Apparent Lad Ultra Seed Dressing

AIRR Apparent Pty Ltd.

Chemwatch Hazard Alert Code: 3

Chemwatch: **9656-05**
Version No: **2.1**
Safety Data Sheet according to Work Health and Safety Regulations (Hazardous Chemicals) 2023 and ADG requirements

Initial Date: **14/10/2026**
Revision Date: **14/10/2026**
Print Date: **23/11/2026**
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SECTION 1 Identification of the substance / mixture and of the company / undertaking

Product Identifier

Product name	Apparent Lad Ultra Seed Dressing
Chemical Name	Not Applicable
Synonyms	Not Available
Proper shipping name	ENVIRONMENTALLY HAZARDOUS SUBSTANCE, LIQUID, N.O.S. (contains imidacloprid)
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	Agricultural seed dressing fungicide and insecticide. Use according to manufacturer's directions.

Details of the manufacturer or importer of the safety data sheet

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

Emergency telephone number

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

SECTION 2 Hazards identification

Classification of the substance or mixture

Poisons Schedule	S6
Classification [1]	Acute Toxicity (Oral) Category 4, 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	**Warning**

Hazard statement(s)

H302	Harmful if swallowed.
H410	Very toxic to aquatic life with long lasting effects.

Precautionary statement(s) Prevention

P264	Wash all exposed external body areas thoroughly after handling.

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P270	Do not eat, drink or smoke when using this product.
P273	Avoid release to the environment.

Precautionary statement(s) Response

P391	Collect spillage.
P301+P312	IF SWALLOWED: Call a POISON CENTER/doctor/physician/first aider if you feel unwell.
P330	Rinse mouth.

Precautionary statement(s) Storage

Not Applicable

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
138261-41-3	30-35	imidacloprid
107534-96-3	1-1.5	tebuconazole
2634-33-5	<0.05	1,2-benzisothiazoline-3-one
55965-84-9	<0.0015	5-chloro-2-methyl-4-isothiazolin-3-one
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: ▸ Immediately flush body and clothes with large amounts of water, using safety shower if available. ▸ Quickly remove all contaminated clothing, including footwear. ▸ Wash skin and hair with running water. Continue flushing with water until advised to stop by the Poisons Information Centre. ▸ Transport to hospital, or doctor.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
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

for neonicotinoid intoxications:
- No specific antidotes are known.
- It is important to support respiration if signs of paralysis appear and to monitor blood pressure and pulse rate, since bradycardia and hypotonia are possible.
- Since the compounds **do NOT** inhibit cholinesterase activity, treatment with a reactivating oxime is not indicated.
- Symptoms of poisoning may be mediated by either stimulation or inhibition of nicotinic activity, or by other possible mechanisms. Therefore treatment with a nicotinic antagonist might be either ineffective or contraindicated.

Handbook of Neurotoxicology; Vol 1; Ed Edward J. Massaro, Humana Press, 2001

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This compound does not inhibit cholinesterase but toxic symptoms may resemble cholinergic stimulation.
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.

SECTION 5 Firefighting measures

Extinguishing media

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

Special hazards arising from the substance or mixture

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

Advice for firefighters

Fire Fighting	‣ Alert Fire Brigade and tell them location and nature of hazard. ‣ Wear full body protective clothing with breathing apparatus. ‣ Prevent, by any means available, spillage from entering drains or water course. ‣ Use 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. ‣ 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

