Apparent Myagi 250 Insecticide

AIRR Apparent Pty Ltd.

Chemwatch Hazard Alert Code: **4**

Chemwatch: **9656-39**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **30/10/2026**
Revision Date: **30/10/2026**
Print Date: **23/11/2026**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Apparent Myagi 250 Insecticide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	PYRETHROID PESTICIDE, LIQUID, TOXIC
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	A liquid insecticide for control of certain insect pests in crops. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**AIRR Apparent Pty Ltd.**
Address	15/16 Princes Street, Newport NSW 2106 Australia
Telephone	+61 3 5820 8400
Fax	Not Available
Website	www.apparent.com.au
Email	enquiries@apparentag.com.au

Emergency telephone number

Association / Organisation	**AIRR Apparent Pty Ltd.**
Emergency telephone number(s)	1800 033 111 (24 Hours)
Other emergency telephone number(s)	Not Available

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 3, Acute Toxicity (Dermal) Category 4, Skin Corrosion/Irritation Category 1B, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 1, Acute Toxicity (Inhalation) Category 1, Sensitisation (Respiratory) Category 1, Carcinogenicity Category 2, Specific Target Organ Toxicity - Repeated Exposure Category 1, Hazardous to the Aquatic Environment Acute Hazard Category 3, Hazardous to the Aquatic Environment Long-Term Hazard Category 1
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI***

Label elements

Hazard pictogram(s)	
Signal word	**Danger**

Hazard statement(s)

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H301	Toxic if swallowed.
H312	Harmful in contact with skin.
H314	Causes severe skin burns and eye damage.
H317	May cause an allergic skin reaction.
H330	Fatal if inhaled.
H334	May cause allergy or asthma symptoms or breathing difficulties if inhaled.
H351	Suspected of causing cancer.
H372	Causes damage to organs through prolonged or repeated exposure.
H402	Harmful to aquatic life.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P260	Do not breathe mist/vapours/spray.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P271	Use only a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P284	[In case of inadequate ventilation] wear respiratory protection.
P273	Avoid release to the environment.
P202	Do not handle until all safety precautions have been read and understood.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER/doctor/physician/first aider.
P301+P330+P331	IF SWALLOWED: Rinse mouth. Do NOT induce vomiting.
P303+P361+P353	IF ON SKIN (or hair): Take off immediately all contaminated clothing. Rinse skin with water [or shower].
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P308+P313	IF exposed or concerned: Get medical advice/ attention.
P342+P311	If experiencing respiratory symptoms: Call a POISON CENTER/doctor/physician/first aider.
P302+P352	IF ON SKIN: Wash with plenty of water.
P363	Wash contaminated clothing before reuse.
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.
P362+P364	Take off contaminated clothing and wash it before reuse.
P391	Collect spillage.

Precautionary statement(s) Storage

P403+P233	Store in a well-ventilated place. Keep container tightly closed.
P405	Store locked up.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.

No further product hazard information.

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
91465-08-6	25	cyhalothricacid
9016-87-9	1-10	MDI oligomer
2634-33-5	<1	1,2-benzisothiazoline-3-one
Not Available	balance	Ingredients determined not to be hazardous
Legend:	***1. Classified by Chemwatch; 2. Classification drawn from HCIS; 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

Eye Contact	If this product comes in contact with the eyes:

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	‣ Immediately hold eyelids apart and flush the eye continuously with 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. ‣ Continue flushing until advised to stop by the Poisons Information Centre or a doctor, or for at least 15 minutes. ‣ Transport to hospital or doctor without delay. ‣ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin or hair contact occurs: ‣ Immediately flush body and clothes with large amounts of water, using safety shower if available. ‣ Quickly remove all contaminated clothing, including footwear. ‣ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ‣ Transport to hospital, or doctor.
Inhalation	‣ 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, without delay. Following uptake by inhalation, move person to an area free from risk of further exposure. Oxygen or artificial respiration should be administered as needed. Asthmatic-type symptoms may develop and may be immediate or delayed up to several hours. Treatment is essentially symptomatic. A physician should be consulted.
Ingestion	‣ For advice, contact a Poisons Information Centre or a doctor at once. ‣ Urgent hospital treatment is likely to be needed. ‣ 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. ‣ Transport to hospital or doctor without delay.

Indication of any immediate medical attention and special treatment needed

For chronic or short term repeated exposures to pyrethrum and synthetic pyrethroids:
- Mammalian toxicity of pyrethrum and synthetic pyrethroids is low, in part because of poor bioavailability and a large first pass extraction by the liver.
- The most common adverse reaction results from the potent sensitising effects of pyrethrins.
- Clinical manifestations of exposure include contact dermatitis (erythema, vesiculation, bullae); anaphylactoid reactions (pallor, tachycardia, diaphoresis) and asthma. [***Ellenhorn Barceloux***]
- In cases of skin contact, it has been reported that topical application of Vitamin E Acetate (alpha-tocopherol acetate) has been found to have high therapeutic value, eliminating almost all skin pain associated with exposure to synthetic pyrethroids. [***Incitec***]

For sodium channel blockers:
· Immediate initial management must begin with an assessment of airway, breathing, and circulation. Many patients present with hypotension, bradycardia or tachycardia and altered mental status. An endotracheal tube or other advanced airways should be placed in patients who are unable to protect their airway.
· The cornerstone of treatment is the administration of sodium bicarbonate. It is indicated for patients with an ECG demonstrating a QRS duration >100 ms or any suspicious QT prolongation or dysrhythmia. Sodium bicarbonate is beneficial in raising the serum pH and increasing the extracellular sodium. Alkalinization leads to an increase of the electrochemical gradient across cell membranes which helps to offload sodium channels. It might also increase the protein binding of the offending agent. Patients should be given 1-2 mEq/kg as a bolus dose. Bolus doses can be administered until the QRS duration is less than 100 ms. This can be followed with a continuous infusion of sodium bicarbonate of 2-3 50 mEq ampules in one litre of dextrose 5% in water (D5W). Hypertonic saline has been used but is not routinely recommended; it remains an option in dire circumstances as reported in a case of flecainide overdose.
· Management of hypotension requires a combination of volume resuscitation and vasopressor and inotropic support. Use of inotropic agents such as dobutamine helps increase cardiac output while the effects of the toxicity dissipate. Addition of vasopressors such as norepinephrine, vasopressin, or epinephrine might be considered for haemodynamic support. These agents lead to vasoconstriction and an increase in the systemic vascular resistance, resulting in increased systemic blood pressure.
· Patients with sodium channel blocker toxicity from lidocaine have benefited from the administration of 20% lipid emulsion if they are hemodynamically unstable. The mechanism of action of lipid emulsion is unclear; however, it is hypothesized that it acts as a lipid sink, with an electrochemical gradient drawing the lidocaine into the lipid. Patients should be given a 1.5 mL/kg bolus followed by a 0.25 mL/kg infusion. Few studies exist on lipid emulsion for other sodium channel toxicities. There is controversy over its utility in TCA overdose.
· Extracorporeal membrane oxygenation (ECMO) has been used in a refractory case with reported survival. In general, the drugs which cause sodium channel blockade toxicity are highly lipophilic, have a wide volume of distribution, and are not dialyzable.
· Seizure management is accomplished with benzodiazepine medications, such as lorazepam and midazolam. For refractory seizures, loading of antiepileptic medications such as levetiracetam is recommended. Phenytoin and its derivatives should be avoided in this situation as phenytoin is itself a sodium channel blocker and will likely lead to clinical deterioration. Intubation and sedation with propofol should be considered for seizures refractory to other management.
· Complications of sodium channel blocker toxicity include cardiogenic shock, hypotension, bradycardia or tachycardia, cardiovascular collapse, respiratory depression, encephalopathy, status epilepticus, and death.
· The high mortality rate of tricyclic antidepressant (TCA) overdose is well known.
· Class I antiarrhythmic toxicity is associated with a significantly higher mortality rate (22.5%) compared with other drugs (1%). Prompt recognition and treatment are vital to minimize morbidity and mortality. Diarrhea, nausea, headache and dizziness are also common side effects of many Class I drugs. Quinidine enhances digitalis toxicity, especially if hypokalemia is present. Quinidine, by delaying repolarization, can precipitate torsades de pointes (especially in patients with long-QT syndrome), a ventricular tachyarrhythmia caused by afterdepolarizations. Disopyramide is contraindicated for patients with uncompensated heart failure because of its negative inotropic actions; propafenone can also depress inotropy. IC compounds can cause increased risk of sudden death in patients with a prior history of myocardial infarction or sustained ventricular arrhythmias.
· There tends to be no significant effect on the atrioventricular (AV) node as it does not depend on fast sodium channels. Instead, sodium channel blockade produces a variety of cardiac effects as demonstrated by their impact on the myocyte action potential. With varying degrees of potency, class I agents decrease the slope and amplitude of phase 0, and thereby reduce the rate of depolarization and the conduction velocity through the myocyte. This leads to a slower depolarization of the cell and is important for the desired therapeutic suppression of tachydysrhythmias through reentrant mechanisms. Concomitant anticholinergic effects of many of these drugs can complicate their sodium channel effects.
Sodium Channel Blocker Toxicity: Kaylinn Dokken; Patrick Fairley. Treasure Island (FL): StatPearls Publishing; 2020 Jan-.
https://www.ncbi.nlm.nih.gov/books/NBK430685/
For sub-chronic and chronic exposures to isocyanates:
- This material may be a potent pulmonary sensitiser which causes bronchospasm even in patients without prior airway hyperreactivity.
- Clinical symptoms of exposure involve mucosal irritation of respiratory and gastrointestinal tracts.
- Conjunctival irritation, skin inflammation (erythema, pain vesiculation) and gastrointestinal disturbances occur soon after exposure.
- Pulmonary symptoms include cough, burning, substernal pain and dyspnoea.
- Some cross-sensitivity occurs between different isocyanates.
- Noncardiogenic pulmonary oedema and bronchospasm are the most serious consequences of exposure. Markedly symptomatic patients should receive oxygen, ventilatory support and an intravenous line.
- Treatment for asthma includes inhaled sympathomimetics (epinephrine [adrenalin], terbutaline) and steroids.
- Activated charcoal (1 g/kg) and a cathartic (sorbitol, magnesium citrate) may be useful for ingestion.
- Mydriatics, systemic analgesics and topical antibiotics (Sulamyd) may be used for corneal abrasions.
- There is no effective therapy for sensitised workers.

