

## Pinnacle®

### Arxada NZ Limited

Chemwatch: 5376-59  
Version No: 3.1  
Safety Data Sheet according to the Health and Safety at Work (Hazardous Substances) Regulations 2017

Chemwatch Hazard Alert Code: 2

Issue Date: 08/09/2021  
Print Date: 24/11/2021  
L.GHS.NZL.EN

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

### Product Identifier

Product name	Pinnacle®
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains fluazinam)
Chemical formula	Not Applicable
Other means of identification	Not Available

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

Relevant identified uses	Fungicide. Use according to manufacturer's directions.
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### Details of the supplier of the safety data sheet

Registered company name	Arxada NZ Limited
Address	13-15 Hudson Road Bell Block New Plymouth 4312 New Zealand
Telephone	+64 6 755 9234
Fax	+64 6 755 1174
Website	<a href="http://www.arxada.co.nz">www.arxada.co.nz</a>
Email	office-newplymouth@arxada.com

### Emergency telephone number

Association / Organisation	Arxada NZ Limited
Emergency telephone numbers	0800 243 622
Other emergency telephone numbers	+64 4 917 9888 (International)

## SECTION 2 Hazards identification

### Classification of the substance or mixture

Classification [1]	Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2, Acute Toxicity (Inhalation) Category 4, Reproductive Toxicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 2, Hazardous to the Aquatic Environment Long-Term Hazard Category 1, Hazardous to the Aquatic Environment Acute Hazard 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

### Label elements

Hazard pictogram(s)	
Signal word	Warning

### Hazard statement(s)

H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H332	Harmful if inhaled.
H361	Suspected of damaging fertility or the unborn child.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

### Precautionary statement(s) Prevention

P201	Obtain special instructions before use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P273	Avoid release to the environment.
P264	Wash all exposed external body areas thoroughly after handling.
P272	Contaminated work clothing should not be allowed out of the workplace.

### Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P302+P352	IF ON SKIN: Wash with plenty of water.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER/doctor/physician/first aider/if you feel unwell.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P337+P313	If eye irritation persists: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.

### Precautionary statement(s) Storage

P405	Store locked up.
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### Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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## SECTION 3 Composition / information on ingredients

### Substances

See section below for composition of Mixtures

### Mixtures

CAS No	%[weight]	Name
79622-59-6	40-50	<u>fluazinam</u>
107-21-1	1-10	<u>ethylene glycol</u>
Not Available	balance	Ingredients determined not to be hazardous
<b>Legend:</b>	1. Classified by Chemwatch; 2. Classification drawn from CCID EPA NZ; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI; 4. Classification drawn from C&L; * EU IOELVs available	

## SECTION 4 First aid measures

### Description of first aid measures

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

### Indication of any immediate medical attention and special treatment needed

Any material aspirated during vomiting may produce lung injury. Therefore emesis should not be induced mechanically or pharmacologically. Mechanical means should be used if it is

considered necessary to evacuate the stomach contents; these include gastric lavage after endotracheal intubation. If spontaneous vomiting has occurred after ingestion, the patient should be monitored for difficult breathing, as adverse effects of aspiration into the lungs may be delayed up to 48 hours.

Symptoms of vasodilation and reflex tachycardia may present following organic nitrate overdose; most organic nitrates are extensively metabolised by hydrolysis to inorganic nitrites. Organic nitrates and nitrites are readily absorbed through the skin, lungs, mucosa and gastro-intestinal tract.

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

- ▶ Gastric lavage with copious amounts of water.
- ▶ It may be beneficial to instill 60 ml of mineral oil into the stomach.
- ▶ Oxygen and artificial respiration as needed.
- ▶ Electrolyte balance: it may be useful to start 500 ml. M/6 sodium bicarbonate intravenously but maintain a cautious and conservative attitude toward electrolyte replacement unless shock or severe acidosis threatens.
- ▶ To protect the liver, maintain carbohydrate intake by intravenous infusions of glucose.
- ▶ Haemodialysis if coma is deep and persistent. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products, Ed 5]

#### BASIC TREATMENT

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

#### ADVANCED TREATMENT

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

#### EMERGENCY DEPARTMENT

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

For C8 alcohols and above.

Symptomatic and supportive therapy is advised in managing patients.

The toxicity of nitrates and nitrites result from their vasodilating properties and their propensity to form methaemoglobin.

- ▶ Most produce a peak effect within 30 minutes.
- ▶ Clinical signs of cyanosis appear before other symptoms because of the dark pigmentation of methaemoglobin.
- ▶ Initial attention should be directed towards improving oxygen delivery, with assisted ventilation, if necessary. Hyperbaric oxygen has not demonstrated conclusive benefits.
- ▶ Institute cardiac monitoring, especially in patients with coronary artery or pulmonary disease.
- ▶ Hypotension should respond to Trendelenburg's position and intravenous fluids; otherwise dopamine may be needed.
- ▶ Naloxone, glucose and thiamine should be given if a multiple ingestion is suspected.
- ▶ Decontaminate using Ipecac Syrup for alert patients or lavage for obtunded patients who present within 2-4 hours of ingestion.
- ▶ Symptomatic patients with methaemoglobin levels over 30% should receive methylene blue. (Cyanosis alone, is not an indication for treatment). The usual dose is 1-2 mg/kg of a 1% solution (10 mg/ml) IV over 5 minutes; repeat, using the same dose if symptoms of hypoxia fail to subside within 1 hour.

