Quantum ProTeb 420 SC Fungicide

Quantum Agrosciences Holdings Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **7998-70**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **15/01/2026**
Revision Date: **15/01/2026**
Print Date: **15/01/2026**
S.GHS.AUS.EN.E

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

Product Identifier

Product name	Quantum ProTeb 420 SC Fungicide
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains tebuconazole and prothioconazole)
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	Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

Registered company name	**Quantum Agrosciences Holdings Pty Ltd**
Address	Suite 2, Level 7, 330 Collins Street, Melbourne, Victoria 3000 Australia
Telephone	1300 658 988
Fax	Not Available
Website	www.quantumag.au
Email	vincent@quantumag.au

Emergency telephone number

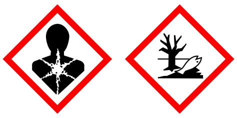
Association / Organisation	**CHEMWATCH EMERGENCY RESPONSE (24/7)**
Emergency telephone number(s)	+61 1800 951 288 (ID#: 7998-70)
Other emergency telephone number(s)	+61 3 9573 3188

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S5
Classification [1]	Reproductive Toxicity Category 1B, 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)

H360Fd	May damage fertility. Suspected of damaging the unborn child.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P202	Do not handle until all safety precautions have been read and understood.
P280	Wear protective gloves and protective clothing.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P308+P313	IF exposed or concerned: Get medical advice/ attention.
P391	Collect spillage.

Precautionary statement(s) Storage

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
178928-70-6	10-30	prothioconazole
107534-96-3	10-30	tebuconazole
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: - 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: - Flush skin and hair with running water (and soap if available). - Seek medical attention in event of irritation.
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.
Ingestion	- **IF SWALLOWED, REFER FOR MEDICAL ATTENTION, WHERE POSSIBLE, WITHOUT DELAY.** - For advice, contact a Poisons Information Centre or a doctor. - Urgent hospital treatment is likely to be needed. - In the mean time, qualified first-aid personnel should treat the patient following observation and employing supportive measures as indicated by the patient's condition. - If the services of a medical officer or medical doctor are readily available, the patient should be placed in his/her care and a copy of the SDS should be provided. Further action will be the responsibility of the medical specialist. - If medical attention is not available on the worksite or surroundings send the patient to a hospital together with a copy of the SDS. **Where medical attention is not immediately available or where the patient is more than 15 minutes from a hospital or unless instructed otherwise:** - **INDUCE** vomiting with fingers down the back of the throat, **ONLY IF CONSCIOUS**. Lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. **NOTE:** Wear a protective glove when inducing vomiting by mechanical means.

Indication of any immediate medical attention and special treatment needed

Due to their role in detoxifying potentially toxigenic xenobiotics and widespread nature throughout numerous organ systems, CYP enzymes are subject to a myriad of potential reactions and serve as a backbone in clinical research. The various effects of medications and other compounds on CYP enzymes are key in drug development to determine their safety and efficacy in the general public. Certain drugs are known inhibitors and inducers of specific CYP enzymes and require careful monitoring in patients taking multiple agents metabolized by the same subfamily. Two isozymes, CYP3A4 and CYP2D6, make up the bulk of drug metabolism, and drugs that interact with these enzymes should, therefore, merit closer evaluation and monitoring.[
Most medications can still be administered despite this issue, barring any potential comorbidities, such as cirrhosis or viral hepatitis, that can alter the baseline activity of these enzymes. This is due to their intrinsic capacity to catalyze multiple substrates simultaneously at different sites. However, if patients develop any signs of significant dysfunction, it is essential to look at a detailed patient history, including medications and a review of associated adverse effects. In cases of drug toxicity due to CYP inhibition, presenting symptoms would display signs of overdose. These instances can typically be treated by withholding the causative agent until plasma levels of the drug stabilize, or in more urgent scenarios, antidotes may work for rapid reversal. In cases of treatment failure due to CYP induction, treatment goals would not be adequately met and would warrant an adjustment in medication dosage or change in medication
Treat symptomatically.
For employees potentially exposed to antineoplastic and/ or cytotoxic agents on a regular basis, a preplacement physical examination and history (noting risk factors) is recommended. Periodic follow-up examinations should also be undertaken and should be overseen by a physician familiar with the toxic effects of the substance and full details of the nature of work undertaken by the employee.
Following administration of antineoplastics, control of nausea and vomiting may be attempted by giving phenothiazines such as perphenazine, prochlorperazine, promethazine or thiethylperazine. In bone-marrow depression, transfusion of blood or platelets reduces the risk of life-threatening haemorrhage. Granulocyte transfusions and injection of antibiotics may be necessary to combat infection in the neutropenic patient. Hyperuricaemia is avoided by the addition of allopurinol to treatment schedules and measures such as alkalisation of the urine and hydration may be adopted. MARTINDALE: The Extra Pharmacopoeia, 28th Edition.
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

- Establish a patent airway with suction where necessary.