[Ellenhorn and Barceloux; Medical Toxicology]

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NOTE: Isocyanates cause airway restriction in naive individuals with the degree of response dependant on the concentration and duration of exposure. They induce smooth muscle contraction which leads to bronchoconstrictive episodes. Acute changes in lung function, such as decreased FEV1, may not represent sensitivity.
[Karol & Jin, Frontiers in Molecular Toxicology, pp 56-61, 1992]
Personnel who work with isocyanates, isocyanate prepolymers or polyisocyanates should have a pre-placement medical examination and periodic examinations thereafter, including a pulmonary function test. Anyone with a medical history of chronic respiratory disease, asthmatic or bronchial attacks, indications of allergic responses, recurrent eczema or sensitisation conditions of the skin should not handle or work with isocyanates. Anyone who develops chronic respiratory distress when working with isocyanates should be removed from exposure and examined by a physician. Further exposure must be avoided if a sensitivity to isocyanates or polyisocyanates has developed.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substance or mixture

Fire Incompatibility	‣ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result

Advice for firefighters

Fire Fighting	‣ 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 fire fighting procedures suitable for surrounding area. ‣ **Do not** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire. ‣ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	‣ 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 acrid smoke. ‣ Mists containing combustible materials may be explosive. Combustion products include: ‣ carbon dioxide (CO_2) isocyanates and minor amounts of hydrogen cyanide hydrogen chloride phosgene hydrogen fluoride nitrogen oxides (NOx) sulfur oxides (SOx) ‣ other pyrolysis products typical of burning organic material. May emit poisonous fumes.
HAZCHEM	2X

SECTION 6 Accidental release measures

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	Environmental hazard - contain spillage. ‣ 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	Environmental hazard - contain spillage. ‣ 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 ($Na_2S_2O_5$) or sodium bisulfite ($NaHSO_3$), or 12% sodium sulfite (Na_2SO_3) 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.

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SECTION 7 Handling and storage

Precautions for safe handling

Safe handling	‣ Avoid skin contact, including inhalation. ‣ Wear protective clothing when risk of exposure occurs. ‣ Use in a well-ventilated area. ‣ Prevent concentration in hollows and sumps. ‣ **DO NOT** enter confined spaces until atmosphere has been checked. ‣ **DO NOT** allow material to come in direct contact with human skin or eyes. ‣ **DO NOT** allow material to come in contact with exposed food or food contact surfaces. ‣ Suitable PPE must be worn at all times. ‣ Avoid contact with incompatible materials. ‣ When handling, **DO NOT** eat, drink or smoke. ‣ Keep containers securely sealed when not in use. ‣ Avoid physical damage to containers. ‣ Always wash hands with soap and water after handling. ‣ Work clothes should be laundered separately. Launder contaminated clothing before re-use. ‣ Use good occupational work practice. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS. ‣ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained. ‣ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	‣ Store in original containers. ‣ Keep containers securely sealed. ‣ Store in a cool, dry, well-ventilated area. ‣ Store away from incompatible materials and foodstuff containers. ‣ Protect containers against physical damage and check regularly for leaks. ‣ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	‣ Glass container is suitable for laboratory quantities ‣ Lined metal can, lined metal pail/ can. ‣ Plastic pail. ‣ Polyliner drum. ‣ Packing as recommended by manufacturer. ‣ Check all containers are clearly labelled and free from leaks. For low viscosity materials ‣ Drums and jerricans must be of the non-removable head type. ‣ Where a can is to be used as an inner package, the can must have a screwed enclosure. For materials with a viscosity of at least 2680 cSt. (23 deg. C) and solids (between 15 C deg. and 40 deg C.): ‣ Removable head packaging; ‣ Cans with friction closures and ‣ low pressure tubes and cartridges may be used. - Where combination packages are used, and the inner packages are of glass, there must be sufficient inert cushioning material in contact with inner and outer packages *. - In addition, where inner packagings are glass and contain liquids of packing group I and II there must be sufficient inert absorbent to absorb any spillage *. - * unless the outer packaging is a close fitting moulded plastic box and the substances are not incompatible with the plastic.
Storage incompatibility	Pyrethrins and permethrins: ‣ are unstable in the presence of light, heat, moisture and air ‣ are hydrolysed by oxygen and/ or sunlight ‣ may react with strong oxidisers to produce fire and explosions ‣ are incompatible with alkalis ‣ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	MDI oligomer	Isocyanates, all (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Not Available
Australia Workplace exposure limits for airborne contaminants (WEL list) - Appendix A - Workplace Exposure Limits	MDI oligomer	Isocyanates, (poly-) (as-NCO)	0.02 mg/m3	0.07 mg/m3	Not Available	Workers exposed to this chemical may require specific health monitoring (see regulations 368-378, Schedule 14 to the model WHS Regulations).

MATERIAL DATA

Airborne particulate or vapour must be kept to levels as low as is practicably achievable given access to modern engineering controls and monitoring hardware. Biologically active compounds may produce idiosyncratic effects which are entirely unpredictable on the basis of literature searches and prior clinical experience (both recent and past). 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%.
For isocyanates:
Some jurisdictions require that health surveillance be conducted on occupationally exposed workers. This should emphasise:
‣ demography, occupational and medical history and health advice
‣ completion of a standardised respiratory questionnaire
‣ physical examination of the respiratory system and skin
‣ standardised respiratory function tests such as FEV1, FVC and FEV1/FVC

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Various portable or stationary instruments are available for the continuous measurement of isocyanates in the air. All of them function on the principle of colourimetric evaluation of an indicator paper strip. They are operating continuously and unattended. Paper tape systems are easy to use and do not require skilled analysts to operate them. They give rapid results and are therefore suitable for leak detection and in emergency situations. However,:

- they may read incorrect at very high or very low humidity,
- are unsuitable for aerosols
- and may not be accepted for purposes of regulatory compliance.

Air monitoring of isocyanates requires sound analytical knowledge. In order to obtain reliable results only laboratories with experience in that specific area should be engaged with such measurements