[Ellenhorn and Barceloux: Medical Toxicology]

#### BIOLOGICAL EXPOSURE INDEX - BEI

These represent the determinants observed in specimens collected from a healthy worker who has been exposed at the Exposure Standard (ES or TLV):

Determinant	Index	Sampling Time	Comments
1. Methaemoglobin in blood	1.5% of haemoglobin	During or end of shift	B,NS,SQ

B: Background levels occur in specimens collected from subjects **NOT** exposed

NS: Non-specific determinant; also observed after exposure to other materials

SQ: Semi-quantitative determinant - Interpretation may be ambiguous; should be used as a screening test or confirmatory test.

## SECTION 5 Firefighting measures

### Extinguishing media

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

**Special hazards arising from the substrate or mixture**

<b>Fire Incompatibility</b>	<ul style="list-style-type: none"> <li>▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result</li> </ul>
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**Advice for firefighters**

<b>Fire Fighting</b>	<ul style="list-style-type: none"> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves in the event of a fire.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water courses.</li> <li>▶ Use fire fighting procedures suitable for surrounding area.</li> <li>▶ <b>DO NOT</b> approach containers suspected to be hot.</li> <li>▶ Cool fire exposed containers with water spray from a protected location.</li> <li>▶ If safe to do so, remove containers from path of fire.</li> <li>▶ Equipment should be thoroughly decontaminated after use.</li> </ul>
<b>Fire/Explosion Hazard</b>	<p>carbon dioxide (CO<sub>2</sub>) hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NO<sub>x</sub>) other pyrolysis products typical of burning organic material.</p> <ul style="list-style-type: none"> <li>▶ The material is not readily combustible under normal conditions.</li> <li>▶ However, it will break down under fire conditions and the organic component may burn.</li> <li>▶ Not considered to be a significant fire risk.</li> <li>▶ Heat may cause expansion or decomposition with violent rupture of containers.</li> <li>▶ Decomposes on heating and may produce toxic fumes of carbon monoxide (CO).</li> <li>▶ May emit acrid smoke.</li> </ul> <p>Combustion products include:</p>

**SECTION 6 Accidental release measures****Personal precautions, protective equipment and emergency procedures**

See section 8

**Environmental precautions**

See section 12

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

<b>Minor Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clean up all spills immediately.</li> <li>▶ Avoid breathing vapours and contact with skin and eyes.</li> <li>▶ Control personal contact with the substance, by using protective equipment.</li> <li>▶ Contain and absorb spill with sand, earth, inert material or vermiculite.</li> <li>▶ Wipe up.</li> <li>▶ Place in a suitable, labelled container for waste disposal.</li> </ul>
<b>Major Spills</b>	<p>Environmental hazard - contain spillage.</p> <ul style="list-style-type: none"> <li>▶ Clear area of personnel and move upwind.</li> <li>▶ Alert Fire Brigade and tell them location and nature of hazard.</li> <li>▶ Wear breathing apparatus plus protective gloves.</li> <li>▶ Prevent, by any means available, spillage from entering drains or water course.</li> <li>▶ Stop leak if safe to do so.</li> <li>▶ Contain spill with sand, earth or vermiculite.</li> <li>▶ Collect recoverable product into labelled containers for recycling.</li> <li>▶ Neutralise/decontaminate residue (see Section 13 for specific agent).</li> <li>▶ Collect solid residues and seal in labelled drums for disposal.</li> <li>▶ Wash area and prevent runoff into drains.</li> <li>▶ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using.</li> <li>▶ If contamination of drains or waterways occurs, advise emergency services.</li> </ul>

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

**SECTION 7 Handling and storage****Precautions for safe handling**

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

- ▶ 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

<b>Suitable container</b>	HDPE Jerry can. ▶ Polyethylene or polypropylene container. ▶ Packing as recommended by manufacturer. ▶ Check all containers are clearly labelled and free from leaks.
<b>Storage incompatibility</b>	▶ Avoid reaction with oxidising agents, bases and strong reducing agents. ▶ Avoid strong acids, acid chlorides, acid anhydrides and chloroformates.



- X — Must not be stored together  
 O — May be stored together with specific precautions  
 + — May be stored together

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

## 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	Ethylene glycol (vapour and mist)	Not Available	Not Available	50 ppm / 127 mg/m <sup>3</sup>	Not Available

#### Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
ethylene glycol	30 ppm	150 ppm	900 ppm

Ingredient	Original IDLH	Revised IDLH
fluazinam	Not Available	Not Available
ethylene glycol	Not Available	Not Available

#### Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
fluazinam	E	≤ 0.01 mg/m <sup>3</sup>

