AC Pacer 550EC Fungicide Axichem Pty Ltd

Chemwatch: **5266-59** Version No: **6.1**

Safety Data Sheet according to WHS Regulations (Hazardous Chemicals) Amendment 2020 and ADG requirements

Chemwatch Hazard Alert Code: 2

Issue Date: **27/10/2023**Print Date: **14/02/2024**L.GHS.AUS.EN

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

Product Identifier

Product name	AC Pacer 550EC Fungicide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains propiconazole)
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	Systemic foliar fungicide.
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Details of the manufacturer or supplier of the safety data sheet

Registered company name	Axichem Pty Ltd	
Address	Palings Court Nerang QLD 4211 Australia	
Telephone	07 5596 1736	
Fax	Not Available	
Website	www.axichem.com.au	
Email	msds@axichem.com.au	

Emergency telephone number

Association / Organisation	CHEMWATCH EMERGENCY RESPONSE (24/7)	
Emergency telephone numbers	+61 1800 951 288	
Other emergency telephone numbers	+61 3 9573 3188	

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

SECTION 2 Hazards identification

Classification of the substance or mixture

COMBUSTIBLE LIQUID, regulated for storage purposes only

Poisons Schedule	S6
Classification ^[1]	Flammable Liquids Category 4, Acute Toxicity (Oral) Category 4, Skin Corrosion/Irritation Category 2, Sensitisation (Skin) Category 1, Serious Eye Damage/Eye Irritation Category 2A, Specific Target Organ Toxicity - Single Exposure (Respiratory Tract Irritation) Category 3, Specific Target Organ Toxicity - Repeated Exposure Category 2, 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

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Hazard pictogram(s)







Signal word

Warning

Hazard statement(s)

H227	Combustible liquid.
H302	Harmful if swallowed.
H315	Causes skin irritation.
H317	May cause an allergic skin reaction.
H319	Causes serious eye irritation.
H335	May cause respiratory irritation.
H373	May cause damage to organs through prolonged or repeated exposure.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P210	Keep away from heat, hot surfaces, sparks, open flames and other ignition sources. No smoking.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P280	Wear protective gloves, protective clothing, eye protection and face protection.
P264	Wash all exposed external body areas thoroughly after handling.
P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.
P272	Contaminated work clothing should not be allowed out of the workplace.

Precautionary statement(s) Response

P370+P378	In case of fire: Use alcohol resistant foam or normal protein foam to extinguish.	
P302+P352	IF ON SKIN: Wash with plenty of water.	
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.	
P333+P313	If skin irritation or rash occurs: Get medical advice/attention.	
P337+P313	If eye irritation persists: Get medical advice/attention.	
P362+P364	Take off contaminated clothing and wash it before reuse.	
P391	Collect spillage.	
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.	
P304+P340	IF INHALED: Remove person to fresh air and keep comfortable for breathing.	
P330	Rinse mouth.	

Precautionary statement(s) Storage

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

Precautionary statement(s) Disposal

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

SECTION 3 Composition / information on ingredients

Substances

See section below for composition of Mixtures

Mixtures

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CAS No	%[weight]	Name
60207-90-1	40-60	propiconazole
Not Available		(550 g/L)
Various	30-40	liquid hydrocarbons
Not Available	balance	Ingredients determined not to be hazardous
Legend:		2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - awn from C&L * EU IOELVs available

SECTION 4 First aid measures

Description of first aid measures

If this product comes in contact with the eyes: Wash out immediately with fresh running water. Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids.

- Seek medical attention without delay; if pain persists or recurs seek medical attention.
 - Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.

Skin Contact Immediately remove all contaminated clothing, including footwear. Flush skin and hair with running water (and soap if available).

► Other measures are usually unnecessary.

- Seek medical attention in event of irritation.
 If fumes, aerosols or combustion products are inhaled remove from contaminated area.
- 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 project operation.
- prevent aspiration.

 Observe the patient carefully.
- ▶ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious.
- ▶ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink.
- ► Seek medical advice.

If skin contact occurs:

Indication of any immediate medical attention and special treatment needed

In rats enzymatic attack occurs on the propyl side-chain and on the dioxolane ring with some attack at the 2,4-dichlorophenyl and 1,2,4-triazole rings. In mice the major metabolic pathway involves cleavage of the dioxolane ring.

As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).

For poisons (where specific treatment regime is absent):

BASIC TREATMENT

Inhalation

Ingestion

Establish a patent airway with suction where necessary.