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- 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

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 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 water delivered as a fine spray to control fire and cool adjacent area. ‣ Avoid spraying water onto liquid pools. ‣ **DO NOT** approach containers suspected to be hot. ‣ Cool fire exposed containers with water spray from a protected location. ‣ If safe to do so, remove containers from path of fire.
Fire/Explosion Hazard	‣ 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) hydrogen chloride phosgene nitrogen oxides (NOx) sulfur oxides (SOx) ‣ other pyrolysis products typical of burning organic material.
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

Minor Spills	Environmental hazard - contain spillage. It is recommended that areas handling final finished product have cytotoxic spill kits available. Spill kits should include: ‣ impermeable body covering, ‣ shoe covers, ‣ latex and utility latex gloves, ‣ goggles, ‣ approved respirator (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent), see Section 8, ‣ disposable dust pan and scoop, ‣ absorbent towels, ‣ spill control pillows, ‣ disposable sponges, ‣ sharps container, ‣ disposable garbage bag and ‣ hazardous waste label Where spills are treated with loose absorbents, such as vermiculite, ensure dust exposure is strictly avoided. To avoid accidental exposure due to waste handling of cytotoxics: ‣ Place waste residue in a segregated sealed plastic container. ‣ Used syringes, needles and sharps should not be crushed, clipped, recapped, but placed directly into an approved sharps container.

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	▸ Dispose of any cleanup materials and waste residue according to all applicable laws and regulations e.g, secure chemical landfill disposal. All personnel likely to involved in a antineoplastic (cytotoxic) spill must receive practical training in: ▸ the correct procedures for handling cytotoxic drugs or waste in order to prevent and minimise the risk of spills ▸ the location of the spill kit in the area ▸ the arrangements for medical treatment of any affected personnel ▸ the procedure for containment of the spill, and decontamination of personnel and the environment, including the different procedures for major and minor spills ▸ the procedure for waste disposal according to the nature and extent of the spill ▸ 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	▸ Clean up all spills immediately. ▸ Avoid breathing dust and contact with skin and eyes. ▸ Wear protective clothing, gloves, safety glasses and dust respirator. ▸ Wet residue with water to prevent dusting ▸ Sweep up, shovel up or ▸ Vacuum up (consider explosion-proof machines designed to be grounded during storage and use). ▸ Place spilled material in clean, dry, sealable, labelled container. 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. ▸ 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

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. ▸ Avoid smoking, naked lights or ignition sources. ▸ Avoid contact with incompatible materials. ▸ When handling, **DO NOT** eat, drink or smoke. ▸ Keep containers securely sealed when not in use. ▸ Avoid physical damage to containers. ▸ Always wash hands with soap and water after handling. ▸ Work clothes should be laundered separately. ▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS. ▸ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ No smoking, naked lights or ignition sources. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Glass container is suitable for laboratory quantities ▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	High nitrogen compounds are often unstable or explosive; the tendency is exaggerated by attachment of azide or diazonium groups, or a high-nitrogen heterocyclic nucleus. High-nitrogen chemical families include ▸ azides ▸ diazoazoles ▸ diazonium salts ▸ hydrazinium salts ▸ N-nitro compounds ▸ tetrazoles ▸ tetrazines ▸ triazenes ▸ triazoles ▸ Avoid reaction with oxidising agents

SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

Exposure controls

Appropriate engineering controls	**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. ▸ 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:** **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. **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.
Individual protection measures, such as personal protective equipment	
Eye and face protection	▸ Chemical protective goggles with full seal. [AS/NZS 1337.1, EN166 or national equivalent] ▸ Shielded mask (gas-type). ▸ 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
Hands/feet protection	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.

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	Personal hygiene is a key element of effective hand care. Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. Suitability and durability of glove type is dependent on usage. Important factors in the selection of gloves include: · frequency and duration of contact, · chemical resistance of glove material, · glove thickness and · dexterity Select gloves tested to a relevant standard (e.g. Europe EN 374, US F739, AS/NZS 2161.1 or national equivalent). · When prolonged or frequently repeated contact may occur, a glove with a protection class of 5 or higher (breakthrough time greater than 240 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · When only brief contact is expected, a glove with a protection class of 3 or higher (breakthrough time greater than 60 minutes according to EN 374, AS/NZS 2161.10.1 or national equivalent) is recommended. · Some glove polymer types are less affected by movement and this should be taken into account when considering gloves for long-term use. · Contaminated gloves should be replaced. As defined in ASTM F-739-96 in any application, gloves are rated as: · Excellent when breakthrough time > 480 min · Good when breakthrough time > 20 min · Fair when breakthrough time < 20 min · Poor when glove material degrades For general applications, gloves with a thickness typically greater than 0.35 mm, are recommended. It should be emphasised that glove thickness is not necessarily a good predictor of glove resistance to a specific chemical, as the permeation efficiency of the glove will be dependent on the exact composition of the glove material. Therefore, glove selection should also be based on consideration of the task requirements and knowledge of breakthrough times. Glove thickness may also vary depending on the glove manufacturer, the glove type and the glove model. Therefore, the manufacturers technical data should always be taken into account to ensure selection of the most appropriate glove for the task. Note: Depending on the activity being conducted, gloves of varying thickness may be required for specific tasks. For example: · Thinner gloves (down to 0.1 mm or less) may be required where a high degree of manual dexterity is needed. However, these gloves are only likely to give short duration protection and would normally be just for single use applications, then disposed of. · Thicker gloves (up to 3 mm or more) may be required where there is a mechanical (as well as a chemical) risk i.e. where there is abrasion or puncture potential Gloves must only be worn on clean hands. After using gloves, hands should be washed and dried thoroughly. Application of a non-perfumed moisturiser is recommended. ‣ 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.
Body protection	See Other protection below
Other protection	‣ When handling antineoplastic materials, it is recommended that a disposable work-uniform (such as Tyvek or closed front surgical-type gown with knit cuffs) is worn. ‣ Potentially contaminated bodily fluids should be handled in accordance with local standards or codes of practice (appendix 10 of 'Cytotoxic Drugs and Related Waste' - Workcover New South Wales, HSE Information Sheet MISC615, OSHA Technical Manual (OTM) Section VI: Chapter 2) ‣ 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