**Notes:** Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

#### MATERIAL DATA

#### Exposure controls

<b>Appropriate engineering controls</b>	<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. The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <p>General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA 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.</p>											
	<table border="1"> <thead> <tr> <th>Type of Contaminant:</th> <th>Air Speed:</th> </tr> </thead> <tbody> <tr> <td>solvent, vapours, degreasing etc., evaporating from tank (in still air)</td> <td>0.25-0.5 m/s (50-100 f/min)</td> </tr> <tr> <td>aerosols, fumes from pouring operations, intermittent container filling, low speed conveyer transfers, welding, spray drift, plating acid fumes, pickling (released at low velocity into zone of active generation)</td> <td>0.5-1 m/s (100-200 f/min.)</td> </tr> <tr> <td>direct spray, spray painting in shallow booths, drum filling, conveyer loading, crusher dusts, gas discharge (active generation into zone of rapid air motion)</td> <td>1-2.5 m/s (200-500 f/min)</td> </tr> <tr> <td>grinding, abrasive blasting, tumbling, high speed wheel generated dusts (released at high initial velocity into zone of very high rapid air motion).</td> <td>2.5-10 m/s (500-2000 f/min.)</td> </tr> </tbody> </table>	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.)	
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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.	
<b>Personal protection</b>		
<b>Eye and face protection</b>	<ul style="list-style-type: none"> <li>▶ Safety glasses with side shields.</li> <li>▶ Chemical goggles.</li> <li>▶ 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]</li> </ul>	
<b>Skin protection</b>	See Hand protection below	
<b>Hands/feet protection</b>	<ul style="list-style-type: none"> <li>▶ Wear chemical protective gloves, e.g. PVC.</li> <li>▶ Wear safety footwear or safety gumboots, e.g. Rubber</li> </ul> <p><b>NOTE:</b></p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.</li> </ul> <p>The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.</p> <p>The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.</p> <p>Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p> <p>Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include:</p> <ul style="list-style-type: none"> <li>· frequency and duration of contact,</li> <li>· chemical resistance of glove material,</li> <li>· glove thickness and</li> <li>· dexterity</li> </ul> <p>Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent).</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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.</li> <li>· Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use.</li> <li>· Contaminated gloves should be replaced.</li> </ul> <p>As defined in ASTM F-739-96 in any application, gloves are rated as:</p> <ul style="list-style-type: none"> <li>· Excellent when breakthrough time &gt; 480 min</li> <li>· Good when breakthrough time &gt; 20 min</li> <li>· Fair when breakthrough time &lt; 20 min</li> <li>· Poor when glove material degrades</li> </ul> <p>For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.</p> <p>It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.</p> <p>Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers' technical data should always be taken into account to ensure selection of the most appropriate glove for the task.</p> <p>Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:</p> <ul style="list-style-type: none"> <li>· 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.</li> <li>· 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</li> </ul> <p>Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.</p>	
<b>Body protection</b>	See Other protection below	
<b>Other protection</b>	<ul style="list-style-type: none"> <li>▶ Overalls.</li> <li>▶ P.V.C apron.</li> <li>▶ Barrier cream.</li> <li>▶ Skin cleansing cream.</li> <li>▶ Eye wash unit.</li> </ul>	

**Recommended material(s)****GLOVE SELECTION INDEX**

Glove selection is based on a modified presentation of the:

**Respiratory protection**

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

Continued...

**"Forsberg Clothing Performance Index".**

The effect(s) of the following substance(s) are taken into account in the **computer-generated** selection:  
Pinnacle®

Material	CPI
NATURAL RUBBER	A
NATURAL+NEOPRENE	A
NEOPRENE	A
NEOPRENE/NATURAL	A
NITRILE	A
NITRILE+PVC	A
PE/EVAL/PE	A
PVC	A
TEFLON	A
PVA	B

\* 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.

ANSI Z88 or national equivalent)

Selection of the Class and Type of respirator will depend upon the level of breathing zone contaminant and the chemical nature of the contaminant. Protection Factors (defined as the ratio of contaminant outside and inside the mask) may also be important.

Required minimum protection factor	Maximum gas/vapour concentration present in air p.p.m. (by volume)	Half-face Respirator	Full-Face Respirator
up to 10	1000	A-AUS / Class1 P2	-
up to 50	1000	-	A-AUS / Class 1 P2
up to 50	5000	Airline *	-
up to 100	5000	-	A-2 P2
up to 100	10000	-	A-3 P2
100+			Airline**

\* - Continuous Flow \*\* - Continuous-flow or positive pressure demand

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

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

## SECTION 9 Physical and chemical properties

### Information on basic physical and chemical properties

<b>Appearance</b>	Yellow-brown liquid suspension; dispersible in water.		
<b>Physical state</b>	Liquid	<b>Relative density (Water = 1)</b>	Not Applicable
<b>Odour</b>	Not Available	<b>Partition coefficient n-octanol / water</b>	Not Available
<b>Odour threshold</b>	Not Available	<b>Auto-ignition temperature (°C)</b>	Not Applicable
<b>pH (as supplied)</b>	7.0-9.0	<b>Decomposition temperature</b>	Not Applicable
<b>Melting point / freezing point (°C)</b>	Not Available	<b>Viscosity (cSt)</b>	Not Available
<b>Initial boiling point and boiling range (°C)</b>	Not Available	<b>Molecular weight (g/mol)</b>	Not Applicable
<b>Flash point (°C)</b>	Not Applicable	<b>Taste</b>	Not Available
<b>Evaporation rate</b>	Not Available	<b>Explosive properties</b>	Not Available
<b>Flammability</b>	Not Applicable	<b>Oxidising properties</b>	Not Available
<b>Upper Explosive Limit (%)</b>	Not Applicable	<b>Surface Tension (dyn/cm or mN/m)</b>	Not Available
<b>Lower Explosive Limit (%)</b>	Not Applicable	<b>Volatile Component (%vol)</b>	Not Available
<b>Vapour pressure (kPa)</b>	Not Available	<b>Gas group</b>	Not Available
<b>Solubility in water</b>	Miscible	<b>pH as a solution (%)</b>	Not Available
<b>Vapour density (Air = 1)</b>	Not Available	<b>VOC g/L</b>	Not Available