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

ADVANCED TREATMENT

▶ Consider orotracheal or nasotracheal intubation for airway control in unconscious patient or where respiratory arrest has occurred.

- Positive-pressure ventilation using a bag-valve mask might be of use.
- Monitor and treat, where necessary, for arrhythmias.
- Start an IV D5W TKO. If signs of hypovolaemia are present use lactated Ringers solution. Fluid overload might create complications.
- Drug therapy should be considered for pulmonary oedema.
- Hypotension with signs of hypovolaemia requires the cautious administration of fluids. Fluid overload might create complications.
- Treat seizures with diazepam.
- ▶ Proparacaine hydrochloride should be used to assist eye irrigation.

BRONSTEIN, A.C. and CURRANCE, P.L.

EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

Treat symptomatically.

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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 substrate or mixture

Fire I	ncompatibi	lity
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Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may

Advice for firefighters			
Fire Fighting	 Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water courses. Use water delivered as a fine spray to control fire and cool adjacent 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 (CO2) hydrogen chloride phosgene nitrogen oxides (NOx) 443dh 3ds11 		
HAZCHEM	•3Z		

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

methods and material for	containment and cleaning up
Minor Spills	 Environmental hazard - contain spillage. 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. Moderate hazard. Clear area of personnel and move upwind. Alert Fire Brigade and tell them location and nature of hazard. Wear breathing apparatus plus protective gloves. Prevent, by any means available, spillage from entering drains or water course. No smoking, naked lights or ignition sources. Increase ventilation. Stop leak if safe to do so. Contain spill with sand, earth or vermiculite. Collect recoverable product into labelled containers for recycling. Absorb remaining product with sand, earth or vermiculite. Collect solid residues and seal in labelled drums for disposal.

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- Wash area and prevent runoff into drains.
- If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 Handling and storage

Precautions for safe handling

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

Safe handling

- ► Store in original containers.
- Keep containers securely sealed.
- Other information
- No smoking, naked lights or ignition sources.
- Store in a cool, dry, well-ventilated area.
 Store away from incompatible materials and foodstuff containers.
- ▶ Protect containers against physical damage and check regularly for leaks.
- ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container

- ► Metal can or drum
- ▶ Packaging as recommended by manufacturer.
- Check all containers are clearly labelled and free from leaks.

Storage incompatibility

Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Emergency Limits

Ingredient	TEEL-1	TEEL-2	TEEL-3
AC Pacer 550EC Fungicide	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
propiconazole	Not Available	Not Available
liquid hydrocarbons	Not Available	Not Available

Occupational Exposure Banding

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
propiconazole	E	≤ 0.1 ppm
Notes:	Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.	

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No exposure limits set by NOHSC or ACGIH

Exposure controls

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

The basic types of engineering controls are:

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

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

General exhaust is adequate under normal operating conditions. If risk of overexposure exists, wear SAA approved respirator. Correct fit is essential to obtain adequate protection. Provide adequate ventilation in warehouse or closed storage areas. Air contaminants generated in the workplace possess varying "escape" velocities which, in turn, determine the "capture velocities" of fresh circulating air required to effectively remove the contaminant.

Appropriate engineering controls

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

Within each range the appropriate value depends on:

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

Simple theory shows that air velocity falls rapidly with distance away from the opening of a simple extraction pipe. Velocity generally decreases with the square of distance from the extraction point (in simple cases). Therefore the air speed at the extraction point should be adjusted, accordingly, after reference to distance from the contaminating source. The air velocity at the extraction fan, for example, should be a minimum of 1-2 m/s (200-400 f/min.) for extraction of solvents generated in a tank 2 meters distant from the extraction point. Other mechanical considerations, producing performance deficits within the extraction apparatus, make it essential that theoretical air velocities are multiplied by factors of 10 or more when extraction systems are installed or used.

Individual protection measures, such as personal protective equipment











Eye and face protection

- Safety glasses with side shields.
- ► Chemical goggles. [AS/NZS 1337.1, EN166 or national equivalent]
- Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59].

Skin protection

See Hand protection below

NOTE:

▶ Wear chemical protective gloves, e.g. PVC.

▶ Wear safety footwear or safety gumboots, e.g. Rubber

Hands/feet protection

- 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

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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
- \cdot Fair when breakthrough time < 20 min
- · Poor when glove material degrades

For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended.

It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times.

Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example:

- Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of.
- · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential

Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended.