In the evaluation of the German MAK Commission the justification of the OEL for 4,4 - MDI/ pMDI is established based on the isocyanate (NCO) group which is common to the monomeric, oligomeric and the polymeric MDIs. This NCO group is highly reactive (see toxicokinetics and category justification for details). Due to this high reactivity of the functional NCO group towards nucleophilic biomolecules the primary health effect of MDI is irritation at the point of contact, which can be demonstrated by the numerous acute, subacute and chronic bioassays, and sensitization.
The most sensitive health effect resulting from acute inhalation exposure to respirable aerosols of MDI is irritation to predominately the bronchio-alveolar part of the respiratory tract.
After inhalation exposure, MDI reacts with nucleophilic low and higher-molecular components of the liquid films that cover the airways, glutathione (GSH) represents the most important nucleophile in quantitative terms. The low-molecular adducts or conjugates of MDI are absorbed and direct transcarbamoylation results in plasma protein adducts (albumin, haemoglobin). All observed health effects resulting from exposure to respirable aerosols in acute, subchronic or chronic bioassays and human studies can be allocated to primary alveolar reactivity (respiratory irritation and/or sensitization). No systemic effect other than secondary to primary irritation has been described.
Biomonitoring for exposure to diisocyanates typically looks to assay derivatives (diamines) following hydrolysis of biological fluids and is routinely employed to measure occupational exposure to MDI and other diisocyanates. For diamine analysis, samples of urine are typically used, although blood samples can also be used. However, these markers are not specific for the diisocyanate exposure. The urine biomarkers (after acid or base hydrolysis) reflected recent exposures whereas certain haemoglobin (Hb) biomarkers did not necessarily correlate with the urine biomarkers, and were considered to reflect overall exposures over a longer term. The hydrolysis methods and conditions used release differing amounts of the diamine analyte.
Hydrolysis analytes are at low concentrations and proportionally little of the dose is in urine or blood, and that there is no standardised method for measuring biomarkers in hydrolyzed urine. Investigation of diisocyanate specific biomarkers has focused on the conjugated molecules in blood. Typically, conjugates with Hb or albumin (Alb) have been assessed, and there has been progress in application of experiments in animals to biomonitoring of human exposures to MDI.
The role for glutathione as an intermediary in transport of diisocyanates is now supported by good evidence using various model compounds. The most probable reactions of isocyanates with biological macromolecules are with the amine (mixed urea), the hydroxyl (carbamate) and the sulphydryl (thiolytic acid ester) and that latter is of a reversible nature.
The thiocarbamate bond of isocyanate-sulphydryl is reversible, and various authors have found release and transfer of MDI moieties from thiocarbamate conjugates to other nucleophiles, notably protein. The conjugates were shown to be recognised by serum IgG from MDI exposed workers, demonstrating a non-enzymatic, thiol-mediated transcarbamolyating mechanism to protein.
The methods to identify and quantify MDI-adducts to plasma proteins particularly albumin (Alb) and to haemoglobin (Hb) have now been applied to biological monitoring, particularly useful since the amount of adducts would be indicative of an integrated exposure. In addition these adducts are specific for MDI exposure. However the total amount of the analyte MDA, retrievable from Hb-adducts and urinary precursors, accounts for less than 0.5 % of the applied dose of MDI, and the lack of linearity of biomarker to exposure dose makes uncertain the extrapolation from the yield of biomarkers in urine or blood towards inhalative MDI exposure. The use of protein adducts for biomonitoring appears to overcome some of these difficulties and benefits from specificity of the analyte. To date no diisocyanate specific urinary biomarker has been identified. For blood, the MDI-specific methods developed are: Hb-conjugate derived hydantoin, Alb-lysine conjugates and peptide conjugates. On the basis of limits of detection, the Hb-hydantoin method is most sensitive compared to the Alb-lysine method which in turn is more sensitive than the signature peptide method. The Hb-hydantoin method covers a longer period of exposure than the Alb-lysine, due to the longer half life of the erythrocyte compared to serum albumin.
CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas)
(CEL = Chemwatch Exposure Limit)
For pyrethrum and its active components:
IDLH Level: 5000 mg/m3
Pyrethrum and/or its active components, the pyrethrins, cause dermatitis and sensitisation. Ingestion of massive doses can induce convulsions, vomiting and bradycardia. Animals exhibit liver damage and death through respiratory failure. The recommended TLV-TWA is equivalent to an occupational dose of 0.7 mg/kg/day and is thought to minimise the potential for systemic effects. The TLV may NOT prevent the development of hypersensitisation, particularly among those with pre-existing allergies to pollen and related agents.
Synthetic pyrethrins (pyrethroids) often produce a range of toxic effects resembling pyrethrum; in the absence of a regulated exposure limit prudence dictates that the value for pyrethrum serves as a reference.

Exposure controls

Appropriate engineering controls	**For potent pharmacological agents:** **Solutions Handling:** ▸ Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area. ▸ Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation. ▸ In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. ▸ Ensure gloves are protective against solvents in use. **Unless written procedures, specific to the workplace are available, the following is intended as a guide:** ▸ **For Laboratory-scale handling of Substances assessed to be toxic by inhalation.** ***Quantities of up to 25 grams*** may be handled in Class II biological safety cabinets *; ***Quantities of 25 grams to 1 kilogram*** may be handled in Class II biological safety cabinets* or equivalent containment systems; ***Quantities exceeding 1 kg*** may be handled either using specific containment, a hood or Class II biological safety cabinet*, ▸ HEPA terminated local exhaust ventilation should be considered at point of generation of dust, fumes or vapours. ▸ The need for respiratory protection should also be assessed where incidental or accidental exposure is anticipated. Dependent on levels of contamination, PAPR, full face air purifying devices with P2 or P3 filters or air supplied respirators should be evaluated. When handling: ***Quantities of up to 25 grams***, an approved respirator with HEPA filters or cartridges should be considered; ***Quantities of 25 grams to 1 kilogram,*** a half-face negative pressure, full negative pressure, or powered helmet-type air purifying respirator should be considered. ***Quantities in excess of 1 kilogram*****,** a full face negative pressure, helmet-type air purifying, or supplied air respirator should be considered. Written procedures, specific to a particular work-place, may replace these recommendations * For Class II Biological Safety Cabinets, Types B2 or B3 should be considered. Where only Class I, open fronted Cabinets are available, glove panels may be added, Laminar flow cabinets do not provide sufficient protection when handling these materials unless especially designed to do so. **Pilot Plant and Production** ▸ Wear appropriate gloves; lab coat, nylon coveralls or disposable Tyvek suit; safety glasses, safety shoes, and disposable booties. Use good manufacturing practices (i.e., cGMPs). ▸ Protective garment (coveralls, Tyvek, lab coat) is not to be worn outside the work area. ▸ Clean/dirty/decontamination areas are to be established. ▸ Negative/positive air pressure relationships and buffer zones required (i.e., ante-room/degowning room/airlock). ▸ Area access is to be restricted.

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	‣ High-energy operations such as milling, particle sizing, spraying or fluidising should be done within an approved emission control or containment system. ‣ Develop cleaning procedures and techniques that limit potential exposure **For potent pharmacological agents:** **Powders** To prevent contamination and overexposure, no open handling of powder should be allowed. ‣ Powder handling operations are to be done in a powders weighing hood, a glove box, or other equivalent ventilated containment system. ‣ In situations where these ventilated containment hoods have not been installed, a non-ventilated enclosed containment hood should be used. ‣ Pending changes resulting from additional air monitoring data, up to 300 mg can be handled outside of an enclosure provided that no grinding, crushing or other dust-generating process occurs. ‣ An air-purifying respirator should be worn by all personnel in the immediate area in cases where non-ventilated containment is used, where significant amounts of material (e.g., more than 2 grams) are used, or where the material may become airborne (as through grinding, etc.). ‣ Powder should be put into solution or a closed or covered container after handling. ‣ If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. **Solutions Handling:** ‣ Solutions can be handled outside a containment system or without local exhaust ventilation during procedures with no potential for aerosolisation. If the procedures have a potential for aerosolisation, an air-purifying respirator is to be worn by all personnel in the immediate area. ‣ Solutions used for procedures where aerosolisation may occur (e.g., vortexing, pumping) are to be handled within a containment system or with local exhaust ventilation. ‣ In situations where this is not feasible (may include animal dosing), an air-purifying respirator is to be worn by all personnel in the immediate area. If using a ventilated enclosure that has not been validated, wear a half-mask respirator equipped with HEPA cartridges until the enclosure is validated for use. ‣ Ensure gloves are protective against solvents in use. ‣ All processes in which isocyanates are used should be enclosed wherever possible. ‣ Total enclosure, accompanied by good general ventilation, should be used to keep atmospheric concentrations below the relevant exposure standards. ‣ If total enclosure of the process is not feasible, local exhaust ventilation may be necessary. Local exhaust ventilation is essential where lower molecular weight isocyanates (such as TDI or HDI) is used or where isocyanate or polyurethane is sprayed. ‣ Where other isocyanates or pre-polymers are used and aerosol formation cannot occur, local exhaust ventilation may not be necessary if the atmospheric concentration can be kept below the relevant exposure standards. ‣ Where local exhaust ventilation is installed, exhaust vapours should not be vented to the exterior in such a manner as to create a hazard.
Individual protection measures, such as personal protective equipment	
Eye and face protection	For laboratory, larger scale or bulk handling or where regular exposure in an occupational setting occurs: ‣ Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent] ‣ Face shield. Full face shield may be required for supplementary but never for primary protection of eyes ‣ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lens 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].
Skin protection	See Hand protection below
Hands/feet protection	‣ When handling corrosive liquids, wear trousers or overalls outside of boots, to avoid spills entering boots. ‣ Elbow length PVC gloves **NOTE:** ‣ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ‣ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application. The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice. Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

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	Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ▸ Rubber gloves (nitrile or low-protein, powder-free latex, latex/ nitrile). Employees allergic to latex gloves should use nitrile gloves in preference. ▸ Double gloving should be considered. ▸ PVC gloves. ▸ Change gloves frequently and when contaminated, punctured or torn. ▸ Wash hands immediately after removing gloves. ▸ Protective shoe covers. [AS/NZS 2210] ▸ Head covering. ▸ Butyl rubber gloves · Nitrile rubber gloves (Note: Nitric acid penetrates nitrile gloves in a few minutes.)
Body protection	See Other protection below
Other protection	All employees working with isocyanates must be informed of the hazards from exposure to the contaminant and the precautions necessary to prevent damage to their health. They should be made aware of the need to carry out their work so that as little contamination as possible is produced, and of the importance of the proper use of all safeguards against exposure to themselves and their fellow workers. Adequate training, both in the proper execution of the task and in the use of all associated engineering controls, as well as of any personal protective equipment, is essential. Employees exposed to contamination hazards should be educated in the need for, and proper use of, facilities, clothing and equipment and thereby maintain a high standard of personal cleanliness. Special attention should be given to ensuring that all personnel understand instructions, especially newly recruited employees and those with local-language difficulties, where they are known. ▸ For quantities up to 500 grams a laboratory coat may be suitable. ▸ For quantities up to 1 kilogram a disposable laboratory coat or coverall of low permeability is recommended. Coveralls should be buttoned at collar and cuffs. ▸ For quantities over 1 kilogram and manufacturing operations, wear disposable coverall of low permeability and disposable shoe covers. ▸ For manufacturing operations, air-supplied full body suits may be required for the provision of advanced respiratory protection. ▸ Eye wash unit. ▸ Ensure there is ready access to an emergency shower. ▸ For Emergencies: Vinyl suit

Respiratory protection

Full face respirator with supplied air.