## SECTION 10 Stability and reactivity

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

## SECTION 11 Toxicological information

### Information on toxicological effects

<p><b>Inhaled</b></p>	<p>Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation.</p> <p>The material is not thought to produce respiratory irritation (as classified by EC Directives using animal models). Nevertheless inhalation of vapours, fumes or aerosols, especially for prolonged periods, may produce respiratory discomfort and occasionally, distress.</p> <p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Exposure to aliphatic alcohols with more than 3 carbons may produce central nervous system effects such as headache, dizziness, drowsiness, muscle weakness, delirium, CNS depression, coma, seizure, and neurobehavioural changes. Symptoms are more acute with higher alcohols.</p> <p>Respiratory tract involvement may produce irritation of the mucosa, respiratory insufficiency, respiratory depression secondary to CNS depression, pulmonary oedema, chemical pneumonitis and bronchitis. Cardiovascular involvement may result in arrhythmias and hypotension. Gastrointestinal effects may include nausea and vomiting. Kidney and liver damage may result following massive exposures. The alcohols are potential irritants being, generally, stronger irritants than similar organic structures that lack functional groups (e.g. alkanes) but are much less irritating than the corresponding amines, aldehydes or ketones. Alcohols and glycols (diols) rarely represent serious hazards in the workplace, because their vapour concentrations are usually less than the levels which produce significant irritation which, in turn, produce significant central nervous system effects as well.</p> <p>Inhalation of dusts, generated by the material, during the course of normal handling, may produce toxic effects.</p>
<p><b>Ingestion</b></p>	<p>The principal concern with exposure to inorganic nitrate is its biological reduction to reactive and toxic nitrite. Nitrate itself is relatively harmless. Where bacteria are present and the environment is anaerobic, nitrate can be reduced to nitrite. The main site for this reaction is mouth and stomach, but nitrite formation in the lower intestine and in the bladder (urinary infection) may also be of some toxicological importance.</p> <p>Adults have tolerated large doses of nitrate as sodium and ammonium salt (&gt; 100 mg NO<sub>3</sub>-/kg) in some cases repeated for several days for medical or experimental purposes with only minor effects in some subjects (light methaemoglobinaemia, diarrhoea, vomiting). Death and severe effects of nitrate ingestion are generally associated with doses above 10 g NO<sub>3</sub>-. Doses between 2 and 9 g NO<sub>3</sub>- have been reported to cause methaemoglobinaemia. These values correspond to 33 to 150 mg NO<sub>3</sub>-/kg</p> <p>The half-life in the body for an oral dose of nitrate to be approximately 5 hours. As blood absorption depends on food matrix and route of exposure, and as larger doses may increase the urinary excretion rate, the biological half-life for both nitrate and nitrite should be expected to be 3 to 8 hours. Nitrate does not accumulate in the body.</p> <p>The major acute toxic effect of nitrate and nitrite poisoning is methaemoglobinaemia.</p> <p>The lethal oral dose of nitrite for adults has been variously reported to be between 0.7 and 6 g NO<sub>2</sub>- (approximately 10 to 100 mg NO<sub>2</sub>-/kg). Lower doses may apply for children (especially neonates), the elderly and people with certain enzyme deficiencies. The first symptoms of oral nitrite poisoning develop within 15 to 45 minutes</p> <p>In humans, inorganic nitrites produce smooth muscle relaxation, methaemoglobinaemia and cyanosis. The primary effect of nitrite intoxication in animals is methaemoglobinaemia whilst secondary effects include vasodilation, relaxation of smooth muscle and lowering of blood pressure. Other nitrite-induced toxic effects include abdominal pain, diarrhoea, atrophied intestinal villi and apoptotic cell death in the intestinal crypts.</p> <p>Nitrite may also cause sudden fall in blood pressure due to its vasodilating properties. Nitrite has vasodilating properties, probably through transformation into nitric oxide (NO) or a NO-containing molecule acting as a signal factor for smooth muscle relaxation.</p> <p>Fatal poisonings in infants, resulting from ingestion of nitrates in water or spinach, have been reported.</p> <p>When sodium nitrite was administered in drinking water for 6 weeks (0.06-1%), mice showed a slight degeneration and spotty necrosis of hepatocytes and haemosiderin deposition in the liver, spleen and lymph nodes, indicating haemolysis. At 2%, mice died within 3 weeks. In rats, subject to the same treatment regime, abnormal blood and spleen colours, due to MHG, were seen in 0.5% and 1.0% treatment groups. Hepatic microsomal lipoperoxidation (as measured by malondialdehyde formation) was increased in male rats given 0.2% sodium nitrite in drinking water. Liver lysosomal enzymes (acid phosphatase and cathepsin) and superoxide dismutase activities were also increased. This data suggests that the nitrite stimulates generation of superoxide radicals in the liver causing damage to cellular and subcellular membranes. Decreased plasma vitamin E and greater reduced glutathione-per erythrocyte were also reported in male rats receiving sodium nitrite in drinking water.