Body protection

See Other protection below

Other protection

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

Respiratory protection

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

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

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

^{* -} Continuous Flow ** - Continuous-flow or positive pressure demand

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

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SECTION 9 Physical and chemical properties

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Information on basic physical and chemical properties

Appearance	Clear yellowish liquid with solvent odour; mixes with water.		
Physical state	Liquid	Relative density (Water = 1)	~1.2
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Applicable	Decomposition temperature (°C)	>320
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	>61	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	Combustible.	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)	34
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Miscible	pH as a solution (1%)	Not Applicable
Vapour density (Air = 1)	>1	VOC g/L	340

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

Inhaled	Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination
Ingestion	Accidental ingestion of the material may be harmful; animal experiments indicate that ingestion of less than 150 gram may be fatal or may produce serious damage to the health of the individual. Aromatase inhibitors (including triazoles and azoles) produce several side effects including mood swing, depression, weight gain, hot flushes, vaginal dryness, bloating, early onset of menopause. Long-term use may result in bone weakness, increased risk of blood clots, gastrointestinal disturbance, and sweats. Aromatase inhibitors lower the level of oestrogen in post-menopausal women who have hormone-receptor-positive breast cancers. Prior to menopause oestrogen is mostly produced in the ovaries. Post-menopausal women produce oestrogen from another hormone, androgen. Aromatase inhibitors prevent the enzyme, aromatase from catalysing this reaction. Breast cancer cell growth in post-menopausal women is stimulated by oestrogen.
Skin Contact	Evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. The material may accentuate any pre-existing skin condition

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Eye

Evidence exists, or practical experience predicts, that the material may cause eye irritation in a substantial number of individuals and/or may produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals.

Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.

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

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 hyperresponsive.

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.

Azole antifungals cause hepatotoxicity by inducing the expression of liver cytochrome P450 enzymes (CYP1, CYP2, and CYP3 families), which in turn increases the abundance of reactive oxygen species in liver cells, resulting in lipid peroxidation and DNA damage. In addition, azole antifungals have the potential to inhibit liver P450 enzymes, interfering in the phase I metabolism of xenohiotics

Disruption of the endocrine system, by azoles, can lead to impaired reproduction, alterations in sexual differentiation, impaired growth and development, and the formation of hormone-dependent cancers Azole antifungals disrupt the endocrine system by inhibiting several highly substrate-selective cytochrome P450 enzymes involved in mammalian steroid hormone biosynthesis. These include aromatase (CYP19) (which catalyzes the C-10 demethylation of androgens to estrogens), CYP11A (which converts cholesterol to pregnenolone), steroid 21-hydroxylase (CYP21), aldosterone synthase (CYP11B2), steroid 11beta-hydroxylase (CYP11B1), steroid 17alpha-hydroxylase/17,20-lyase (CYP17), and lanosterol 14alpha-demethylase (CYP51), disturbing the in vivo hormonal balance.

Azole fungicides show a broad antifungal activity and are used either to prevent fungal infections or to cure an infection. Therefore, they are important tools in integrated agricultural production. According to their chemical structure, azole compounds are classified into triazoles and imidazoles; however, their antifungal activity is due to the same molecular mechanism. The difference between the imidazoles and the triazoles involves the mechanism of inhibition of the cytochrome P450 enzyme. The N3 of the imidazole compound binds to the heme iron atom of ferric cytochrome P450, whereas the N4 of the triazoles bind to the heme group. The triazoles have been shown to have a higher specificity for the cytochrome P450 than imidazoles, thereby making them more potent than the imidazoles

Chronic

The cell membrane assembly of fungi and yeast is disturbed by blocking the synthesis of the essential membrane component ergosterol. This fundamental biochemical mechanism is the basis for the use of azole fungicides in agriculture and in human and veterinary antimycotic therapies. The enzyme involved is sterol 14[alpha]-demethylase, which is found in several phyla. In mammals, it converts lanosterol into the meiosis-activating sterols (MAS) which regulate or modify cell division. These precursors of cholesterol have been discovered to moderate the development of male and female germ (sexual) cells. Several metabolites of lanosterol have been regarded only as precursors of cholesterol without any biological function in animals. This view dramatically changed recently with the observation that FF-MAS isolated from human follicle fluid and T-MAS isolated from bull testis as well as the MAS-412 and MAS-414 induced resumption of meiosis in cultivated mouse oocytes Aromatase is another target enzyme of azole compounds. In steroidogenesis, it converts androgens into the corresponding oestrogens. The importance of androgens and oestrogens for the development of reproductive organs, for fertility, and in certain sex steroid-dependent diseases is well known. Therefore, azole compounds can be directed against aromatase to treat oestrogen-responsive diseases. Based on the inhibitory activity of azoles on key enzymes involved in sex steroid hormone synthesis, it is likely that effects on fertility, sexual behavior, and reproductive organ development will occur depending on dose level and duration of treatment of laboratory animals. Several azole compounds were shown to inhibit the aromatase and to disturb the balance of androgens and estrogens in vivo. In fact, the clinical use of azole compounds in estrogen-dependent diseases is based on this effect. Additionally, azole antifungals developed to inhibit the sterol 14[alpha]-demethylase of fungi and yeast in agriculture and medicine are also inhibiting aromatase. Therefore, these antifungals may unintentionally disturb the balance of androgens and estrogens. Until now, it is not clear whether this effect is compensated by an increased expression of aromatase or by other unknown mechanisms.