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	▸ 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	Australian Standard (AS2639) and the National Institute of Health (USA) recommends that the preparation of injectable antineoplastic drugs should be performed in a Class II laminar flow biological safety cabinet and that personnel preparing drugs of this class should wear appropriate personal protective gear. Emphasise controls on containment. ▸ 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. ▸ **DO NOT** allow clothing wet with material to stay in contact with skin
Other information	Antineoplastics (cytotoxics): ▸ should be clearly identifiable to all personnel involved in their handling ▸ should be stored in impervious break-resistant containers ▸ should be stored in separate, clearly marked storage areas to minimise the risk of breakage, and to limit contamination in the event of leakage. Spill kits should be available in storage areas. ▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Metal can or drum ▸ Packaging as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid strong acids, bases. · Imidazole may be regarded as possessing pyrrole and pyridine like properties and therefore its reactivity might resemble that of the others. In general imidazole, in common with pyrazole, is less reactive than pyrrole and more reactive than benzene. · One peculiarity of imidazole is the impossibility to distinguish the two nitrogen atoms in solution. The hydrogen moves according to a tautomeric equilibria (that is exactly 50% of each form) from one nitrogen to the other. · In imidazole C4 and C5 are electron rich,whilst C2 is electron deficient. Imidazole can behave as both an electrophile and a nucleophile. The nucleophilic reaction leads ot N-substituted imidazoles. · Imidazole is an amphoteric substance. The acid - base behaviour of imidazole is important in determining its reactivity, because it is not just an amphoteric substance, thanks to the pyrrole-like and pyridine-like nitrogen but is also consistently more basic than pyridine (pKa of the conjugated acid 5.3) and more acidic than pyrrole (pKa 17.5). It all depends on the symmetry of the nitrogen atoms, that can equally stabilize either the positive (a proton) or the negative charge. ▸ Avoid reaction with oxidising agents

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SECTION 8 Exposure controls / personal protection

Control parameters

Occupational Exposure Limits (OEL)

INGREDIENT DATA

Not Available

MATERIAL DATA

1,2-Benzisothiazoline-3-one (BIT) produces sensitising effects and causes skin irritation at concentrations of 0.05%. Solutions containing the substance should contain levels considerably lower than 0.05%.
CEL TWA: 0.1 mg/m3; STEL 0.3 mg/m3 total isothiazolinones (Rohm and Haas)
(CEL = Chemwatch Exposure Limit)

Exposure controls

Appropriate engineering 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.

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

- 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

- Wear chemical protective gloves, e.g. PVC.
- Wear safety footwear or safety gumboots, e.g. Rubber

NOTE:

- The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact.
- Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed.

The selection of suitable gloves does not only depend on the material, but also on further marks of quality which vary from manufacturer to manufacturer. Where the chemical is a preparation of several substances, the resistance of the glove material can not be calculated in advance and has therefore to be checked prior to the application.
The exact break through time for substances has to be obtained from the manufacturer of the protective gloves and has to be observed when making a final choice.
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.

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	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.
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) ‣ Overalls. ‣ P.V.C apron. ‣ Barrier cream. ‣ Skin cleansing cream. ‣ Eye wash unit.

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	Red liquid.

Physical state	Liquid	**Relative density (Water = 1)**	1.17
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Available
pH (as supplied)	6-8	**Decomposition temperature (°C)**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (mm2/s)**	Not Available

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Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	>100	**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	Miscible	**pH as a solution (1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	Not Available
Heat of Combustion (kJ/g)	Not Available	**Ignition Distance (cm)**	Not Available
Flame Height (cm)	Not Available	**Flame Duration (s)**	Not Available
Enclosed Space Ignition Time Equivalent (s/m3)	Not Available	**Enclosed Space Ignition Deflagration Density (g/m3)**	Not Available

SECTION 10 Stability and reactivity

Reactivity	See section 7
Chemical stability	‣ Unstable in the presence of incompatible materials. ‣ Product is considered stable. ‣ Hazardous polymerisation will not occur.
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 Toxicological information