</p> <p>The substance and/or its metabolites may bind to haemoglobin inhibiting normal uptake of oxygen. This condition, known as "methaemoglobinemia", is a form of oxygen starvation (anoxia).</p> <p>Symptoms include cyanosis (a bluish discolouration skin and mucous membranes) and breathing difficulties. Symptoms may not be evident until several hours after exposure.</p> <p>At about 15% concentration of blood methaemoglobin there is observable cyanosis of the lips, nose and earlobes. Symptoms may be absent although euphoria, flushed face and headache are commonly experienced. At 25-40%, cyanosis is marked but little disability occurs other than that produced on physical exertion. At 40-60%, symptoms include weakness, dizziness, lightheadedness, increasingly severe headache, ataxia, rapid shallow respiration, drowsiness, nausea, vomiting, confusion, lethargy and stupor. Above 60% symptoms include dyspnea, respiratory depression, tachycardia or bradycardia, and convulsions. Levels exceeding 70% may be fatal.</p> <p>Effects on the nervous system characterise over-exposure to higher aliphatic alcohols. These include headache, muscle weakness, giddiness, ataxia, (loss of muscle coordination), confusion, delirium and coma. Gastrointestinal effects may include nausea, vomiting and diarrhoea. In the absence of effective treatment, respiratory arrest is the most common cause of death in animals acutely poisoned by the higher alcohols.</p> <p>Aspiration of liquid alcohols produces an especially toxic response as they are able to penetrate deeply in the lung where they are absorbed and may produce pulmonary injury. Those possessing lower viscosity elicit a greater response. The result is a high blood level and prompt death at doses otherwise tolerated by ingestion without aspiration. In general the secondary alcohols are less toxic than the corresponding primary isomers. As a general observation, alcohols are more powerful central nervous system depressants than their aliphatic analogues. In sequence of decreasing depressant potential, tertiary alcohols with multiple substituent OH groups are more potent than secondary alcohols, which, in turn, are more potent than primary alcohols. The potential for overall systemic toxicity increases with molecular weight (up to C7), principally because the water solubility is diminished and lipophilicity is increased.</p> <p>Within the homologous series of aliphatic alcohols, narcotic potency may increase even faster than lethality</p> <p>Only scanty toxicity information is available about higher homologues of the aliphatic alcohol series (greater than C7) but animal data establish that lethality does not continue to increase with increasing chain length. Aliphatic alcohols with 8 carbons are less toxic than those immediately preceding them in the series. 10 -Carbon n-decyl alcohol has low toxicity as do the solid fatty alcohols (e.g. lauryl, myristyl, cetyl and stearyl). However the rat aspiration test suggests that decyl and melted dodecyl (lauryl) alcohols are dangerous if they enter the trachea. In the rat even a small quantity (0.2 ml) of these behaves like a hydrocarbon solvent in causing death from pulmonary oedema.</p> <p>Primary alcohols are metabolised to corresponding aldehydes and acids; a significant metabolic acidosis may occur. Secondary alcohols are converted to ketones, which are also central nervous system depressants and which, in the case of the higher homologues persist in the blood for many hours. Tertiary alcohols are metabolised slowly and incompletely so their toxic effects are generally persistent.</p> <p>for ethylene glycol:</p> <p>Ingestion symptoms include respiratory failure, central nervous depression, cardiovascular collapse, pulmonary oedema, acute kidney failure, and even brain damage. Ingestion of 100 ml has caused death. (ChemInfo)</p> <p>Toxicity of ethylene glycol to human (KB) cell cultures has been reported as less than that of ethanol. (NIOSH/ITC)</p> <p>Ethylene glycol produces a three-stage response with the severity of each stage dependent on the amount of ingestion. Hepatic damage is usually minimal. Central nervous system depression characterise the first 12 hours post ingestion.</p> <p>Transient exhalation occurs without the odour of ethanol.</p> <p>Gastrointestinal complaints include nausea and vomiting. Acidosis, coma, convulsions and myoclonic jerks may also be evident. The optic fundus is usually normal although the presence of papilloedema may confuse the presentation with that produced by methanol. Nystagmus and ophthalmoplegias may appear.</p>