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.
- 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

For spraying or operations which might generate aerosols:
Full face respirator with supplied air.

- In certain circumstances, personal protection of the individual employee is necessary. Personal protective devices should be regarded as being supplementary to substitution and engineering control and should not be used in preference to them as they do nothing to eliminate the hazard.
- However, in some situations, minimising exposure to isocyanates by enclosure and ventilation is not possible, and occupational exposure standards may be exceeded, particularly during on-site mixing of paints, spray-painting, foaming and maintenance of machine and ventilation systems. In these situations, air-line respirators or self-contained breathing apparatus complying with the appropriate nationals standard must be used.
- Organic vapour respirators with particulate pre- filters and powered, air-purifying respirators are NOT suitable.
- Personal protective equipment must be appropriately selected, individually fitted and workers trained in their correct use and maintenance. Personal protective equipment must be regularly checked and maintained to ensure that the worker is being protected.
- Air- line respirators or self-contained breathing apparatus complying with the appropriate national standard should be used during the clean-up of spills and the repair or clean-up of contaminated equipment and similar situations which cause emergency exposures to hazardous atmospheric concentrations of isocyanate.

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Beige to Tan coloured liquid.

Physical state	Liquid	**Relative density (Water = 1)**	Not Available
Odour	Slight	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	Not Available	**Decomposition temperature (°C)**	Not Available

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Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>103	**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)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water	Partly miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ 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

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	There is sufficient evidence to classify this material as skin corrosive or irritating.
c) Serious Eye Damage/Irritation	There is sufficient evidence to classify this material as eye damaging or irritating
d) Respiratory or Skin sensitisation	There is sufficient evidence to classify this material as sensitising to skin or the respiratory system
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	There is sufficient evidence to classify this material as carcinogenic
g) Reproductivity	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	There is sufficient evidence to classify this material as toxic to specific organs through repeated exposure
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	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. Inhalation of pyrethrins may produce nausea, vomiting, sneezing, serious nasal discharge, nasal stuffiness and asthma. High concentrations may produce hyperexcitability, incoordination, tremors, muscular paralysis and death (due to respiratory failure). There have been some reports of transient facial tingling (paraesthesia) which lasts a few hours after exposure. In common with natural pyrethrins, the material may produce central nervous system stimulation with nausea, vomiting, gastroenteritis, diarrhoea, hypersensitivity, incoordination, tremors, muscle paralysis, convulsion, coma, and respiratory failure. Synthetic pyrethroids are neuropoisons acting on the axons in the peripheral and central nervous systems by interacting with sodium channels in mammals and/or insects. A single dose produces toxic signs in mammals, such as tremors, hyperexcitability, salivation, choreoathetosis (a condition characterised by aimless muscle movements and involuntary motions), and paralysis. The signs disappear fairly rapidly, and the animals recover, generally within a week. At near-lethal dose levels, synthetic pyrethroids cause transient changes in the nervous system, such as axonal swelling and/or breaks and myelin degeneration in the sciatic nerves. They are not considered to cause delayed neurotoxicity of the kind induced by some organophosphorus compounds. Pyrethroids have at least two distinct actions- a short-term pharmacological effect at near-lethal dose levels and a more long-term neurotoxic effect that results in sparse axonal nerve damage. This synthetic pyrethroid produces Type II syndrome (or CS syndrome characteristic of those ester containing the alpha-cyano substituent) and, in rats, causes coarse tremors, clonic seizures , sinus writhing (choreoathetosis) and profuse salivation without lachrymation: hence the term CS (choreoathetosis/salivation) syndrome. The bulk of evidence indicates that Type II syndrome principally involves an action in the mammalian central nervous system. In poorly ventilated spaces, exposure may result in toxic effects in humans. Ingestion (associated with sloppy handling) of deltamethrin, fenvalerate and cypermethrin (all Type II compounds), amongst Chinese cotton crop workers, produced epigastric pain, nausea and vomiting, headache, dizziness, anorexia, fatigue, tightness of the chest, blurred vision, paraesthesia, palpitations, course muscular fasciculations in large muscle extremities and disturbances of consciousness. Severe poisonings produced convulsive attacks (lasting 30-120 seconds) accompanied by flexion of upper limbs, extension of lower limbs and loss of consciousness. These seizures occurred between 10 and 20 times a day in the first week after exposure, gradually decreasing in frequency, with complete recovery in two to three weeks. direct neurotoxicity) there is little evidence of allergic-type responses amongst humans exposed to the synthetic pyrethroid esters.

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	The vapour/mist may be highly irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning for several hours after exposure. Sensitized people can react to very low doses, and should not be allowed to work in situations allowing exposure to this material. Continued exposure of sensitised persons may lead to possible long term respiratory impairment. Inhalation hazard is increased at higher temperatures. Inhalation of dusts, generated by the material, during the course of normal handling, may produce severely toxic effects; these may be fatal. Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may produce severely toxic effects. Relatively small amounts absorbed from the lungs may prove fatal.
Ingestion	The material can produce chemical burns within the oral cavity and gastrointestinal tract following ingestion. 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 swallowing. Ingestion of pyrethrins may produce nausea, vomiting, headache and other central nervous system disturbances. Excitation, muscular tremors and a period of shock may be followed by death. Dogs fed 5000 ppm of pyrethrum, for 90 days, developed dyspnae, tremors, ataxia and excessive salivation. An estimated fatal human dose is thought to be 100 gms. for a typical 70 kg man (1430 mg/kg). No cases of acute human poisoning with cyhalothrin have been reported. After oral administration cyhalothrin is well absorbed and extensively metabolised. It is eliminated as polar conjugates in urine. In a 90-day feeding study in which rats were fed cyhalothrin at dose levels up to 250 mg/kg diet, reduced body weight gains were observed in males at 250 mg/kg diet. Marginal effects on mean erythrocyte volumes were noted in some treated groups as well as some liver changes. Lambda-cyhalothrin produced similar effects. The no-observed-effect-level was 50 mg/kg diet. Dogs produced clinical signs of pyrethroid neurotoxicity at doses of 10 mg/kg body weight in a 26 week oral study. At levels of 250 mg/kg diet for 28 days rats showed signs of neurotoxicity including high-stepping gait, ataxia and hypersensitivity. Thymic atrophy, adrenal enlargement with vacuolisation and incomplete spermatogenesis occurred at 750 mg/kg diet.
Skin Contact	The material can produce chemical burns following direct contact with the skin. 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 skin contact. Solutions of 0.5% strength 1,2-benzisothiazoline-3-one (BIT) are irritating to the skin. Allergenic 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%. 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. 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 Skin contact with natural pyrethrins may result in severe dermatitis and may also be associated with allergic rhinitis and asthma. Absorption through the skin may result in a toxic syndrome similar to that produced by inhalation. Systemic effects, following skin absorption, may include liver and kidney damage. Prolonged or repeated exposure may cause central nervous system effects and allergic skin reaction. 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. One form of toxicity associated with alpha-substituted synthetic pyrethroids, is a cutaneous parasthaesia observed amongst workers involved in spraying the material. This develops several hours following exposure and produces a stinging or burning sensation on the skin progressing, in some cases, to tingling and numbness, effects lasting for 12-18 hours. Chinese cotton crop workers reported dizziness, burning, itching or tingling sensations of exposed skin which was exacerbated by sweating and washing with warm water. These symptoms disappeared by 24 hours after exposure. Spilling these agents on the head, face and eyes resulted in pain, lachrymation, photophobia, congestion and oedema of the conjunctiva and eyelids. [Cassaret & Doull's Toxicology: The Basic Science of Poisoning, 4th Ed] Open cuts, abraded or irritated skin should not be exposed to this material Symptoms of subjective facial sensation to cyhalothrin have been described in manufacturing, formulation and laboratory work. This effect generally lasts for up to 72-hours after exposure. Medical examination has not revealed any neurological abnormalities. This sensation is believed to be brought about by repetitive firing of sensory nerve terminals in skin. Facial irritation has been reported following use of cyhalothrin in sheep sprays were the concentration was very low (0.002% w/v) - the characteristics of the spray were unclear. In 21-day dermal study at dose-levels up to 1000 mg/kg per day (in a polyethylene glycol vehicle), clinical signs of toxicity were observed at the highest dose levels. Cyhalothrin (but not lambda-cyhalothrin) is a moderate skin sensitiser in guinea-pigs. 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.
Eye	The material can produce chemical burns to the eye following direct contact. Vapours or mists may be extremely irritating. When applied to the eye(s) of animals, the material produces severe ocular lesions which are present twenty-four hours or more after instillation.
Chronic	On the basis, primarily, of animal experiments, concern has been expressed 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. Repeated or prolonged exposure to corrosives may result in the erosion of teeth, inflammatory and ulcerative changes in the mouth and necrosis (rarely) of the jaw. Bronchial irritation, with cough, and frequent attacks of bronchial pneumonia may ensue. Gastrointestinal disturbances may also occur. Chronic exposures may result in dermatitis and/or conjunctivitis. Practical evidence shows that inhalation of the material is capable of inducing a sensitisation reaction in a substantial number of individuals at a greater frequency than would be expected from the response of a normal population. Pulmonary sensitisation, resulting in hyperactive airway dysfunction and pulmonary allergy may be accompanied by fatigue, malaise and aching. Significant symptoms of exposure may persist for extended periods, even after exposure ceases. Symptoms can be activated by a variety of nonspecific environmental stimuli such as automobile exhaust, perfumes and passive smoking. 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. 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. Substances than can cuase 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