Type A 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	-
up to 50	1000	-	A-AUS / Class 1
up to 50	5000	Airline *	-
up to 100	5000	-	A-2
up to 100	10000	-	A-3
100+			Airline**

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

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

SECTION 9 Physical and chemical properties

Information on basic physical and chemical properties

Appearance	Off-white liquid with light characteristic odour; dispersibles in water.

Physical state	Liquid	**Relative density (Water = 1)**	~1.08 @ 20 C

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Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	5-8	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	~0 (freezing point)	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	~100	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Available	**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	Based on available data, the classification criteria are not met.
b) Skin Irritation/Corrosion	Based on available data, the classification criteria are not met.
c) Serious Eye Damage/Irritation	Based on available data, the classification criteria are not met.
d) Respiratory or Skin sensitisation	Based on available data, the classification criteria are not met.
e) Mutagenicity	Based on available data, the classification criteria are not met.
f) Carcinogenicity	Based on available data, the classification criteria are not met.
g) Reproductivity	There is sufficient evidence to classify this material as toxic to reproductivity
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	Based on available data, the classification criteria are not met.
j) Aspiration Hazard	Based on available data, the classification criteria are not met.

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.
Ingestion	Accidental ingestion of the material may be damaging to the health of the individual.
Skin Contact	Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream, through, for example, cuts, abrasions 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	There is some evidence to suggest that this material can cause eye irritation and damage in some persons.
Chronic	Ample evidence exists from experimentation that reduced human fertility is directly caused by exposure to the material. Ample evidence exists, from results in experimentation, that developmental disorders are directly caused by human exposure to the material. Based on experience with animal studies, exposure to the material may result in toxic effects to the development of the foetus, at levels which do not cause significant toxic effects to the mother. Anti-cancer drugs used for chemotherapy can depress the bone marrow with reduction in the number of white blood cells and platelets and bleeding. Susceptibility to infections and bleeding is increased, which can be life- threatening. Triazole pesticides are the products of plant, fungal and animal bioconversion. They are toxic and are metabolised into variable products depending on the nature of the parent compound. Studies done with animals showed that they may be slightly irritating to the skin, but severely irritating to the eye. They affect the nervous, reproductive and blood systems, and have been shown to developmental toxicity. Limited evidence predicts that they are not likely to cause genetic damage but may cause cancers especially of the liver and thyroid. Azole fungicides show broad antifungal activity, and can be used to prevent or cure fungal infections. They are therefore important in agricultural production.

Continued...

	TOXICITY	IRRITATION
Quantum ProTeb 420 SC Fungicide	Not Available	Not Available

	TOXICITY	IRRITATION
prothioconazole	dermal (rat) LD50: >2000 mg/kg[2]	Not Available
	Oral (Rat) LD50: >6200 mg/kg[2]	

	TOXICITY	IRRITATION
tebuconazole	dermal (rat) LD50: >5000 mg/kg[2]	Not Available
	Inhalation (Rat) LC50: >0.8 mg/L4h[2]	
	Oral (Mouse) LD50; 2000 mg/kg[2]	