	<p>Cardiopulmonary effects are seen 12-24 hours post-ingestion and are characterised by tachycardia, tachypnea, and mild hypertension. Congestive heart failure and circulatory collapse may occur in severe intoxications.</p> <p>Renal effects are seen 24-72 hours post-ingestion and are characterised by oliguria, flank pain, acute tubular necrosis, renal failure, and rarely, bone marrow arrest. Renal damage may be permanent.</p> <p>Toxic effects of ethylene glycol are similar to those produced by ethanol but ethylene glycol produces toxic metabolites. Metabolic acidosis and anion gap result primarily from glycolic acid formation and some lactic acid formation. The citric acid cycle is inhibited as a result of reduced NAD/NADH ratios and to a limited extent, the formation of oxalic acid, and to metabolic acidosis. Oxalate formation produces myocardial depression and acute tubular necrosis. Glycoaldehyde, glycolic acid and glyoxylic acid may contribute to CNS depression and may also produce renal toxicity by producing renal oedema. Hypocalcaemia may result from chelation by oxalate. Oxalic acid, glycoxalic acid, glycoaldehyde and formic acid appear to form to only a limited degree during intoxication. Oral administration to pregnant mice and rats produced birth defects amongst the off-spring.</p> <p>Accidental ingestion of the material may be damaging to the health of the individual.</p>
<p><b>Skin Contact</b></p>	<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. Most liquid alcohols appear to act as primary skin irritants in humans. Significant percutaneous absorption occurs in rabbits but not apparently in man.</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.</p> <p>Skin contact with the material may damage the health of the individual; systemic effects may result following absorption.</p>
<p><b>Eye</b></p>	<p>Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.</p> <p>Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p>
<p><b>Chronic</b></p>	<p>Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.</p> <p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>Substances that can cause occupational asthma (also known as asthmagens and respiratory sensitisers) can induce a state of specific airway hyper-responsiveness via an immunological, irritant or other mechanism. Once the airways have become hyper-responsive, further exposure to the substance, sometimes even to tiny quantities, may cause respiratory symptoms. These symptoms can range in severity from a runny nose to asthma. Not all workers who are exposed to a sensitiser will become hyper-responsive and it is impossible to identify in advance who are likely to become hyper-responsive.</p> <p>Substances that can cause occupational asthma should be distinguished from substances which may trigger the symptoms of asthma in people with pre-existing air-way hyper-responsiveness. The latter substances are not classified as asthmagens or respiratory sensitisers</p> <p>Wherever it is reasonably practicable, exposure to substances that can cause occupational asthma should be prevented. Where this is not possible the primary aim is to apply adequate standards of control to prevent workers from becoming hyper-responsive.</p> <p>Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance.</p> <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.</p> <p>Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or 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>There is some evidence to provide a presumption that human exposure to the material may result in impaired fertility on the basis of: some evidence in animal studies of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects but which is not a secondary non-specific consequence of other toxic effects.</p> <p>Chronic exposure to nitro compounds of aromatic hydrocarbons have been known to cause liver and kidney damage with production of acute yellow atrophy, toxic hepatitis and fatty degeneration of the kidneys.</p> <p>[OHS 24320]</p> <p>The major concern of possible long-term effects of exposure to nitrate and nitrite is associated with formation of nitroso compounds, many of which are carcinogenic. This formation may take place wherever nitrite and nitrosable compounds are present, but it is favoured by acidic conditions or the presence of some bacteria. The gastrointestinal tract and especially the stomach is regarded as the main formation site, but nitrosation reactions can also take place in an infected urinary bladder.</p> <p>Nitrite is mutagenic in a number of in vitro assays against microorganisms or cultured mammalian cells. Nitrates show no mutagenic activity in microbial tests under aerobic conditions. Activity has been reported under anaerobic conditions, probably due to reduction of nitrate into nitrite</p> <p>The mutagenic effects of nitrites were observed in an in vivo and in vitro experiment using Syrian hamsters. In vivo assays have been equivocal, both positive and negative results having been reported</p> <p>Exposure to sodium nitrite in drinking water resulted in an increased incidence of epithelial hyperplasia in the forestomach of male and female rats and in the glandular stomach of male mice.</p> <p>There was equivocal evidence of carcinogenic activity of sodium nitrite in female B6C3F1 mice based on the positive trend in the incidences of squamous cell papillomas or carcinomas (combined) of the forestomach. There was no evidence of carcinogenic activity in male and female F344/N rats or B6C3F1 male mice exposed to 750, 1500 or 3000 ppm.</p> <p>NTP Technical Report Series No. 495, May 2001</p> <p>Under certain conditions, nitrites can react with secondary amines, either alone or in biological systems, to form carcinogenic nitrosamines.</p> <p>Sodium nitrite (60 mg/kg) administered in drinking water to pregnant guinea pigs produced maternal anaemia and increased the incidences of abortion and foetal mortality. Administration of 2000-3000 mg/l sodium nitrite in drinking water, to pregnant rats, produced 30-53% foetal mortality.</p> <p>In rat dams given 0.025-0.5% in feed, sodium nitrite caused an increase in foetal and pup mortality and decreases in pre-weaning body weights.</p> <p>Human volunteers exposed to ethylene glycol, 20 to 22 hours/day at mean daily concentrations ranging from 1.4 to 27 ppm for about 4 weeks complained of throat irritation, mild headache and low backache. Complaints became marked when the concentration in the exposure chamber was raised above 56 mg/m<sup>3</sup> for part of the day. The most common complaint was irritation of the upper respiratory tract. Concentrations above 80 ppm were intolerable with a burning sensation along the trachea and a burning cough. Excessively exposed workers have reported drowsiness.</p>