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Wherever it is reasonably practicable, exposure to substances that can cuase 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.
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.
Toxic: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed.
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.
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.
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.
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 %.
Chronic poisoning by natural pyrethrins may result in convulsion, tetanic paralysis, rapid and uneven heart beat, liver and kidney damage, or death.
The natural pyrethrins may produce hypersensitivity, especially following previous sensitising exposure. In general, repeated exposures over 2 or 3 years are required to elicit a response and involve exposure to pyrethrum rather than its individual components (including pyrethrins).
The sesquiterpene lactone (pyrethrosin) and the pyrethrum glycoproteins account for the immediate and delayed hypersensitivity seen in guinea pigs following a single injection of ground chrysanthemum in Freud's adjuvant. Mild erythematic vesicular dermatitis (with papules), pruritus, localized oedema (particularly of the face, lips and eyelids), rhinitis, tachycardia, pallor and sweating are the most common syndromes. An initial skin sensitisation can progress to marked dermal oedema and skin cracking. Pyrethrum dermatitis appears to increase in hot weather or under conditions were heavy perspiration is produced. The active ingredients of pyrethrum (except pyrethrin II) are inactive in patch tests. Those patients allergic to ragweed pollen are particularly sensitive to pyrethrin.
Rats fed on a diet of pyrethrins for 5000 ppm for 2 years showed some signs of tissue damage including liver lesions, bile duct proliferation and focal necrosis of the liver cells. A no-effect level of 1000 ppm found in animal experiments correspond to a daily dose of 3600 mg/man.
The isothiazolinones are known contact sensitisers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy.
Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones.
The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation.
Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* :

- The strongest sensitisers 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 sensitisers, the nonchlorinated isothiazolinones are considerably less potent sensitisers than are the chlorinated isothiazolinones.
- By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced.
- Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons.
- Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become available.

* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196
Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in ***Salmonella typhimurium*** strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells ***in vitro*** and of cytogenetic effects and DNA-binding ***in vivo.*** The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.
A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.
Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses
Persons with a history of asthma or other respiratory problems or are known to be sensitised, should not be engaged in any work involving the handling of isocyanates.
The chemistry of reaction of isocyanates, as evidenced by MDI, in biological milieu is such that in the event of a true exposure of small MDI doses to the mouth, reactions will commence at once with biological macromolecules in the buccal region and will continue along the digestive tract prior to reaching the stomach. Reaction products will be a variety of polyureas and macromolecular conjugates with for example mucus, proteins and cell components.
This is corroborated by the results from an MDI inhalation study. Following an inhalation exposure of rats to radiolabelled MDI, 79% of the dose was excreted in faeces. The faecal excretion in these animals was considered entirely due to ingestion of radioactivity from grooming and ingestion of deposited material from the nasopharangeal region via the mucociliary escalator, i.e. not following systemic absorption. The faecal radioactivity was tentatively identified as mixed molecular weight polyureas derived from MDI. Diamine was not present. Thus, for MDI and diisocyanates in general the oral gavage dosing route is inappropriate for toxicological studies and risk assessment.
It is expected that oral gavage dosing will result in a similar outcome to that produced by TDI or MDI, that is (1) reaction with stomach contents and (2) polymerization to solid polyureas.

- Reaction with stomach contents is very plausibly described in case reports of accidental ingestion of polymeric MDI based glue in domestic animals. Extensive polymerization and CO2 liberation resulting in an expansion of the gastric content is described in the stomach, without apparent acute chemical toxicity
- Polyurea formation in organic and aqueous phases has been described. In this generally accepted chemistry of hydrolysis of an isocyanate the initially produced carbamate decarboxylates to an amine which. The amine, as a reactive intermediate, then reacts very readily with the present isocyanate to produce a solid and inert polyurea. This urea formation acts as a pH buffer in the stomach, thus promoting transformation of the diisocyanate into polyurea, even under the acidic conditions.

At the resorbtive tissues in the small intestine, these high molecular reaction products are likely to be of very low bioavailability, which is substantiated by the absence of systemic toxicity in acute oral bioassays with rats at the OECD limit dose (LC50>2 g/kg bw).
The respiratory tract may be regarded as the main entry for systemically available isocyanates as evidenced following MDI exposures.
A detailed summary on urinary, plasma and in vitro metabolite studies is provided below. Taken together, all available studies provide convincing evidence that MDI-protein adduct and MDI-metabolite formation proceeds:

- via formation of a labile isocyanate glutathione (GSH)-adduct,
- then transfer to a more stable adduct with larger proteins, and
- without formation of free MDA. MDA reported as a metabolite is actually formed by analytical workup procedures (strong acid or base hydrolysis) and is not an identified metabolite in urine or blood

In two 104-week feeding studies with cyhalothrin no oncogenic effects were seen at dose levels up to 250 mg/kg diet and 500 mg/kg diet for rats and mice respectively. The-no-observed-effect level for systemic toxicity was 50 mg/kg diet (1.8 mg/kg rat body weight per day) and 20

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	mg/kg diet (1.9 mg/kg mouse body weight). Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

Apparent Myagi 250 Insecticide	**TOXICITY**	**IRRITATION**
	Not Available	Not Available
cyhalothricacid	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: 632 mg/kg[2]	Eye: no adverse effect observed (not irritating)[1]
	Inhalation (Rat) LC50: 0.083 mg/L4h[2]	Skin: no adverse effect observed (not irritating)[1]
	Oral (Rat) LD50: 144 mg/kg[2]	
MDI oligomer	**TOXICITY**	**IRRITATION**
	Dermal (rabbit) LD50: >9400 mg/kg[2]	Eye (Rodent - rabbit): 100mg - Mild
	Inhalation (Rat) LC50: 0.49 mg/L4h[2]	
	Oral (Rat) LD50: 43000 mg/kg[2]	
1,2-benzisothiazoline-3-one	**TOXICITY**	**IRRITATION**
	dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
	Oral (Rat) LD50: 454 mg/kg[1]	Skin (Human - man): 0.05%
		Skin (Human): 1%/1H
		Skin (Human): 5%/48H - Mild
		Skin: no adverse effect observed (not irritating)[1]

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***

CYHALOTHRICACID	Oral (rat) LD50: 56-79 mg/kg* Dermal (rat) LD50: 1293-1507 mg/kg* for lambda-cyhalothrin: CAS RN: 91465-08-6 RTECS No.: GZ 1227780 Convulsions, ataxia and changes in salivary glands recorded. lambda-cyhalothrin: ADI: 0.001 mg/kg/day NOEL: 0.1 mg/kg/day
MDI OLIGOMER	product Isocyanate vapours/mists are irritating to the upper respiratory tract and lungs; the response may be severe enough to produce bronchitis with wheezing, gasping and severe distress, even sudden loss of consciousness, and pulmonary oedema. Possible neurological symptoms arising from isocyanate exposure include headache, insomnia, euphoria, ataxia, anxiety neurosis, depression and paranoia. Gastrointestinal disturbances are characterised by nausea and vomiting. Pulmonary sensitisation may produce asthmatic reactions ranging from minor breathing difficulties to severe allergic attacks; this may occur following a single acute exposure or may develop without warning after a period of tolerance. A respiratory response may occur following minor skin contact. Skin sensitisation is possible and may result in allergic dermatitis responses including rash, itching, hives and swelling of extremities. Isocyanate-containing vapours/ mists may cause inflammation of eyes and nasal passages. Onset of symptoms may be immediate or delayed for several hours after exposure. Sensitised people can react to very low levels of airborne isocyanates. Unprotected or sensitised persons should not be allowed to work in situations allowing exposure to this material. The material may produce moderate eye irritation leading to inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis.
1,2-BENZISOTHIAZOLINE-3-ONE	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration. The predominant fate of the thiazole ring is oxidative ring scission catalysed by cytochrome P450 (CYP) and formation of the corresponding alpha-dicarbonyl metabolites and thioamide derivatives. The well-established toxicity associated with thioamides and thioureas has led to the speculation that thiazole toxicity is attributed to ring scission yielding the corresponding thioamide metabolite. Ring opening has also been observed in benzothiazoles. For instance, benzothiazole itself is converted to S-methylmercaptoaniline. **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. 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. **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. **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 sternebrae) but not external or visceral abnormalities. **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.