Information on toxicological effects

a) Acute Toxicity	There is sufficient evidence to classify this material as acutely toxic.
b) Skin Irritation/Corrosion	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	Based on available data, the classification criteria are not met.
h) STOT - Single Exposure	Based on available data, the classification criteria are not met.
i) STOT - Repeated Exposure	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 harmful. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by inhalation. The material is not classified as causing respiratory irritation under Regulation (EC) No 1272/2008 (CLP), as amended. Nevertheless, inhalation of vapours, fumes or aerosols, particularly following prolonged exposure, may cause respiratory discomfort and, occasionally, respiratory distress.
Ingestion	Toxic effects may result from the accidental ingestion of the material; animal experiments indicate that ingestion of less than 40 gram may be fatal or may produce serious damage to the health of the individual. Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by swallowing. Antineoplastic action is dependent on cytotoxic action which is not selective for malignant cells alone but may affect all rapidly dividing cells. The spectrum of effects seen with these agents is therefore similar although differences do arise. Acute adverse effects commonly produced by these agents include anorexia, nausea and vomiting (often central in origin and occurring minutes or hours after administration), allergic reaction (skin rashes, pruritus, erythema, hypotension, malaise, and anaphylaxis) and local irritant effects. Hyperuricaemia and acute renal failure (due to uric acid nephropathy) may result from the lysis of large numbers of cells and breakdown of nucleoproteins. The insecticidal activity of neonicotinoids (nitromethylene, chlorothiazoles, chlorpyridines, spinosads) is attributed to binding of the molecule to nicotinic acetylcholine receptors (nAChR) located in the insect central nervous system (CNS). This group of insecticides have much lower activity in vertebrate tissues due to differences in binding to nAChR subtypes. Poor penetration of the blood-brain barrier is an additional factor that acts to reduce the toxicity of neonicotinoids in vertebrates. Nevertheless at relatively high levels of exposure, these insecticides are neuroactive and produce neurotoxic effects. The principal effect may involve stimulation or inhibition. Tremors have occurred in mice treated with representative compounds. These compounds produce a variety of neurotoxic signs following acute exposure, with complete recovery within several hours or a few days following treatment. The most consistent finding at lower doses is evidence of decreased activity. At higher doses, tremors, impaired pupillary function (either dilated or pin-point pupils) and hypothermia are the most common effects. Finally, at near lethal doses, neurotoxic effects are assorted and include motor incoordination, (uncoordinated gait or impaired aerial righting), autonomic signs (lachrymation, urine staining) and CNS depression (marked decreased motor activity and decreased response to stimuli). Deaths associated with treatment occurred within 4-24 hours. There was no evidence of neuropathology associated with these compounds.