Pinnacle®	TOXICITY	IRRITATION
fluazinam	Not Available  dermal (rat) LD50: >200 mg/kg <sup>[2]</sup> Inhalation(Rat) LC50; 0.47 mg/L4h <sup>[2]</sup> Oral(Rat) LD50; >5000 mg/kg <sup>[2]</sup>	Not Available  Eye (rabbit): SEVERE * Skin (rabbit): SEVERE *
ethylene glycol	dermal (mouse) LD50: >3500 mg/kg <sup>[1]</sup> Oral(Rat) LD50; >2000 mg/kg <sup>[2]</sup>	Eye (rabbit): 100 mg/1h - mild Eye (rabbit): 12 mg/m3/3D Eye (rabbit): 1440mg/6h-moderate Eye (rabbit): 500 mg/24h - mild Eye: no adverse effect observed (not irritating) <sup>[1]</sup> Skin (rabbit): 555 mg(open)-mild Skin: no adverse effect observed (not irritating) <sup>[1]</sup>
<b>Legend:</b>	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	

FLUAZINAM	<p>ADI: 0.004 mg/kg/day NOEL: 0.4 mg/kg/day Patch testing on 10 volunteers showed that the product caused a type IV allergic reaction. Symptoms observed include localised swelling itchiness and red spots in affected skin regions. Once exposure had stopped full recovery occurs within a short period of time. * NuFarm NZ SDS Shirilan Sensitising in guinea pig assay</p> <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>Substances that interfere with oxidative phosphorylation could produce severe side effects. Even though there are marketed drugs that inhibit mitochondrial activity, three of these drugs have been withdrawn by the U.S. Food and Drug Administration since 1994 due to mitochondrial side effects. Although cells can tolerate diminished mitochondrial activity as long as a minimal capacity is maintained, a loss of mitochondrial function can make cells increasingly unable to respond to other stress factors that eventually results in necrosis or apoptosis, depending on the rate of decline. Mitochondrial impairment (mitotoxicity) typically affects tissues with higher aerobic activity, such as the kidney or heart, or the liver, which is exposed to higher concentrations of drugs. Besides these tissue-specific effects, mitochondrially-toxic compounds are more likely to display drug-drug interactions.</p> <p>The disruption of cell function that accompanies mitotoxicity can cause both mild and severe problems in people. The most commonly observed symptom is muscle weakness, or myopathy. Others include peripheral neuropathy (numbness in the fingers and toes) and pancreatitis (inflammation of the pancreas), with the most severe being lactic acidosis, in which a build-up of lactic acid in the tissues of the body leads to loss of energy, organ failure, and eventually death</p> <p>These effects are often revealed in large phase III trials, because for rare occurrences, statistically more than 10,000 patients would have to be exposed before the probability of the event occurring becomes significant.</p>
ETHYLENE GLYCOL	<p>[Estimated Lethal Dose (human) 100 ml; RTECS quoted by Orica] Substance is reproductive effector in rats (birth defects). Mutagenic to rat cells. For ethylene glycol:</p> <p>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 CO<sub>2</sub>, which is one of the major elimination products of ethylene glycol. In addition to exhaled CO<sub>2</sub>, 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.</p> <p><b>Respiratory Effects.</b> 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).</p> <p><b>Cardiovascular Effects.</b> 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.</p> <p><b>Gastrointestinal Effects.</b> 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.</p> <p><b>Musculoskeletal Effects.</b> 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.</p> <p><b>Hepatic Effects.</b> Central hydropic or fatty degeneration, parenchymal necrosis, and calcium oxalate crystals in the liver have been observed at</p>

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.

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

Pinnacle®	Endpoint	Test Duration (hr)	Species	Value	Source
	Not Available	Not Available	Not Available	Not Available	Not Available

  

fluazinam	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50(ECx)	96h	Algae or other aquatic plants	0.001-325800mg/L	4
	LC50	96h	Fish	0.06-0.101mg/L	4
	EC50	48h	Crustacea	0.253-0.402mg/L	4
	EC50	96h	Algae or other aquatic plants	0.001-325800mg/L	4

  

ethylene glycol	Endpoint	Test Duration (hr)	Species	Value	Source
	EC50	48h	Crustacea	>100mg/l	2
	LC50	96h	Fish	>10000mg/l	1
	EC50(ECx)	Not Available	Algae or other aquatic plants	6500-7500mg/l	1
	EC50	96h	Algae or other aquatic plants	6500-13000mg/l	1

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

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

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

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

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

The nitrates are of environmental concern because of their high water solubility and consequent leaching, diffusion, and environmental mobility in soil and water. Nitrate can contaminate groundwater to unacceptable levels. Nitrite is formed from nitrate or ammonium ion by micro-organisms in soil, water, sewage and the alimentary tract. The concern with nitrate in the environment is related to its conversion to nitrite.

Methaemoglobinaemia is caused following exposure to high levels of nitrite and produces difficulties in oxygen transport in the blood. Thousands of cases involving poisoning of infants, particularly in rural areas, have been reported as a result of drinking nitrate rich well-water.

Other concerns deriving from exposure to environmental nitrates relate to the production of nitrosamines following the reaction of food nitrites and secondary amines. Other nitroso-

compounds may result following reaction with nitrates and amides, ureas, carbamates and other nitrogenous compounds. Nitrosamines produce liver damage, haemorrhagic lung lesions, convulsions and coma in rats, and teratogenic effects in experimental animals.

The N-nitroso class of compounds include potent carcinogens and mutagens: induction of tumors by single doses of N-nitroso compounds testify to this.

**DO NOT discharge into sewer or waterways.**

#### Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
fluazinam	HIGH	HIGH
ethylene glycol	LOW (Half-life = 24 days)	LOW (Half-life = 3.46 days)

#### Bioaccumulative potential

Ingredient	Bioaccumulation
fluazinam	HIGH (LogKOW = 5.8477)
ethylene glycol	LOW (BCF = 200)

#### Mobility in soil

Ingredient	Mobility
fluazinam	LOW (KOC = 371700)
ethylene glycol	HIGH (KOC = 1)

### SECTION 13 Disposal considerations

#### Waste treatment methods

<b>Product / Packaging disposal</b>	<ul style="list-style-type: none"> <li>▶ Containers may still present a chemical hazard/ danger when empty.</li> <li>▶ Return to supplier for reuse/ recycling if possible.</li> </ul> <p>Otherwise:</p> <ul style="list-style-type: none"> <li>▶ 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.</li> <li>▶ Where possible retain label warnings and SDS and observe all notices pertaining to the product.</li> </ul> <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"> <li>▶ Reduction</li> <li>▶ Reuse</li> <li>▶ Recycling</li> <li>▶ Disposal (if all else fails)</li> </ul> <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"> <li>▶ <b>DO NOT allow wash water from cleaning or process equipment to enter drains.</b></li> <li>▶ It may be necessary to collect all wash water for treatment before disposal.</li> <li>▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first.</li> <li>▶ Where in doubt contact the responsible authority.</li> <li>▶ Recycle wherever possible or consult manufacturer for recycling options.</li> <li>▶ Consult State Land Waste Authority for disposal.</li> <li>▶ Bury or incinerate residue at an approved site.</li> <li>▶ Recycle containers if possible, or dispose of in an authorised landfill.</li> </ul>
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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	
HAZCHEM	*3Z