The broad use of biologically active compounds in human therapy as well as in nonhuman applications may involve some risks, as exemplified by emerging antibiotic resistance. In agriculture, fungi and yeast are well known to develop resistance to azoles, and some molecular mechanisms of resistance development have been described. The significance of the agricultural azole resistance for human clinical antimycotic therapies has been discussed in Europe, but is not clarified yet. The actual target enzyme of azole antifungals, the fungal sterol 14[alpha]-demethylase, is expressed in many species including humans, and it is highly conserved through evolution. Hence, it seems reasonable to assume that most of the azole antifungals used in agriculture and medicine as well as azoles used in management of breast cancer also act as inhibitors on human sterol 14[alpha]-demethylase to an unknown extent. The toxicologic profiles of individual azole fungicides provide evidence for endocrine effects. In fact, many of these fungicides have effects on prostate, testis, uterus, and ovaries as well as on fertility, development, and sexual behavior. The current database does not allow us to establish causal relationships of these effects with inhibition of sterol 14[alpha]-demethylase and/or aromatase, but the overall view strongly suggests a connection with disturbed steroidogenesis. Zam et al: Environmental Health Perspectives-3/1/2003

Some azoles have been associated with prolongation of the QT interval on the electrocardiogram.

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Azole fungicides are broad spectrum antifungal compounds used in agriculture and in human and veterinary medicine. The mechanism of antifungal action relies on inhibition of CYP51 (sterol 14-demethylase), resulting in inhibition of fungal cell growth. Known adverse health effects of azole fungicides are mainly linked to cytochrome P450 (CYP) inhibition. The relatively poor selectivity of the agricultural azoles for the fungal CYP51 over the human homolog raises the concern that exposure to azole fungicide residues might disrupt sterol biosynthesis and other endogenous downstream cytochrome P450 metabolic systems, such as human steroidogenesis and phase I metabolism of xenobiotics in the liver.

Agricultural azoles have the general ability to inhibit the P450 enzymes in steroidogenesis, albeit with different potencies. Since the introduction of large-scale use of azole antifungals increasing evidence of hepatotoxicity and associated hepatic tumors has been reported with liver tissue concentrations of ketoconazole and itraconazole being reported as 22- and 10-fold higher, respectively, than plasma levels indicating azole toxicity in the liver being more acute than in other tissues.

Additionally, azole fungicide-induced neurotoxicity has been reported. Several azole fungicides induced a nonspecific inhibition of voltage-gated calcium channels (VGCCs), each with varying potency. These findings demonstrate modulation of intracellular calcium ion via inhibition of VGCCs as a novel mode of action of azole fungicides Calcium plays a pivotal role in neuronal survival and functioning.

Triazoles are important antifungal compounds, and all drugs in this class inhibit ergosterol synthesis by blocking the 14-alphademethylase enzyme, resulting in the accumulation of toxic methylsterols that may culminate in fungal death.

Triazole pesticides all contain a triazole ring with nitrogen atoms at the 1,2 and 4 positions. 1,2,4-Triazole (1,2,4-T) and its conjugates, triazole alanine (TA), triazole acetic acid (TAA), triazole pyruvic acid, and triazole lactic acid are the metabolic products of plant, fungal and animal bioconversion. These compounds all possess potentially significant toxicological properties. Following application of a triazole-derivative fungicide, biological and/or chemical processes may cause the triazole ring to be released from the parent compound. In rats and livestock, 1,2,4-triazole is relatively stable and is the terminal form of the triazole ring. In plants, the 1,2,4-triazole molecule may become conjugated to serine. The resulting compound, triazole alanine, may be oxidised to form triazole acetic acid. Triazole alanine and triazole acetic acid are the primary terminal forms of the triazole ring in plants, though some 1,2,4-triazole may remain. The degree of formation of any given form of the triazole ring is highly dependent on the nature and properties of the parent compound. Although other triazole conjugates such as triazole lactic acid and triazole pyruvate have been observed in plant metabolism studies, TA and TAA are the predominant conjugates that need to be included in the dietary risk assessment.