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***

PROTHIOCONAZOLE

Prothioconazole has low acute toxicity by oral, dermal, and inhalation routes. It is not a dermal sensitizer, or a skin or eye irritant.
Prothioconazole- desthio also has low acute toxicity by oral, dermal, and inhalation routes. It is not a dermal sensitizer, or a skin irritant, but it is a slight eye irritant. Subchronic studies show that the target organs at the LOAEL include the liver, kidney, urinary bladder, thyroid and blood. Significant clinical chemistry findings were also made. NOAEL/ LOAEL values across the family of chemicals (i.e., prothioconazole, and prothioconazole-desthio and prothioconazole sulfonic acid potassium salt metabolites) in the toxicity database indicate that prothioconazole-desthio is a most toxic chemical. In addition to the target organs and effects observed in the subchronic studies (i.e., liver, kidney, urinary bladder, thyroid, haematology and clinical chemistry), chronic toxicity at the LOAEL also included body weight and food consumption changes, and toxicity to the lymphatic and GI systems. The relative potency of prothioconazole-desthio was greater than prothioconazole.
The acute toxicity of prothioconazole is low, the oral LD50 being > 6200 mg/kg bw in rats. At this dose, there were no deaths and clinical signs were limited to decreased motility and diarrhoea 1-6 h after dosing. The dermal LD50 in rats was > 2000 mg/kg bw and the inhalation LC50, also in rats, was > 4.9 mg/L for a 4-h exposure. Prothioconazole is not irritating to rabbit skin and eyes and is not sensitizing either in the Buehler skin patch test in guinea-pigs or in the local lymph node assay in mice.
Initial studies with repeated doses showed that prothioconazole could be unstable when formulated with diet, hence most studies were performed using dosing by gavage. A 4-week study in rats given prothioconazole by different dosing routes established that plasma concentrations in rats dosed by gavage at 1000 mg/kg bw per day were 3-6-fold those in rats given diets containing prothioconazole at 10000 ppm, equivalent to 1000 mg/kg bw per day, and this was consistent with
the observation of more marked effects in rats dosed by gavage.
The liver was consistently identified as a target organ in short-term studies in rats, mice and dogs, although there were some species differences in the hepatic effects observed. Increased liver weights and increased activities of several liver enzymes were observed in mice, rats (particularly females) and dogs. Microscopic lesions were also observed in the liver, including an increase in pigmented material in dogs, centrilobular fatty change and focal necrosis in mice and cytoplasmic changes and centrilobular hepatocellular hypertrophy in rats and mice. Some of these effects were consistent with induction of hepatic enzymes. None of the effects recorded in the liver persisted after 4- and 8-week recovery periods in rats and dogs, respectively.
The kidney was the primary target organ in dogs and was also identified as a target organ in rats, but not in mice. The effects on the kidneys consisted of increased weights and changes in histology, namely increased incidence and severity of basophilic tubules and tubular dilatation in rats, and interstitial fibrosis and inflammation in dogs. These findings did not persist after a recovery period in rats, but there was only partial recovery in dogs. In rats, these kidney changes correlated with greatly increased water intakes, indicating disturbance of kidney function and systemic water
homeostasis.
The following NOAELs were derived from short-term studies in which prothioconazole was administered orally:
. In a 14 week study in mice dosed by gavage, the NOAEL was 25 mg/kg bw per day on the basis of increased liver weights and various histological changes in the liver at 100 mg/kg bw per day;
. In studies of up to 14 weeks in rats dosed by gavage, the NOAEL was 100 mg/kg bw per day on the basis of increased water consumption, decreased urine output, increased liver weights in females, and histological changes in the liver and kidney at 500 mg/kg bw per day;
. In 13-week and 1-year studies in dogs dosed by gavage, the overall NOAEL was 25 mg/kg bw per day on the basis of minimal histological changes in the kidneys at 40 mg/kg bw per day.
-
In long-term studies in rats and mice dosed by gavage, the primary target organs were the liver and kidney. There was no evidence for any carcinogenic potential in rats or mice. The hepatic effects observed in rats were increased incidences of eosinophilic or clear-cell foci. The other liver effects observed in rats and mice (increased weights, centrilobular hypertrophy with cytoplasmic changes) were consistent with induction of hepatic enzymes. There was slight alteration in the concentrations of plasma thyroid hormones in rats, but there was no associated thyroid histopathology.
-
In long-term studies in rats and mice dosed by gavage, the primary target organs were the liver and kidney. There was no evidence for any carcinogenic potential in rats or mice. The hepatic effects observed in rats were increased incidences of eosinophilic or clear-cell foci. The other liver effects observed in rats and mice (increased weights, centrilobular hypertrophy with cytoplasmic changes) were consistent with induction of hepatic enzymes. There was slight alteration in the concentrations of plasma thyroid hormones in rats, but there was no associated thyroid histopathology.
-
Prothioconazole was toxic to the reproductive system and to developing offspring at a dose that was accompanied by toxicity in parental rats.
The NOAEL for maternal toxicity was 80 mg/kg bw per day on the basis of reduced body-weight gain, increased water consumption, reduced food consumption and clinical chemical indications for functional impairment of liver and kidney function at 750 mg/kg bw per day. The NOAEL for developmental toxicity was 80 mg/kg bw per day on the basis of a statistically significant increase in the incidence of rudimentary supernumerary 14th ribs at 726 mg/kg bw per day.
Prothioconazole is unlikely to cause neurotoxicity in humans
There were no indications of immunotoxicity in general studies of toxicity in dogs, rats and mice.
* APVMA Report

Studies in the rat and mouse, using both prothioconazole and prothioconazole-desthio, showed no evidence of carcinogenicity. The data show that dosing was adequate, except in the rat cancer study using prothioconazole, where the dosing was considered too high. The data indicate that prothioconazole and the three metabolites evaluated (i.e., prothioconazole-desthio, prothioconazole sulfonic acid potassium salt, and prothioconazole-deschloro) variously produce pre-natal developmental effects at levels equal to or below maternally toxic levels. Prothioconazole-desthio is the most toxic orally and dermally, with LOAELs significantly below that of the other chemicals. The rabbit is the more sensitive species. Lastly, prothioconazole-desthio is a developmental neurotoxicant, producing changes in brain morphometrics and increases in the occurrence of peripheral nerve lesions in the neonate. A NOAEL was not determined, since these observations were looked for only at the high dose level. Reproduction studies in the rat, conducted using prothioconazole and prothioconazole-desthio, suggested that these chemicals may not be primary reproductive toxicants. Reproductive and offspring toxicities were observed only in the presence of parental toxicity. Indeed, the parental LOAELs are lower. The data show that prothioconazole- desthio is more toxic by an order of

Continued...