**Land transport (UN)**

<b>UN number</b>	3082	
<b>UN proper shipping name</b>	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains fluazinam)	
<b>Transport hazard class(es)</b>	Class	9
	Subrisk	Not Applicable
<b>Packing group</b>	III	
<b>Environmental hazard</b>	Environmentally hazardous	
<b>Special precautions for user</b>	Special provisions	274; 331; 335; 375
	Limited quantity	5 L

**Air transport (ICAO-IATA / DGR)**

<b>UN number</b>	3082	
<b>UN proper shipping name</b>	Environmentally hazardous substance, liquid, n.o.s. * (contains fluazinam)	
<b>Transport hazard class(es)</b>	ICAO/IATA Class	9
	ICAO / IATA Subrisk	Not Applicable
	ERG Code	9L
<b>Packing group</b>	III	
<b>Environmental hazard</b>	Environmentally hazardous	
<b>Special precautions for user</b>	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

**Sea transport (IMDG-Code / GGVSee)**

<b>UN number</b>	3082	
<b>UN proper shipping name</b>	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains fluazinam)	
<b>Transport hazard class(es)</b>	IMDG Class	9
	IMDG Subrisk	Not Applicable
<b>Packing group</b>	III	
<b>Environmental hazard</b>	Marine Pollutant	
<b>Special precautions for user</b>	EMS Number	F-A , S-F
	Special provisions	274 335 969
	Limited Quantities	5 L

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

Not Applicable

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

Product name	Group
fluazinam	Not Available
ethylene glycol	Not Available

**Transport in bulk in accordance with the ICG Code**

Product name	Ship Type
fluazinam	Not Available
ethylene glycol	Not Available

**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
HSR100383	Not Available

Continued...

Please refer to Section 8 of the SDS for any applicable tolerable exposure limit or Section 12 for environmental exposure limit.

#### fluazinam is found on the following regulatory lists

New Zealand Approved Hazardous Substances with controls  
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)

#### ethylene glycol is found on the following regulatory lists

Chemical Footprint Project - Chemicals of High Concern List  
New Zealand Approved Hazardous Substances with controls  
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 Workplace Exposure Standards (WES)

#### Hazardous Substance Location

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

Hazard Class	Quantities
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

#### Maximum quantities of certain hazardous substances permitted on passenger service vehicles

Subject to Regulation 13.14 of the Health and Safety at Work (Hazardous Substances) Regulations 2017.

Hazard Class	Gas (aggregate water capacity in mL)	Liquid (L)	Solid (kg)	Maximum quantity per package for each classification
6.5A or 6.5B	120	1	3	

#### Tracking Requirements

Not Applicable

#### National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (fluazinam)
Canada - DSL	No (fluazinam)
Canada - NDSL	No (fluazinam; ethylene glycol)
China - IECSC	No (fluazinam)
Europe - EINEC / ELINCS / NLP	No (fluazinam)
Japan - ENCS	No (fluazinam)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (fluazinam)
USA - TSCA	No (fluazinam)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (fluazinam)
<b>Legend:</b>	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.

#### SECTION 16 Other information

Revision Date	08/09/2021
Initial Date	05/12/2019

#### SDS Version Summary

Version	Date of Update	Sections Updated
2.1	05/12/2019	Chronic Health, Classification
3.1	08/09/2021	Acute Health (eye), Acute Health (inhaled), Acute Health (skin), Acute Health (swallowed), Advice to Doctor, Appearance, Chronic Health, Classification, Engineering Control, Environmental, Fire Fighter (extinguishing media), Fire Fighter (fire/explosion hazard), Fire Fighter (fire incompatibility), First Aid (eye), First Aid (swallowed), Ingredients, Physical Properties, Spills (major), Spills (minor), Storage (storage incompatibility), Storage (storage requirement), Storage (suitable container)

**Other information****Ingredients with multiple cas numbers**

Name	CAS No
ethylene glycol	107-21-1, 1371582-33-0, 2088100-90-5, 37221-95-7, 71767-64-1

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  
 ES: Exposure Standard  
 OSF: Odour Safety Factor  
 NOAEL :No Observed Adverse Effect Level  
 LOAEL: Lowest Observed Adverse Effect Level  
 TLV: Threshold Limit Value  
 LOD: Limit Of Detection  
 OTV: Odour Threshold Value  
 BCF: BioConcentration Factors  
 BEI: Biological Exposure Index  
 AIIC: Australian Inventory of Industrial Chemicals  
 DSL: Domestic Substances List  
 NDSL: Non-Domestic Substances List  
 IECSC: Inventory of Existing Chemical Substance in China  
 EINECS: European INventory of Existing Commercial chemical Substances  
 ELINCS: European List of Notified Chemical Substances  
 NLP: No-Longer Polymers  
 ENCS: Existing and New Chemical Substances Inventory  
 KECI: Korea Existing Chemicals Inventory  
 NZIoC: New Zealand Inventory of Chemicals  
 PICCS: Philippine Inventory of Chemicals and Chemical Substances  
 TSCA: Toxic Substances Control Act  
 TCSI: Taiwan Chemical Substance Inventory  
 INSQ: Inventario Nacional de Sustancias Químicas  
 NCI: National Chemical Inventory  
 FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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