Although for most pesticides, mammals convert only a small proportion to free triazole (less than 25%), two compounds (tetraconazole and flusilazole) demonstrate relatively high conversion (68-77%) in rat metabolism studies.

Available acute data indicate that 1,2,4-triazole is slightly toxic by the oral route (with oral LD50 values ranging from 666 mg/kg in rabbits to 3650 mg/kg in mice) and slightly to moderately toxic by the dermal route (dermal LD50s were less than 2000 mg/kg in rabbits, and 3000-4000 mg/kg in rats). Limited available information indicates that 1,2,4-triazole is slightly irritating or non-irritating to the skin, but severely irritating to the eye. Based on the limited acute toxicity data, as well as the available developmental toxicity data (see below), it appears that rabbits may be substantially more susceptible to 1,2,4-triazole than are rats or mice.

Studies indicate that 1,2,4-triazole affects the central and peripheral nervous systems, reproductive tissues of both sexes, and the hematological system. Developmental and reproductive effects have been noted for this compound. Based on the available metabolism data from rats and livestock, 1,2,4-triazole may form in humans following exposure to parent triazole compounds. Relative to triazole alanine, fewer studies are available depicting the toxicological effects of the other triazole conjugates. It is assumed that the triazole conjugates are all toxicologically equivalent to triazole alanine. The available studies found developmental skeletal effects, decreased body weight and body weight gain, and decreased leukocytes and triglycerides. A number of target organs and critical effects have been identified. 1,2,4-triazole targets the nervous system, both central and peripheral, as brain lesions (most notably in the cerebellum) were seen in both rats and mice, and peripheral nerve degeneration was also seen in the subchronic neurotoxicity study in rats. In addition, brain weight decreases were seen in several studies, including in the offspring in the reproductive toxicity study. In the subchronic/neurotoxicity study, there is evidence that effects progress over time, with an increase in incidence of clinical signs (including tremors and muscle fasciculations) during weeks 8 and 13 that were not seen during earlier evaluations.

There is no evidence that exposure to triazole alanine results in neurotoxicity. No clinical signs of neurotoxicity, changes in brain weights, changes in brain gross or microscopic pathology, or any other neurotoxic effects were observed in the short-term rat studies, the subchronic rat and dog feeding studies, the rat developmental toxicity study, or the two-generation reproduction study.

Effects were also seen on reproductive organs in both sexes, most notably ovaries (in rats) and testes (in rats and mice), in both the reproductive toxicity and subchronic toxicity studies. Hematological changes, including slightly decreased hemoglobin and/or hematocrit, have also been seen in multiple studies and species (in rats at doses of 33 mg/kg/day and above, and in mice at doses of 487 mg/kg/day and above).

1.2.4-triazole also causes developmental toxicity in both rats and rabbits, including malformations, at doses similar to those inducing maternal toxicity (decreased body weight gain in rats and clinical signs and mortality in rabbits). Developmental toxicity was also seen in the reproductive toxicity study, with offspring showing adverse effects on multiple endpoints (including decreased brain and body weight) at doses lower than those at which effects were seen in parents. In addition, reproductive toxicity was seen in both sexes: at the highest dose (3000 ppm), only two F1 litters (one pup/litter) were produced, and neither

Triazole alanine showed increased incidences of skeletal findings in the offspring at the mid and high doses, while no treatmentrelated effects were seen in the dams up to the limit dose. The skeletal findings included unossified odontoid processes at 300 and 1000 mg/kg/day, with partially ossified transverse processes of the 7th cervical vertebra (bilateral), unossified 5th sternebra. and partially ossified 13th thoracic centrum observed only at 1000 mg/kg/day.