	magnitude. The nature of parental toxicity is similar to what was observed in the subchronic studies, such as body weight and food consumption changes, liver effects, etc. Reproductive effects included decreases in reproductive indices such as those that indicate pup survival and growth. Offspring toxicity was manifested by decreased pup weights and malformations such as cleft palate It is conceivable that the high potency of prothioconazole as an agricultural fungicide is enhanced due to intracellular metabolism of the relatively inactive prothioconazole in the pathogenic fungi to the highly active desthio form and in the host. Treatment of C. albicans cells with prothioconazole, prothioconazole-desthio, and voriconazole resulted in CYP51 inhibition, as evidenced by the accumulation of 14alpha-methylated sterol substrates (lanosterol and eburicol) and the depletion of ergosterol. Inhibitor binding properties of prothioconazole, prothioconazole-desthio, and voriconazole with CaCYP51 were compared. Prothioconazole-desthio and voriconazole bind noncompetitively to CaCYP51 in the expected manner of azole antifungals (with type II inhibitors binding to haeme as the sixth ligand), while prothioconazole binds competitively and does not exhibit classic inhibitor binding spectra. Inhibition of CaCYP51 activity in a cell-free assay demonstrated that prothioconazole-desthio is active, whereas prothioconazole does not inhibit CYP51 activity. Extracts from C. albicans grown in the presence of prothioconazole were found to contain prothioconazole-desthio. leading to the conclusion that the antifungal action of prothioconazole can be attributed to prothioconazole-desthio The desthio metabolite was found almost exclusively in the faeces and represented between 3.5% and 17.7% of the administered dose. The systemic proportion of prothioconazole-desthio was very low; not more than about 0.07% of the administered dose was found in the urine. The S- or O-glucuronide conjugates were the principle systemic metabolites and were found in amounts of up to 7.7% of the administered dose in rat urine. These conjugates were also overall the most abundant, occurring at about 46% of the administered dose in bile, followed by the parent compound, prothioconazole (about 1-22%), and prothioconazole-desthio (about 0.4-18%). An EFSA report states tha prothioconazole-desthio is more toxic than the parent compound. An ADI of 0.01 mg/kg bw/day was set based on the NOAEL of 1.1 mg/kg bw /day (liver histopathology and reduced weight gain in the rat carcinogenicity study), applying a 100 fold assessment factor.
TEBUCONAZOLE	(aerosol) NOEL (2 y)* for rats, 300 mg/kg diet for dogs, 100 mg/kg " for mice, 20 mg/kg " ADI 0.03 mg/kg b.w. * Toxicity Class WHO III; EPA III * Side effects of antiestrogens include hot flashes, osteoporosis, breast atrophy, vaginal dryness, and vaginal atrophy. In addition, they may cause depression and reduced libido. The antiestrogen withdrawal response is a paradoxical improvement in breast cancer caused by discontinuation of antiestrogen therapy for breast cancer. It has been documented rarely with the selective estrogen receptor modulators (SERMs) tamoxifen and raloxifene. The phenomenon indicates that these agents can somehow result in stimulation of breast cancer tumor progression under certain circumstances. One proposed theory for the mechanism is that the sensitivity of breast cells to estrogens shifts with estrogen deprivation, and upon antiestrogen withdrawal, endogenous estrogen acts in the manner of high-dose estrogen therapy in the breast to inhibit breast cancer growth and induce breast cancer cell death. The antioestrogen withdrawal syndrome is analogous to but less common and well-known than the antiandrogen withdrawal syndrome, a phenomenon in which paradoxical improvement in prostate cancer occurs upon discontinuation of antiandrogen therapy. aromatase inhibitors (AIs) are commonly used in breast cancer treatment, but they can cause various side effects, primarily due to estrogen depletion. Common side effects include musculoskeletal problems like joint pain (arthralgia), muscle stiffness, and bone loss leading to osteoporosis and fractures. Additionally, women may experience menopausal symptoms such as hot flashes, vaginal dryness, and sexual dysfunction. Other potential side effects include fatigue, insomnia, and an increased risk of cardiovascular events. ther Common Side Effects: Fatigue: Many patients report feeling tired or weak. Insomnia: Sleep disturbances are also common. Cardiovascular Events: While less frequent, there's a slightly increased risk of heart problems, especially in women with pre-existing conditions. Weight Gain: AIs can contribute to weight gain. Mood Changes: Some women experience depression or mood swings. Less Common but Serious Side Effects: Liver Problems: AIs can occasionally affect liver function. Skin Reactions: Skin rashes and other skin changes can occur. Cardiovascular Risks: Some studies suggest an increased risk of heart attack or stroke. Cytochrome P450 (CYP) enzyme modulators can cause adverse effects, primarily through drug-drug interactions. Specifically, CYP inhibitors can increase the concentration of other drugs in the body, leading to toxicity or adverse reactions, while CYP inducers can decrease drug concentrations, potentially causing therapeutic failure. CYP inhibitors, particularly those affecting CYP3A4, can cause a range of adverse effects due to their impact on drug metabolism. These effects include increased risk of toxicity from other drugs, arrhythmias like torsades de pointes, rhabdomyolysis, and even potentially fatal outcomes epending on the specific CYP inhibitor and the other drugs involved, adverse effects can include gastrointestinal disorders, liver damage, and neurological problems. CYP inhibitors can reduce the metabolism of other drugs, leading to higher concentrations in the bloodstream and potentially increasing the risk of side effects and toxicity People metabolize drugs differently, and factors like age, sex, genetics, and other medical conditions can influence how CYP inhibitors affect them. Incidents of liver injury or failure among modern antifungal medicines are very low to non-existent. However, some can cause allergic reactions in people.[There are also many drug interactions. Patients must read in detail the enclosed data sheet(s) of any medicine. For example, the azole antifungals such as ketoconazole or itraconazole can be both substrates and inhibitors of the P-glycoprotein, which (among other functions) excretes toxins and drugs into the intestines.] Azole antifungals also are both substrates and inhibitors of the cytochrome P450 family CYP3A4,[] causing increased concentration when administering, for example, calcium channel blockers, immunosuppressants, chemotherapeutic drugs, benzodiazepines, tricyclic antidepressants, macrolides and SSRIs.[35] Before oral antifungal therapies are used to treat nail disease, a confirmation of the fungal infection should be made.[Approximately half of suspected cases of fungal infection in nails have a non-fungal cause.[The side effects of oral treatment are significant and people without an infection should not take these drugs.[Azoles are the group of antifungals which act on the cell membrane of fungi. They inhibit the enzyme 14-alpha-sterol demethylase, a microsomal CYP, which is required for biosynthesis of ergosterol for the cytoplasmic membrane. This leads to the accumulation of 14-alpha-methylsterols resulting in impairment of function of certain membrane-bound enzymes and disruption of close packing of acyl chains of phospholipids, thus inhibiting growth of the fungi. Some azoles directly increase permeability of the fungal cell membrane. vntifungal resistance is a subset of antimicrobial resistance, that specifically applies to fungi that have become resistant to antifungals. Resistance to antifungals can arise naturally, for example by genetic mutation or through aneuploidy. Extended use of antifungals leads to development of antifungal resistance through various mechanisms. Some fungi (e.g. Candida krusei and fluconazole) exhibit intrinsic resistance to certain antifungal drugs or classes, whereas some species develop antifungal resistance to external pressures. Antifungal resistance is a One Health concern, driven by multiple extrinsic factors, including extensive fungicidal use, overuse of clinical antifungals, environmental change and host factors.] Unlike resistance to antibacterials, antifungal resistance can be driven by antifungal use in agriculture. Currently there is no regulation on the use of similar antifungal classes in agriculture and the clinic. The emergence of Candida auris as a potential human pathogen that sometimes exhibits multi-class antifungal drug resistance is concerning and has been associated with several outbreaks globally. The WHO has released a priority fungal pathogen list, including pathogens with antifungal resistance conazoles are azole antifungals used in agricultural and pharmaceutical products. Exposure to conazole fungicides leads to several toxic endpoints, including reproductive and endocrine. The results of animal experiments have shown that various conazole fungicides at high doses affect the structure and functions of reproductive organs. In males, adverse effects of conazole fungicides are manifested in the testes, prostate, sperm viability, fertility and sexual behaviour. Reduced testis weight, testis atrophy and reduced or absent sperm production were frequently observed. In female genitalia, structural changes in the ovaries and uterus have been observed. The extent