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Apparent Myagi 250 Insecticide & CYHALOTHRICACID & MDI OLIGOMER & 1,2-BENZISOTHIAZOLINE-3-ONE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested.
Apparent Myagi 250 Insecticide & MDI OLIGOMER	Allergic reactions which develop in the respiratory passages as bronchial asthma or rhinoconjunctivitis, are mostly the result of reactions of the allergen with specific antibodies of the IgE class and belong in their reaction rates to the manifestation of the immediate type. In addition to the allergen-specific potential for causing respiratory sensitisation, the amount of the allergen, the exposure period and the genetically determined disposition of the exposed person are likely to be decisive. Factors which increase the sensitivity of the mucosa may play a role in predisposing a person to allergy. They may be genetically determined or acquired, for example, during infections or exposure to irritant substances. Immunologically the low molecular weight substances become complete allergens in the organism either by binding to peptides or proteins (haptens) or after metabolism (prohaptens). Particular attention is drawn to so-called atopic diathesis which is characterised by an increased susceptibility to allergic rhinitis, allergic bronchial asthma and atopic eczema (neurodermatitis) which is associated with increased IgE synthesis. Exogenous allergic alveolitis is induced essentially by allergen specific immune-complexes of the IgG type; cell-mediated reactions (T lymphocytes) may be involved. Such allergy is of the delayed type with onset up to four hours following exposure. The substance is classified by IARC as Group 3: **NOT** classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing. For diisocyanates: In general, there appears to be little or no difference between aromatic and aliphatic diisocyanates as toxicants. In addition, there are insufficient data available to make any major distinctions between polymeric (<1000 MW) and monomeric diisocyanates. Based on repeated dose studies in animals by the inhalation route, both aromatic and aliphatic diisocyanates appear to be of high concern for pulmonary toxicity at low exposure levels. Based upon a very limited data set, it appears that diisocyanate prepolymers exhibit the same respiratory tract effects as the monomers in repeated dose studies. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. Most members of the diisocyanate category have not been tested for carcinogenic potential. Though the aromatic diisocyanates tested positive and the one aliphatic diisocyanate tested negative in one species, it is premature to make any generalizations about the carcinogenic potential of aromatic versus aliphatic diisocyanates. In the absence of more human data, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Diisocyanates are moderate to strong dermal sensitisers in animal studies. Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. For monomers, effects on the respiratory tract (lungs and nasal cavities) were observed in animal studies at exposure concentrations of less than 0.005 mg/L. The experimental animal data available on prepolymeric diisocyanates show similar adverse effects at levels that range from 0.002 mg/L to 0.026 mg/L. There is also evidence that both aromatic and aliphatic diisocyanates are acutely toxic via the inhalation route. **Oncogenicity:** Most members of the diisocyanate category have not been tested for carcinogenic potential. Commercially available Poly-MDI was tested in a 2-year inhalation study in rats. The tested material contained 47% aromatic 4,4'-methylenediphenyl diisocyanate (MDI) and 53% higher molecular weight oligomers. Interim sacrifices at one year showed that males and females in the highest dose group (6 mg/m3) had treatment related histological changes in the nasal cavity, lungs and mediastinal lymph nodes. The incidence and severity of degeneration and basal cell hyperplasia of the olfactory epithelium and Bowman's gland hyperplasia were increased in males at the mid and high doses and in females at the high dose following the two year exposure period. Pulmonary adenomas were found in 6 males and 2 females, and pulmonary adenocarcinoma in one male in the high dose group. However, aliphatic hexamethylene diisocyanate (HDI) was found not to be carcinogenic in a two year repeated dose study in rats by the inhalation route. HDI has not been tested in mice by the inhalation route. Though the oral route is not an expected route of exposure to humans, it should be noted that in two year repeated dose studies by the oral route, aromatic toluene diisocyanate (TDI) and 3,3'-dimethoxy-benzidine-4,4'-diisocyanate (dianisidine diisocyanate, DADI) were found to be carcinogenic in rodents. TDI induced a statistically significant increase in the incidence of liver tumors in rats and mice as well as dose-related hemangiosarcomas of the circulatory system and has been classified by the Agency as a B2 carcinogen. DADI was found to be carcinogenic in rats, but not in mice, with a statistically increase in the incidence of pancreatic tumors observed. **Respiratory and Dermal Sensitization:** Based on the available toxicity data in animals and epidemiologic studies of humans, aromatic diisocyanates such as TDI and MDI are strong respiratory sensitisers. Aliphatic diisocyanates are generally not active in animal models for respiratory sensitization. However, HDI and possibly isophorone diisocyanate (IPDI), are reported to be associated with respiratory sensitization in humans. Symptoms resulting from occupational exposure to HDI include shortness of breath, increased bronchoconstriction reaction to histamine challenges, asthmatic reactions, wheezing and coughing. Two case reports of human exposure to IPDI by inhalation suggest IPDI is a respiratory sensitiser in humans. In view of the information from case reports in humans, it would be prudent at this time to assume that both aromatic and aliphatic diisocyanates are respiratory sensitisers. Studies in both human and mice using TDI, HDI, MDI and dicyclohexylmethane-4,4'-diisocyanate (HMDI) suggest cross-reactivity with the other diisocyanates, irrespective of whether the challenge compound was an aliphatic or aromatic diisocyanate. Diisocyanates are moderate to strong dermal sensitisers in animal studies. There seems to be little or no difference in the level of reactivity between aromatic and aliphatic diisocyanates. **Dermal Irritation:** Skin irritation studies performed on rabbits and guinea pigs indicate no difference in the effects of aromatic versus aliphatic diisocyanates. The level of irritation ranged from slightly to severely irritating to the skin. One chemical, hydrogenated MDI (1,1-methylenebis-4-isocyanatocyclohexane), was found to be corrosive to the skin in guinea pigs.
Apparent Myagi 250 Insecticide & 1,2-BENZISOTHIAZOLINE-3-ONE	No significant acute toxicological data identified in literature search.
Apparent Myagi 250 Insecticide & CYHALOTHRICACID	Medications that act by sodium channel blockade have a wide variety of clinical applications. Broadly they include Vaughn Williams Class 1 antiarrhythmics, local anesthetics, many medications used to treat neuropathic pain (including tricyclic antidepressants (TCA)), anticonvulsants, and cocaine. Insecticides also cause sodium channel blockade. Toxicity of all these substances, whether intentional or accidental, can lead to catastrophic effects including death. It is essential to understand how these medications work, in order to effectively treat their toxicity and side effects. Sodium channel blocker toxicity results primarily from intentional overdose. Broadly speaking, sodium channel blockers cause metabolic, cardiac, and neurologic symptoms. This leads to haemodynamic compromise and metabolic acidosis, potentiating the effects of the medications and causing further sodium blockade. Sodium channel blockers cross the blood-brain barrier and act through multiple mechanisms. They inhibit the gamma-aminobutyric acid (GABA) system (primarily lidocaine), activate the sodium ouabain-sensitive current, stimulate 5-TH2C receptors, antagonize H1 receptors and block all noradrenaline activating effect. It is through these actions that adrenergic stimulation occurs. These medications in large doses are also pro-convulsant through the above mechanisms. TCAs are well known for their concomitant anticholinergic effects but they also produce potassium channel blockade, peripheral alpha blockade, and norepinephrine reuptake blockade, all of which will potentially cloud the clinical presentation. The anticholinergic effects of IA drugs can produce tachycardia, dry mouth, urinary retention, blurred vision and constipation. For cyhalothrin: Lambda cyhalothrin is moderately toxic in the technical form, but may be highly toxic via some routes in formulation (e.g., as Karate). Available data indicate that lambda cyhalothrin is moderately toxic via the oral route in test animals. Reported oral LD50 values are 79 mg/kg and 56 mg/kg for male and female rats, respectively. The vehicle used was corn oil. The rat oral LD50 has also been reported as 144 mg/kg . The reported rat LD50 for the technical product is similar, 64 mg/kg . These indicate moderate acute toxicity via the oral route of exposure.