Available mutagenicity data are limited but negative. A large number of parent triazole-derivative pesticides have been classified as carcinogens (most also non-mutagenic), but the relevance of that finding to expected effects of free triazole may be limited. The types of tumors associated with exposure to the parent chemicals are most commonly hepatocellular adenomas/carcinomas in mice. Other tumor types vary considerably (including liver tumors, thyroid tumors, ovarian tumors, testicular tumors, and bladder tumors). None of the tumor types are clearly associated with the proportion of free triazole formed in available rat metabolism studies. Evidence indicates that the parent triazole compounds appear to result in a tumor response subsequent to perturbation of liver metabolism, specifically xenobiotic and fatty acid metabolic pathways. In addition the thyroid response

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appears to be secondary to perturbation of thyroid homeostasis. Thus, the conazoles appear to drive a tumor response secondary to epigenetic effects and not from direct interaction with the DNA. An epigenetic mode of action would be consistent with a nonlinear process.

AC Pacer 550EC Fungicide	TOXICITY	IRRITATION	
	Not Available	Not Available	
propiconazole	TOXICITY	IRRITATION	
	dermal (rat) LD50: >4000 mg/kg ^[2]	Eye (non-irritating) *	
	Inhalation(Rat) LC50: >5.8 mg/L4h ^[2]	Skin (non-irritating) *	
	Oral (Rat) LD50: 550 mg/kg ^[1]		
liquid hydrocarbons	TOXICITY	IRRITATION	
	Not Available	Not Available	

Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

PROPICONAZOLE

No sensitisation in guinea pigs * ADI 0.04 mg/kg b.w. * Toxicity Class WHO III NOEL for dogs 50 ppm (1.9 mg/kg b.w. daily) * 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.

[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]

For olefins:

Studies have shown that normal alpha olefins have little or no toxic effect on animals except in very severe inhalation conditions and that they may produce minimal skin and eye irritation, but are not skin sensitisers. Laboratory exposures to very high airborne concentrations of C6-C16 normal alpha olefin vapors or mists produced central nervous system effects including anesthesia. If C20+ products are heated, fumes may produce nausea and irritation of the upper respiratory tract. Although not all products have been tested in genetic toxicity assays, the available data indicate normal alpha olefins are not

Acute toxicity: The weight of evidence indicates alpha and internal olefins with carbon numbers between C6 and C54 have a similar and low level of mammalian toxicity, and the toxicity profile is not affected by changes in the location of the double bond or the addition of branching to the structure. These materials are not eye irritants or skin sensitisers. Prolonged exposure of the skin for many hours may cause skin irritation.

Olefins (alkenes) ranging in carbon number from C6 to C24 alpha (linear) and internal (linear and branched), and C24-54 alpha (linear and branched) demonstrate low acute toxicity by the oral, inhalation and dermal routes of exposure: Rat oral LD50 >5 g/kg; rat 4-hr inhalation LC50 range = 110 mg/L (32,000 ppm) to 6.4 mg/L (693 ppm) for C6 to C16; and rat/rabbit dermal LD50 > highest doses tested (1.43-10 g/kg).

LIQUID HYDROCARBONS

Repeated dose toxicity: Studies, using the inhalation (C6 alpha), dermal (C12-16 alpha), or oral (C6 alpha and internal linear/branched; C8 and C14 alpha; and C16/18, C18 and C20-24 internal linear/branched) routes of exposure, have shown comparable levels of low toxicity in rats. In females, alterations in body and organ weights, changes in certain clinical chemistry/haematology values, and liver effects were noted (NOELs of >= 100 mg/kg oral or >= 3.44 mg/kg [1000 ppm] inhalation). In males, alterations in organ weights, changes in certain clinical chemistry/hematology values, liver effects, and kidney damage were noted (LOELs > =100 mg/kg oral only). The male rat kidney damage suggests alpha2u,- globulin nephropathy, a male rat specific effect that is not considered relevant to human health. The noted liver effects were seen in oral studies with C14 alpha olefins (minimal-to-mild hepatocyte cytoplasmic vacuolation with increased liver weight in males and females) and with C20-24 internal olefins (minimal centrilobular hepatocyte hypertrophy with increased liver weight in females only). No effects were present in the study with C20-24 internal olefins following a 4-week recovery period, indicating reversibility of the observed effects. These liver effects seen only with the larger molecules may be indirect effects of an intensified liver burden, rather than a direct toxic effect of the olefin. Based on evidence from neurotoxicity screens included in repeated dose studies with C6 and C14 alpha olefins and with C6, C16/18 and C20-24 internal linear/branched olefins, the category members

Reproductive/ developmental toxicity: Based on evidence from reproductive/developmental toxicity screens in rats with C6 and C14 alpha olefins and C6 and C18 linear/branched internal olefins, along with the findings of no biologically significant effects on male or female reproductive organs in repeated dose toxicity studies, olefins are not expected to cause reproductive or developmental toxicity

Genotoxicity: Based on the weight of evidence from studies with alpha and internal olefins, category members are not

Carcinogenicity: No carcinogenicity tests have been conducted on C6-54 alpha or internal olefins; however, there are no structural alerts indicating a potential for carcinogenicity in humans.