Continued...

of the changes depends on the dose and duration of treatment. Triazoles affected the expression of multiple genes involved in steroid hormone metabolism and modulate enzyme activity of multiple cytochrome P450 (CYP) and other metabolic enzymes in mammalian liver and other tissues. Conazole fungicides act as endocrine disruptors. Conazoles have been reported to reduce oestradiol and testosterone production and to increase progesterone concentration, indicating the inhibition of enzymes involved in the conversion of progesterone to testosterone. The reproductive effects are consistent with impairment of testosterone homeostasis. The disruption in steroid homeostasis is a common mode of action, leading to abnormal reproductive development and diminished reproductive function. At high doses, azole fungicides affect reproductive organs and fertility in several species.

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 (58), indicating azole toxicity in the liver being more acute than in other tissues.

Rapidly multiplying cancer cells synthesize greater amounts of cholesterol to build their membranes.

Sterol 14alpha-demethylase (CYP51) is potentially is a specific drug target because of its role in the production of cholesterol in animals

Sterol biosynthesis is an essential metabolic pathway in most eukaryotes and in some bacteria. Sterols (cholesterol in humans, sitosterol in plants, ergosterol and its C24-alkylated derivatives in fungi and protozoa) are required components of eukaryotic membranes, where they control fluidity and permeability and modulate functions of membrane-bound enzymes, receptors, and ion channels. These sterols also serve as precursors for multiple regulatory molecules that are crucial for cell division, growth, and development.

Sterol biosynthesis is the target for many drugs. Statins, as inhibitors of HMG-CoA reductase (EC.1.1.1.34), act upstream in the pathway at the step of mevalonate production and serve as major cholesterol-lowering drugs in humans, while azoles, as inhibitors of fungal and protozoan cytochrome P450 sterol 14a-demethylase (CYP51, EC.1.14.13.70), act downstream in the pathway at its postsqualene portion and are widely used as antimicrobial agent.

Human CYP51 [<35% amino acid sequence identity with fungal and <25% identity with protozoan orthologs (9)] has also been considered as a drug target.

Due to the side effects of statins, alternative drugs that target cholesterol biosynthesis in humans more specifically would be highly desirable, and CYP51, the lanosterol demethylase, is a target of interest in that several more distal (post-lanosterol) steps in the cholesterol synthesis pathway are known to be associated with hereditary diseases when they are attenuated (40). For instance, a loss of the sterol 7-reductase (DHCR7) is associated with a severe disease, Smith-Lemli-Opitz syndrome (41). Deficiency in the 3ß-hydroxysteroid dehydrogenase (NSDHL) is responsible for congenital hemidysplasia, ichthyosiform nevus, and limb defects (CHILD) syndrome (42). Mutations in the ?7,?8-sterol isomerase (EBP) are associated with Conradi-Hünermann-Happle syndrome, or X-linked dominant chondrodysplasia punctate type 2 (CDPX2) (40), and mutations in sterol 5-desaturase (SC5D) cause lathosterolosis (43). Compared with these problems, attenuating lanosterol 14-demethylation seems very favourable.