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	No data were available regarding the acute toxicity of the technical compound via the inhalation route, but for Karate the reported 4-hour inhalation LC50s were 0.175 mg/L and 0.315 mg/L for female and male rats, respectively . These data indicate a moderate to high toxicity via the inhalation route for the formulated product Karate. The technical product has reported dermal LD50s of 632 mg/kg and 696 mg/kg for male and female rats (vehicle used was propane-1,2-diol) . It has also been found to be non-irritating to the skin of rabbits. and non-sensitising to the skin of guinea pigs but may cause mild eye irritation in rabbits. The formulated product, Karate, however, causes severe primary skin irritation in rabbits and mild skin sensitisation in guinea pigs . Primary eye irritation also was observed with the technical product . In addition to the corrosive effects to skin and eyes, other acute effects due to exposure to lambda cyhalothrin, like those of other pyrethroids, will be mainly neuropathy (effects on the nervous system) . Cyhalothrin may act on ion channels within the nerve cells (neurons) to disrupt proper function of the cells of both the peripheral and central nervous systems . At lower doses, this may take the form of stable, repetitive firing of the neuron, but high doses may result in depolarisation of the nerve cell and blockage of conduction . These effects may result in observable effects such as: tingling, burning or numbness sensations (particularly at the point of skin contact); tremors, incoordination of movement , paralysis or other disrupted motor function; and confusion or loss of consciousness . Since most pyrethroids are generally absorbed only poorly through the skin , the latter two systemic effects are unlikely unless the compound has been ingested. Effects are generally reversible due to rapid breakdown of the compound in the body. Like many compounds of the pyrethroid family, the observed toxicity of lambda cyhalothrin may vary according to not only the concentration of the active ingredient, but also according to the solvent vehicle . **Chronic toxicity:** The principal toxic effects noted in chronic studies were decreased body weight gain and decreased food consumption. These effects occurred in rats at oral doses of 1.5 mg/kg/day (the highest dose tested) in a three-generational study . In a two-year study in rats, no effects were observed at oral doses of 2.5 mg/kg/day and doses of up to 8.5 mg/kg/day produced no observable changes in the function or structure of the liver or nervous system. In this study, decreased body weight gain and decreased food consumption occurred at doses of 12.5 mg/kg/day as did elevation of plasma triglycerides. In a 26 week feeding study on dogs, doses of 2.5 mg/kg/day disrupted water absorption from the small intestine resulting in liquid faeces , and at doses of 3.5 mg/kg/day and higher, neurological effects were noted . In two teratology studies, no maternal toxicity was observed at doses of 10 mg/kg/day in both rats and rabbits . It is unlikely that lambda cyhalothrin would cause chronic effects in humans under normal conditions. **Reproductive Effects:** In two studies, lambda cyhalothrin caused reduced body weight gain at doses of 15 mg/kg/day in pregnant rats (highest dose tested) and at doses of 30 mg/kg/day in pregnant rabbits (also the highest dose tested) but these doses produced no observable reproductive effects. There were reduced numbers of viable offspring at doses of 50 mg/kg/day in the second and third generations in the three-generational rat study noted above . It is unlikely that lambda cyhalothrin would cause reproductive effects in humans under normal conditions. **Teratogenic Effects:** No teratogenic or foetotoxic effects were observed in teratology studies of lambda cyhalothrin in rats and rabbits at the highest doses tested in both species (15 mg/kg/day and 30 mg/kg/day, respectively.. Based on these data, it is unlikely that lambda cyhalothrin causes teratogenic effects. **Mutagenic Effects:** Lambda cyhalothrin produced negative results in all Ames mutagenicity assays using five different test strains, both with and without metabolic activation. Results of other in-vitro cytogenetic assays and chromosomal structural aberration tests indicated no mutagenic or genotoxic effects were caused by lambda cyhalothrin. The available evidence suggests that lambda cyhalothrin is non-mutagenic and non-genotoxic. **Carcinogenic Effects:** No carcinogenic effects have been noted in studies of lambda cyhalothrin on various test animals (rats, rabbits, dogs) . The evidence regarding the carcinogenicity of lambda cyhalothrin is inconclusive, but suggests that it is probably not carcinogenic. **Organ Toxicity:** No specific target organs or organ systems have been identified in the available studies of chronic toxicity. The nervous system may be affected after acute exposure. **Fate in Humans and Animals:** In rat studies, lambda cyhalothrin is rapidly metabolised and excreted via the urine and faeces . Hydrolytic cleavage of the ester bond occurs, forming more polar, water-soluble compounds which are less toxic and more easily eliminated
Apparent Myagi 250 Insecticide & CYHALOTHRICACID & MDI OLIGOMER	Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production.

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

Legend: ✘ ***– Data either not available or does not fill the criteria for classification***
✔ ***– Data available to make classification***

SECTION 12 Ecological information

Toxicity

Apparent Myagi 250 Insecticide	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available
cyhalothricacid	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	48h	Crustacea	<0.001mg/L	4
	EC50(ECx)	48h	Crustacea	<0.001mg/L	4
	LC50	96h	Fish	<0.001mg/L	4
MDI oligomer	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

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1,2-benzisothiazoline-3-one	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.07mg/L	2
	EC50	48h	Crustacea	0.097mg/L	4
	NOEC(ECx)	72h	Algae or other aquatic plants	0.04mg/L	2
	LC50	96h	Fish	0.067-0.29mg/L	4
Legend:	***Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. US EPA, Ecotox database - Aquatic Toxicity Data 4. ECETOC Aquatic Hazard Assessment Data 5. NITE (Japan) - Bioconcentration Data 6. METI (Japan) - Bioconcentration Data 7. Vendor Data 8. User defined data***				