No significant acute toxicological data identified in literature search.

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

Legend:

★ - Data either not available or does not fill the criteria for classification

Data available to make classification

SECTION 12 Ecological information

Toxicity

	Endpoint	Test Duration (hr)	Species		Value	Source
AC Pacer 550EC Fungicide	Not Available	Not Available	Not Available		Not Available	Not Available
	Endpoint	Test Duration (hr)	Species	Value	•	Source
	EC50	48h	Crustacea	3.354	-4.902mg/L	4
	EC50	96h	Algae or other aquatic plants	1.29mg/l		4
propiconazole	NOEC(ECx)	48h	Fish	0.000	1mg/l	4
	EC50	72h	Algae or other aquatic plants	Algae or other aquatic plants 0.0008mg/l		4
	LC50	96h	Fish	Fish 5.3mg/l		Not Available
	Endpoint	Test Duration (hr)	Species		Value	Source
liquid hydrocarbons	Not Available	Not Available	Not Available		Not Available	Not Available
Legend:	4. US EPA, Ed	otox database - Aquatic Toxicity	ope ECHA Registered Substances - Ecotoxio Data 5. ECETOC Aquatic Hazard Assessm oncentration Data 8. Vendor 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.

For azole-containing substances.

Azole fungicides and systemically used antifungal drugs directly interfere with steroidogenesis by acting as potent inhibitors of steroidogenic enzymes and are known to cause endocrine disruption mainly via this mechanism.

An important P450 enzyme involved in the steroidogenesis is aromatase. Aromatase demethylates C10 and specifically converts androstenedione and testosterone. On the protein level, the amino acid sequence homology between aromatase from fish and humans is about 50% and between rats and humans is about 78%. In mammals, aromatase is mainly expressed in the brain and the gonads, but it is also found in placental, adipose, and bone tissue. The physiologic balance between different sex steroid hormones is crucial for the development, maintenance, and function of the reproductive system as well as for the differentiation of the sexual phenotype during ontogeny. Oestrogens (estrone and estradiol) are products of the androgens (androstenedione and testosterone), and the reaction is catalysed by aromatase. In mammals, differentiation of the male phenotype depends not only on testosterone but also on estradiol generated from testosterone by neuronal aromatase in central nervous system. Therefore, disturbances in aromatase expression and/or changes in its catalytic activity are expected to exhibit negative effects on reproduction parameters

Azole-containing compounds produce profound effects in the environment. In part this is due to inhibition of several enzyme systems including those involving sterol 14[alpha]-demethylase. Sterol 14[alpha]-demethylase is a member of the superfamily of haeme-containing cytochrome P450 enzymes involved in metabolism of endogenous and xenobiotic substances. The antifungal effect of azoles is due to inhibition of sterol 14[alpha]-demethylase in fungi and yeast, thereby blocking the biosynthesis of ergosterol The subsequent lack of ergosterol is detrimental because ergosterol is an essential sterol component in the membranes of fungi and yeast. Sterol 14[alpha]-demethylase is not only expressed in fungi and yeast but is also found in many other species ranging from bacteria to mammals. In plants, the sterol 14[alpha]-demethylase reaction metabolises obtusifoliol and provides precursors for biosynthesis of phytosterols. In animals, the sterol 14[alpha]-demethylase reaction is part of the metabolic pathway leading to biosynthesis of cholesterol. Cholesterol in turn is the substrate for the production of many other sterols (e.g., the sex steroid hormones).

The DNA sequences encoding sterol 14[alpha]-demethylase of many fungi and yeast are known, as well as the sequences of mice, rats, pigs, and humans. On the protein level, the amino acid sequences are highly conserved along the phylogenetic tree. This fact is considered by many authors as an indication of the pivotal role of sterol 14[alpha]-demethylase in all organisms. The homology of the amino acid sequence level between rats and humans is 93% and 40% between fungi and humans. In humans, the sterol 14[alpha]-demethylase is expressed in many different tissues