It is now generally accepted that cancer cells have elevated levels of cholesterol in lipid rafts and contain more lipid rafts than their normal cell line counterparts (26, 27, 29, 31, 44). Enhanced expression of enzymes of the cholesterol pathway has been reported in many cancer cell types (31, 45–47). Furthermore, certain types of cancer exhibit CYP51 gene amplification (https://www.cbioportal.org). It is likely that the earlier attempts to develop human CYP51 inhibitors were unsuccessful because they concentrated on substrate analogs, which had rather low inhibitory potency (10) and could not compete in efficiency with statins.

in comparison with orthologs from other biological kingdoms, human CYP51 has a broader substrate profile, displays faster catalytic rates, and is resistant to inhibition with the azole drugs and drug candidates that target CYP51s of microbial pathogens.

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However, screening a variety of commercial and experimental inhibitors of microbial CYP51 orthologs revealed that most of them (including all clinical antifungals) weakly inhibit human CYP51 activity,

Present in all animals, plants, fungi, in some protozoa and bacteria, the CYP51 protein located in the inner face of the endoplasmic reticulum is a membrane monospanning enzyme Its N-terminus includes an amphipathic helix, which links the catalytic subunit to the lipid bilayer.

Sterol synthesis is a very ancient pathway. After the appearance of molecular oxygen in the atmosphere, squalene-2,3-epoxide is formed and then cyclized to steroid precursors, such as lanosterol. Under the oxidative removal of methyl groups by CYP51, these precursors were transformed into ergosterol, which is critical in membrane permeability and fluidity in the fungal kingdom.

Cytochrome P450s (P450s, CYP) are an abundant hemease superfamily. As the first group of enzymes ranked as “superfamily,” cytochrome P450s play an important role in the primary as well as secondary metabolic pathway.

These members are important for catalyzing the oxidative process of various organic substrates, and play a critical role during heterogeneous metabolism and steroid conversion in biological kingdoms.

Unlike other CYP enzymes, CYP51 has a strong specificity. It only catalyzes the demethylation of a very narrow range of substrates, including lanoserol, obtusifoliol, 24,25-dihydrolanosterol, 24-methylenedihydrolanosterol and 4 beta-desmethyllanosterol. The CYP51-involved catalytic reaction consists of three steps, each of which requires one molecule of oxygen and two molecules of NADPH-sourced reduction equivalent. The first two steps are typical cytochrome P450 monooxygenation processes, during which the 14a methyl is converted to methyl alcohol and further converted to methyl aldehyde. And in the last step, the aldehyde group is transformed into formic acid and detached, accompanied with the synthesis of the delta-14, 15 double bond.

The 14a-demethylase is the only invariant P450 present in all sterol biosynthetic pathways, suggesting that all sterol 14a-demethylases share a common prokaryotic ancestor CYP51s are widely distributed in the fungal kingdom. However, in different species of fungi, there are still differences in the types and subtypes, as shown in the phylogenetic tree.

As potential anticancer agents, human CYP51 inhibitors are expected to have generally the same mode of action as statins in vivo, yet present certain important advantages. First, their specificity for the biosynthesis of cholesterol would help to avoid side effects of statins due to inhibition of other metabolic pathways. Second, as with systemic clinical antifungal azoles, which kill pathogenic cells that invade multiple organs and tissues, they should have broad tissue distribution. Finally, because all mammalian CYP51 enzymes share very high amino acid sequence identity (e.g., human/mouse 88%, human/dog 96%), animal in vivo models should produce highly relevant outcomes in preclinical trials. Potent inhibitors of human CYP51 may also have advantages as alternative medications for treatment of other cholesterol-related human diseases. The dual occupancy may be important in the mechanism of human CYP51 inhibition.

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Continued...

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

Legend: ✗ ***– Data 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
Quantum ProTeb 420 SC Fungicide	Not Available	Not Available	Not Available	Not Available	Not Available

	Endpoint	Test Duration (hr)	Species	Value	Source
prothioconazole	EC50	48h	Crustacea	1.482-1.795mg/L	4
	EC50(ECx)	96h	Algae or other aquatic plants	0.024-0.044mg/L	4
	EC50	96h	Algae or other aquatic plants	0.024-0.044mg/L	4
	LC50	96h	Fish	1.346-4.189mg/L	4

	Endpoint	Test Duration (hr)	Species	Value	Source
tebuconazole	EC50	72h	Algae or other aquatic plants	2.09-3.01mg/l	4
	EC50	48h	Crustacea	2.1-3.94mg/L	4
	NOEC(ECx)	672h	Crustacea	0.001mg/L	4
	EC50	96h	Algae or other aquatic plants	1.45mg/L	4
	LC50	96h	Fish	6.4mg/l	Not Available

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***

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.
DO NOT discharge into sewer or waterways.

Persistence and degradability

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

Bioaccumulative potential

Continued...

