group Standard 2006
HSR002653	Solvents (Subsidiary Hazard) Group Standard 2006
HSR002544	Construction Products (Subsidiary Hazard) Group Standard 2006
HSR002549	Corrosion Inhibitors (Subsidiary Hazard) Group Standard 2006
HSR002552	Cosmetic Products Group Standard 2006
HSR100757	Veterinary Medicine (Limited Pack Size, Finished Dose) Standard 2012
HSR100758	Veterinary Medicines (Non-dispersive Closed System Application) Group Standard 2012
HSR100759	Veterinary Medicines (Non-dispersive Open System Application) Group Standard 2012
HSR100628	Straight-chained Lepidopteran Sex Pheromone Group Standard 2012

ACRYLIC RESIN(VARIOUS) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Not Applicable

1,2-BENZISOTHAZOLINE-3-ONE(2634-33-5) IS FOUND ON THE FOLLOWING REGULATORY LISTS

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

New Zealand Inventory of Chemicals (NZIoC)

ECHA SUMMARY

Ingredient	CAS number	Index No	ECHA Dossier
acrylic resin	Various	Not Available	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
Not Available	Not Available	Not Available	Not Available

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

Ingredient	CAS number	Index No	ECHA Dossier
1,2-benzisothiazoline-3-one	2634-33-5	613-088-00-6	Not Available

Harmonisation (C&L Inventory)	Hazard Class and Category Code(s)	Pictograms Signal Word Code(s)	Hazard Statement Code(s)
1	Acute Tox. 4, Skin Irrit. 2, Skin Sens. 1, Eye Dam. 1, Aquatic Acute 1	GHS07, GHS09, GHS05, Dgr	H302, H315, H317, H318
2	Acute Tox. 4, Skin Irrit. 2, Skin Sens. 1, Eye Dam. 1, Aquatic Acute 1, Aquatic Chronic 1, Acute Tox. 2, Acute Tox. 3, Eye Irrit. 2, Not Classified	GHS09, GHS05, Dgr, GHS06, GHS08	H315, H317, H318, H330, H301

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

Location Test Certificate

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

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

Approved Handler

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

Class of substance	Quantities
Not Applicable	Not Applicable

Continued...

Refer Group Standards for further information

Tracking Requirements

Not Applicable

National Inventory	Status
Australia - AICS	Y
Canada - DSL	Y
Canada - NDSL	N (1,2-benzisothiazoline-3-one)
China - IECSC	Y
Europe - EINEC / ELINCS / NLP	Y
Japan - ENCS	Y
Korea - KECI	Y
New Zealand - NZIoC	Y
Philippines - PICCS	Y
USA - TSCA	Y
Legend:	Y = All ingredients are on the inventory N = Not determined or one or more ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Other information

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

A list of reference resources used to assist the committee may be found at:

www.chemwatch.net

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

Definitions and abbreviations

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

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