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Triazole fungicides share common metabolites, the triazole compounds 1,2,4-triazole (free triazole), triazole alanine, and triazole acetic acid. In environmental fate studies, all three forms of triazole (1,2,4-T, TA, and TAA) have been found and there is evidence that the three can inter-convert in soil and aquatic systems As a plant metabolite, and given the wide use of triazole-derivative pesticides (used as fungicides on many crops as well as on turf) free triazole is found in a variety of food commodities, including animal byproducts. 1,2,4-triazole appears to be relatively stable in the environment, and may be found in rotational crops as well as in water

Studies indicate that 1,2,4-triazole affects the central and peripheral nervous systems, reproductive tissues of both sexes, and the hematological system. Developmental and reproductive effects have been noted for this compound. Based on the available metabolism data from rats and livestock, 1,2,4-triazole may form in humans following exposure to parent triazole compounds.

The available studies on triazole conjugates found developmental skeletal effects, decreased body weight and body weight gain, and decreased leukocytes and triglycerides following exposure in animals.

DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Ingredient	Bioaccumulation	
	No Data available for all ingredients	

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging

disposal

- ▶ 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



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Marine Pollutant



HAZCHEM

:M •3Z

Land transport (ADG)

14.1. UN number or ID number	3082		
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains propiconazole)		
14.3. Transport hazard class(es)	Class Subsidiary Hazard	9 Not Applicable	
14.4. Packing group	III		
14.5. Environmental hazard	Environmentally hazardous		
14.6. Special precautions for user	Special provisions 274 331 335 375 AU01 Limited quantity 5 L		

Environmentally Hazardous Substances meeting the descriptions of UN 3077 or UN 3082 are not subject to this Code when transported by road or rail in;

- (a) packagings;
- (b) IBCs; or
- (c) any other receptacle not exceeding 500 kg(L).
- Australian Special Provisions (SP AU01) ADG Code 7th Ed.

Air transport (ICAO-IATA / DGR)

14.1. UN number	3082		
14.2. UN proper shipping name	Environmentally hazardous substance, liquid, n.o.s. (contains propiconazole)		
	ICAO/IATA Class	9	
14.3. Transport hazard class(es)	ICAO / IATA Subsidiary Hazard	Not Applicable	
olass(cs)	ERG Code	9L	
14.4. Packing group	III		
14.5. Environmental hazard	Environmentally hazardous		
	Special provisions		A97 A158 A197 A215
	Cargo Only Packing Instructions		964
	Cargo Only Maximum Qty / Pack		450 L
14.6. Special precautions for user	Passenger and Cargo Packing Instructions		964
	Passenger and Cargo Maximum Qty / Pack		450 L
	Passenger and Cargo Limited Quantity Packing Instructions		Y964
	Passenger and Cargo Limited Ma	aximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. UN number	3082		
14.2. UN proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains propiconazole)		
14.3. Transport hazard class(es)	IMDG Class IMDG Subsidiary Hazard	9 Not Applicable	
14.4. Packing group	III		
14.5 Environmental hazard	Marine Pollutant		

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14.6. Special precautions for user

EMS Number	F-A, S-F	
Special provisions	274 335 969	
Limited Quantities	5 L	

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
propiconazole	Not Available
liquid hydrocarbons	Not Available

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
propiconazole	Not Available
liquid hydrocarbons	Not Available

SECTION 15 Regulatory information

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

propiconazole is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5

Australian Inventory of Industrial Chemicals (AIIC)

liquid hydrocarbons is found on the following regulatory lists

Not Applicable

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status	
Australia - AIIC / Australia Non-Industrial Use	Yes	
Canada - DSL	No (propiconazole)	
Canada - NDSL	No (propiconazole)	
China - IECSC	Yes	
Europe - EINEC / ELINCS / NLP	Yes	
Japan - ENCS	Yes	
Korea - KECI	Yes	
New Zealand - NZIoC	Yes	
Philippines - PICCS	Yes	
USA - TSCA	No (propiconazole)	
Taiwan - TCSI	Yes	
Mexico - INSQ	Yes	
Vietnam - NCI	Yes	
Russia - FBEPH	No (propiconazole)	
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.	

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SECTION 16 Other information

Revision Date	27/10/2023
Initial Date	23/08/2017

SDS Version Summary

Version	Date of Update	Sections Updated
5.1	20/08/2021	Classification change due to full database hazard calculation/update.
6.1	27/10/2023	UN Number update

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
- ▶ BCF: BioConcentration Factors
- ▶ BEI: Biological Exposure Index
- ▶ DNEL: Derived No-Effect Level
- ▶ PNEC: Predicted no-effect concentration
- ▶ 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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