Ingredient	Bioaccumulation
prothioconazole	MEDIUM (LogKOW = 4.05)
tebuconazole	LOW (LogKOW = 3.7)

Mobility in soil

Ingredient	Mobility
tebuconazole	LOW (Log KOC = 20660)

SECTION 13 Disposal considerations

Waste treatment methods

Product / Packaging disposal

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.

- Antineoplastic (cytotoxic) wastes must be packed directly, ready for incineration, into colour-coded, secure, labelled, leak-proof containers sufficiently robust to withstand handling without breaking, bursting or leaking.
- Containers of special design are available for particular needs (such as disposal of sharps) and should be used.
- Once filled and closed, such containers must never be re-opened.
- Immediate containers must bear a nationally accepted symbol or device depicting cytotoxic substances and be labelled with the words: CYTOTOXIC WASTE - INCINERATE in a style of lettering approved by the national/ state authority.
- Where policies and procedures permit the merging of cytotoxic wastes with medical waste in an outer container used for medical waste, cytotoxic waste must first be placed in identifiable colour-coded/ labelled cytotoxic containers prior to merging.
- Management procedures must ensure that merged medical and cytotoxic waste is subjected to the incineration requirements appropriate for the total destruction of the cytotoxic waste.

WASTE STORAGE OF CYTOTOXIC WASTES
For the storage of cytotoxic waste, segregated or merged with medical waste, provide:

- special storage areas with adequate lighting.
- waste security and restriction of access to authorised persons.
- storage areas designed to facilitate easy routine cleaning and maintenance to hygienic standards, or post-spill decontamination.
- storage of cytotoxic waste in standard, identifying bins or other appropriate containers.

COLLECTION OF CYTOTOXIC WASTES

- Procedures for the collection of cytotoxic wastes, which are compatible with existing operational needs, and which protect workers, other people and the environment, must be developed.
- Waste must be removed from the site by contractors whose workers have been instructed in the protective methods to be used against the hazards involved, and who comply with the safe work practices established by internal and/or national/ state policies. Contractors must instruct, train and direct their personnel in the safe and legal handling of cytotoxic wastes. Contractor's personnel should observe the operating procedures of the waste-generator.
- Transport of cytotoxic wastes, through the community, must comply with the appropriate national/ state codes.

DESTRUCTION OF CYTOTOXIC WASTES

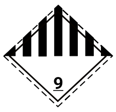
- Destruction of cytotoxic wastes should be carried out in multi-chambered incinerators, licensed for this purpose, operating at 1100 deg. C. or more, with a residence time of at least 1 second.
- Operators must be trained in handling procedures and hazards involved with handling the waste.
- Waste which arrives at the incinerator inappropriately packaged should **NOT** be returned to the waste generator. An authorised representative of the waste generator must attend the incinerator site to rectify the situation.
- **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.
- 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.

SECTION 14 Transport information

Labels Required

	
Marine Pollutant	
HAZCHEM	●3Z

Land transport (ADG)

14.1. **UN number or ID number**	3082
	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains tebuconazole and prothioconazole)

Continued...

14.2. **UN proper shipping name**		
14.3. **Transport hazard class(es)**	Class	9
	Subsidiary Hazard	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 tebuconazole and prothioconazole)	
14.3. **Transport hazard class(es)**	ICAO/IATA Class	9
	ICAO / IATA Subsidiary Hazard	Not Applicable
	ERG Code	9L
14.4. **Packing group**	III	
14.5. **Environmental hazard**	Environmentally hazardous	
14.6. **Special precautions for user**	Special provisions	A97 A158 A197 A215
	Cargo Only Packing Instructions	964
	Cargo Only Maximum Qty / Pack	450 L
	Passenger and Cargo Packing Instructions	964
	Passenger and Cargo Maximum Qty / Pack	450 L
	Passenger and Cargo Limited Quantity Packing Instructions	Y964
	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G

Sea transport (IMDG-Code / GGVSee)

14.1. **UN number**	3082	
14.2. **UN proper shipping name**	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains tebuconazole and prothioconazole)	
14.3. **Transport hazard class(es)**	IMDG Class	9
	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-F
	Special provisions	274 335 375 969
	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
prothioconazole	Not Applicable
tebuconazole	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
prothioconazole	Not Applicable
tebuconazole	Not Applicable

SECTION 15 Regulatory information

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

Continued...

prothioconazole is found on the following regulatory lists

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals

tebuconazole 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

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (prothioconazole; tebuconazole)
Canada - DSL	No (prothioconazole; tebuconazole)
Canada - NDSL	No (prothioconazole; tebuconazole)
China - IECSC	No (prothioconazole)
Europe - EINEC / ELINCS / NLP	No (prothioconazole)
Japan - ENCS	No (prothioconazole)
Korea - KECI	No (prothioconazole)
New Zealand - NZIoC	Yes
Philippines - PICCS	No (prothioconazole)
USA - TSCA	No (prothioconazole; tebuconazole)
Taiwan - TCSI	Yes
Mexico - INSQ	No (prothioconazole)
Vietnam - NCI	Yes
Russia - FBEPH	No (prothioconazole)
UAE - Control List (Banned/Restricted Substances)	No (prothioconazole)
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	15/01/2026
Initial Date	15/01/2026

SDS Version Summary

Version	Date of Update	Sections Updated
2.1	15/01/2026	Composition / information on ingredients - Ingredients

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
- 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

Continued...

- 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

end of SDS