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.
Toxic to bees.
for polyisocyanates:
Polyisocyanates are not readily biodegradable. However, due to other elimination mechanisms (hydrolysis, adsorption), long retention times in water are not to be expected. The resulting polyurea is more or less inert and, due to its molecular size, not bioavailable. Within the limits of water solubility, polyisocyanates have a low to moderate toxicity for aquatic organisms.
For synthetic pyrethroids:
Environmental fate:
Synthetic pyrethroids are examples of optimised insecticidal activity, selectivity and tailored environmental persistence. Through modifications of both acid and alcohol portions of the ester, compounds of desired residual activity have been synthesised whilst maintaining a biodegradable ester linkage. These compounds are generally very toxic to crustaceans and fish in laboratory bio assays. Under field conditions, however, the residues are tightly bound in sediment, and ingested residues are readily metabolised. Their toxicity in natural systems are generally less than laboratory test data might indicate. They are generally non-persistent in the environment.
In pond waters and in laboratory degradation studies, pyrethroid concentrations decrease rapidly due to sorption to sediment, suspended particles and plants. Microbial and photodegradation also occur
Pyrethrins are generally unstable in the presence of light, are hydrolysed rapidly under alkaline conditions and oxidise rapidly in air. Vapour phase pyrethrins may combine chemically with ozone to produce hydroxy radicals. Pyrethroids where the isobutenyl group attached to the cyclopropane moiety has been altered are more stable to sunlight than the early pyrethroids like allethrin or resmethrin. For this reason, pyrethroids such as permethrin, deltamethrin, cyhalothrin, cyfluthrin, and cypermethrin are more frequently applied outdoors to crops in comparison to the rapidly degraded pyrethroids like resmethrin and allethrin.
Because agricultural dose rates are low and biological degradation is generally rapid, residues are unlikely to attain significant levels. Permethrin disappears from ponds and streams within 6-24 hours, pond sediments within 7 days and foliage and forest soil within 58 days. Since pyrethrins and pyrethroids undergo photolysis in the atmosphere, they are also degraded by this mechanism in sunlit surface waters. Photosensitising agents found in natural waters such as fulvic and humic acids increase the rate of photolysis. Pyrethroids and pyrethrins also undergo hydrolysis in the environment at varying rates depending upon pH and temperature. Generally, hydrolysis is only an important environmental fate process under alkaline conditions and at temperatures of 20 deg. C or greater.
Based on the vapor pressure of the pyrethrins and pyrethroids, these compounds are expected to exist in both vapor and particulate phases in the ambient atmosphere. Vapor phase pyrethrins and pyrethroids are rapidly degraded in the atmosphere by direct photolysis and reaction with oxidants found in air such as photochemically-produced hydroxyl radicals, ozone, and nitrate radicals. Particulate phase compounds are slower to degrade, however, and can travel long distances before being removed from the air by wet and dry deposition.
Pyrethrins and pyrethroids are strongly adsorbed to soil surfaces and are not considered very mobile. A wide range of Koc values has been reported by different authors, but most of these values indicate a high degree of adsorption and little leaching potential. Since light is attenuated as a function of depth from the soil surface, photolysis of pyrethrins and pyrethroids is only an important environmental fate process at the surface of the soil. The potential for significant toxicity is not reached in fields. Under aerobic conditions in soil, permethrin degrades in a relatively short time (half-life 28 days).
Volatilisation from water and soil is expected to occur slowly for many of the pyrethroids since these compounds generally have low vapor pressures and Henry's law constants. When released to water, partitioning to suspended solids and sediment occurs rapidly. These compounds adsorb strongly to suspended solids and sediment in the water column, and this process significantly attenuates volatilisation. Volatilisation losses from foliage may be considerably greater than volatilisation from soils because pyrethrins and pyrethroids do not adsorb as strongly to the leafy component of vegetation as to soils. Pyrethrins and pyrethroids are often used indoors in sprays or aerosol bombs, and the volatilisation rates from glass or floor surfaces may be significantly faster than from soils since these compounds are not likely to adsorb as strongly to these surfaces.
Little data exist regarding the uptake and transport of pyrethrins and pyrethroids by plant material. Since many of these compounds are rapidly degraded in the environment, this transport mechanism may not be an important environmental fate process other than the initial settling of these compounds on the canopy following deposition. The aerial surface of a plant, including foliage, is covered by a cuticle, which serves as a barrier to water loss and to prevent penetration of applied chemicals or environmental pollutants. Once deposited on the surface, a chemical may be degraded, bind to the cuticle, or diffuse into the plant through the stomata. Since pyrethrins and pyrethroids adsorb strongly to soils, their uptake from roots and transport within plants is expected to be limited.
The general population is exposed to pyrethrins and pyrethroids primarily from food sources, especially fruits and vegetables. The tendency of young children to ingest soil, either intentionally through pica or unintentionally through hand-to-mouth activity, is well documented. These behavioral traits can result in ingestion of pyrethrins and pyrethroids present in soil and dust. Since these compounds are adsorbed strongly to soils, they may not be in a highly bioavailable form. Young children often play on the ground or on carpets and this will increase the likelihood of dermal exposure and inhalation of contaminated particles from soil, household dust and treated surfaces.
Drinking Water Standards:
pesticide 0.1 ug/l (UK max.)
Ecotoxicity:
Synthetic pyrethroids are extremely effective against insects, but are relatively safe to mammals and birds. One potential problem of pyrethroids is their extreme toxicity to aquatic organisms, where often <1 ug/L will produce toxic effects.
The half-lives for elimination of several pyrethroids by trout are all greater than 48 hours, while elimination half-lives in birds and mammals range from 6 to 12 hours
Pyrethroids are highly toxic to fish; with 96-hour LC50 values generally below 10 ug/l. Corresponding LD50 values in mammals and birds are in the range of several hundred to several thousand mg/kg. Fish sensitivity to the pyrethroids may be explained by their relatively slow metabolism and elimination of these compounds. The half-lives for elimination of several pyrethroids by trout are all greater than 48 hours, while elimination half-lives for birds and mammals range from 6 to 12 hours Generally, the lethality of pyrethroids to fish increases with increasing octanol/water partition coefficients The bioaccumulation factor of cypermethrin in fish is approximately 1000 when measured experimentally
For cyhalothrin:
Log Pow: 7
Environmental fate:
Lambda cyhalothrin is moderately persistent in the soil environment. Reported field half-lives range from four to 12 weeks. Its field half-life is probably close to 30 days in most soils. It shows a high affinity for soil; the reported Koc is 180,000 . Lambda cyhalothrin is not expected to be appreciably mobile in most soils. There is little potential for groundwater contamination. Soils with high sand content or with very low organic matter content may tend to retain the compound to a lesser degree. In field studies of Karate, leaching of lambda cyhalothrin and its degradates from the soil were minimal.
Breakdown products formed in the soil environment are similar to those formed in mammalian systems, via the hydrolysis of the central ester bond and oxidation. Breakdown rates of both the technical product and Karate were similar under aerobic and anaerobic conditions.
Breakdown of Chemical in Surface Water: Lambda cyhalothrin has extremely low water solubility and is tightly bound to soil, it is therefore not expected to be prevalent in surface waters. One possible source of infiltration into surface waters would be surface runoff. In this event, the compound would most probably remain bound to the solid particle and settle to the bottom.
Bioconcentration is possible in aquatic species, but bioaccumulation is not likely. Bioconcentration in channel catfish has been reported as minimal, with rapid depuration (elimination) . A bioconcentration factor of 858 has been reported in fish (species unspecified), but concentration was confined to non-edible tissues and rapid depuration was observed .
Ecotoxicity:
Highly toxic to fish and aquatic invertebrate species.
Slightly to practically non-toxic to birds.
For gamma-cyhalothrin - data ex Dow AgroSciences

Continued...

Fish LC50 (96): rainbow trout 0.00011 mg/l, bluegill sunfish 0.00047 mg/l
Daphnia magna EC50 (48 h): 0.000067 mg/l
Green algae EC50: 2.85 mg/l
Oral (bee) LD50 (48 h): 0.0013 mg/bee
Bird LC50 dietary: bobwhite quail =2644 mg/kg, mallard duck= 4430 mg/kg
Bird LD50 dietary: bobwhite quail = 2000 mg/kg
For lambda-cyhalothrin - data ex Orica:
Fish LC50 (96 h): rainbow trout: 0.00044 mg/l, bluegill sunfish 0.00021 mg/l, sheephead minnow 00081 mg/l
Daphnia magna EC50 (48 h): 0.00036 ug/l
Bird Oral LD50: mallard duck 3950 mg/kg
Bird Oral LC50 (3 d) dietary: bobwhite quail 7530 mg/kg
Oral (bee) LC50: 38 ng/bee
Dermal (bee) LD50: 909 ng/bee
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
cyhalothricacid	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
cyhalothricacid	HIGH (LogKOW = 7)
MDI oligomer	LOW (LogKOW = 10.46)

Mobility in soil

Ingredient	Mobility
cyhalothricacid	LOW (Log KOC = 476000)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal	▸ Containers may still present a chemical hazard/ danger when empty. ▸ Return to supplier for reuse/ recycling if possible. Otherwise: ▸ 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. 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. A Hierarchy of Controls seems to be common - the user should investigate: ▸ Reduction ▸ Reuse ▸ Recycling ▸ Disposal (if all else fails) 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. ▸ **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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

14.1. **UN number or ID number**	3352
	PYRETHROID PESTICIDE, LIQUID, TOXIC

Continued...

14.2. **UN proper shipping name**	
14.3. **Transport hazard class(es)**	Class: 6.1 Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: 61 223 274 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

14.1. **UN number**	3352
14.2. **UN proper shipping name**	Pyrethroid pesticide, liquid, toxic *
14.3. **Transport hazard class(es)**	ICAO/IATA Class: 6.1 ICAO / IATA Subsidiary Hazard: Not Applicable ERG Code: 6L
14.4. **Packing group**	III
14.5. **Environmental hazard**	Environmentally hazardous
14.6. **Special precautions for user**	Special provisions: A3 A4 Cargo Only Packing Instructions: 663 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 655 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y642 Passenger and Cargo Limited Maximum Qty / Pack: 2 L

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3352
14.2. **UN proper shipping name**	PYRETHROID PESTICIDE, LIQUID, TOXIC
14.3. **Transport hazard class(es)**	IMDG Class: 6.1 IMDG Subsidiary Hazard: Not Applicable
14.4. **Packing group**	III
14.5 **Environmental hazard**	Marine Pollutant
14.6. **Special precautions for user**	EMS Number: F-A, S-A Special provisions: 61 223 274 Limited Quantities: 5 L

14.7. Maritime transport in bulk according to IMO instruments

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

Not Applicable

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

Product name	Group
cyhalothricacid	Not Applicable
MDI oligomer	Not Applicable
1,2-benzisothiazoline-3-one	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
cyhalothricacid	Not Applicable
MDI oligomer	Not Applicable
1,2-benzisothiazoline-3-one	Not Applicable

SECTION 15 Regulatory information

Safety, health and environmental regulations / legislation specific for the substance or mixture

Continued...

cyhalothricacid is found on the following regulatory lists

Australia Chemicals with non-industrial uses removed from the Australian Inventory of Chemical Substances (old Inventory)
Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 2
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 7

MDI oligomer is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Model Work Health and Safety Regulations - Schedule 10 - Table 14.1 Hazardous chemicals (other than lead) requiring health monitoring
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6
Australian Inventory of Industrial Chemicals (AIIC)
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs - Not Classified as Carcinogenic

1,2-benzisothiazoline-3-one is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	Yes
Canada - DSL	No (cyhalothricacid)
Canada - NDSL	No (MDI oligomer; 1,2-benzisothiazoline-3-one)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (cyhalothricacid; MDI oligomer; 1,2-benzisothiazoline-3-one)
Europe - EINEC / ELINCS / NLP	No (MDI oligomer)
Japan - ENCS	No (cyhalothricacid)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	Yes
USA - TSCA	All chemical substances in this product have been designated as TSCA Inventory 'Active'
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	Yes
UAE - Control List (Banned/Restricted Substances)	No (MDI oligomer; 1,2-benzisothiazoline-3-one)
Legend:	***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	30/10/2026
Initial Date	30/10/2026

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.

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

Continued...

- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- 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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end of SDS