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	Certain findings (e.g tremors, impaired pupillary function and hypothermia) that are evident at sublethal doses are likely associated with nicotinic stimulation or represent nonspecific toxic effects. Sustained dietary exposure to relatively low doses produces little or no evidence of neurotoxicity. These results suggest that cumulative toxicity is not a concern with neonicotinoid insecticides. This outcome is consistent with their rapid metabolism and excretion in rats.
Skin Contact	Strong evidence exists that exposure to the material may produce very serious irreversible damage (other than carcinogenesis, mutagenesis and teratogenesis) following a single exposure by skin contact. Skin contact is not expected to produce harmful health effects, and the material is not classified as a skin irritant under Regulation (EC) No 1272/2008 (CLP), as amended. However, systemic effects have been identified following exposure of animals by at least one other route, and the material may still cause adverse health effects following entry through wounds, lesions or abrasions. Good hygiene practice requires that exposure be kept to a minimum and that suitable gloves be used in an occupational setting. Solutions of 0.5% strength 1,2-benzisothiazoline-3-one (BIT) are irritating to the skin. Allergenic effects also begin at 0.05% and have been confirmed in a series of case and patch test studies. When the substance was applied to human volunteers under an occlusive patch the maximum tolerated doses was 0.05%. Five hours after application of 0.1% (1000 ppm) one person showed moderate erythema with papule development which was interpreted as a reaction to the sticking plaster; in four persons there was mild reddening of the skin. The reaction had ameliorated in several persons after 72 hours. A second application produced various severe dermal reactions (erythema and papules) in 8 persons. A third application to several of the group produced erythema. Provocation tests with BIT showed the material to be sensitising. Of 20 metal workers with dermatitis, 4 were shown to have been sensitised to BIT in cutting oils. Cases of contact eczema in workers producing polyacrylate emulsions for paints and wax polish, in which BIT was the preservative, have been described. Epicutaneous challenge tests to BIT were positive. Similar findings have been described in the paper-manufacturing industry, in the rubber industry, in the control laboratory of a chemical plant and among workers producing ceramic moulds in which BIT was added to the mould oil Aqueous solutions of isothiazolinones may be irritating or even corrosive depending on concentration. Solutions containing more than 0.5% (5000 ppm active substance) may produce severe irritation of human skin whilst solutions containing more than 100 ppm may irritate the skin. Open cuts, abraded or irritated skin should not be exposed to this material Entry into the blood-stream through, for example, cuts, abrasions, puncture wounds or lesions, may produce systemic injury with harmful effects. Examine the skin prior to the use of the material and ensure that any external damage is suitably protected.
Eye	Although the liquid is not classified as an eye irritant under Regulation (EC) No 1272/2008 (CLP), as amended, direct contact with the eye may produce transient discomfort characterised by tearing or conjunctival redness (as with windburn).
Chronic	Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems. On the basis, primarily, of animal experiments, concern has been expressed that the material may produce carcinogenic or mutagenic effects; in respect of the available information, however, there presently exists inadequate data for making a satisfactory assessment. 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 hyper-responsive. Activities giving rise to short-term peak concentrations should receive particular attention when risk management is being considered. Health surveillance is appropriate for all employees exposed or liable to be exposed to a substance which may cause occupational asthma and there should be appropriate consultation with an occupational health professional over the degree of risk and level of surveillance. Harmful: danger of serious damage to health by prolonged exposure through inhalation, in contact with skin and if swallowed. Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests. Exposure to the material may cause concerns for human fertility, generally on the basis that results in animal studies provide sufficient evidence to cause a strong suspicion of impaired fertility in the absence of toxic effects, or evidence of impaired fertility occurring at around the same dose levels as other toxic effects, but which are not a secondary non-specific consequence of other toxic effects. Exposure to the material may cause concerns for humans owing to possible developmental toxic effects, generally on the basis that results in appropriate animal studies provide strong suspicion of developmental toxicity in the absence of signs of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not a secondary non-specific consequence of other toxic effects. In a teratogenic study in rats concentrations of up to 40 mg/kg 1,2-benzisothiazoline-3-one (BIT) were neither embryotoxic nor teratogenic. The material is not mutagenic. In a 2-year carcinogenicity study with rats, BIT did not produce excess tumours. The results derived from this test are questionable because no dose series was administered and because there were too few animals. A 90-day study with beagle dogs receiving oral doses showed reduced food consumption and body weight gain as well as mild anaemia, increases in the weights of liver and in male animals, brain and spleen weights. The no-observed-effect-level (NOEL) was given as 165 mg/kg (ie 0.5 BIT in the diet). A 90-day study with rats receiving dietary BIT showed reduced liver and pituitary weights in males. The NOEL was less than 0.1 %. The isothiazolinones are known contact sensitisers. Data are presented which demonstrate that, in comparison with the chlorinated and dichlorinated compounds which share immunological cross-reactivity, the non-chlorinated isothiazolinones have a lower potential for sensitization and no documented immunological cross-reaction with the chlorinated isothiazolinones. The risk of sensitization depends on how contact with the product occurs. The risk is greater when the skin barrier has been damaged and smaller when the skin is healthy. Dermatological studies have demonstrated that mixed isothiazolinone concentrations below 20 ppm may cause sensitisation and that allergic reactions can be provoked in sensitized persons even with concentrations in the range of 7-15 ppm active isothiazolinones. The isothiazolinones are a group of heterocyclic sulfur-containing compounds. In general all are electrophilic molecules containing an activated N-S bond that enables them with nucleophilic cell entities, thus exerting biocidal activity. A vinyl activated chlorine atom makes allows to molecule to exert greater antimicrobial efficiency but at the same time produces a greater potential for sensitisation. Several conclusions relating to the sensitising characteristics of the isothiazolinones may therefore be drawn* : ‣ The strongest sensitisers are the chlorinated isothiazolinones. ‣ There are known immunological cross-reactions between at least 2 different chlorinated isothiazolinones. ‣ There appears to be no immunological cross reaction between non-chlorinated isothiazolinones and chlorinated isothiazolinones. ‣ Although classified as sensitisers, the nonchlorinated isothiazolinones are considerably less potent sensitisers than are the chlorinated isothiazolinones. ‣ By avoiding the use of chlorinated isothiazolinones, the potential to induce sensitisation is greatly reduced. ‣ Despite a significant percentage of the population having been previously sensitised to chlorinated and non-chlorinated species, it is likely that careful and judicious use of non-chlorinated isothiazolinones will result in reduced risk of allergic reactions in those persons. ‣ Although presently available data promise that several non-chlorinated isothiazolinones will offer effective antimicrobial protection in industrial and personal care products, it is only with the passage of time that proof of their safety in use or otherwise will become

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available.
* B.R. Alexander: Contact Dermatitis 2002, 46, pp 191-196
Although there have been conflicting reports in the literature, it has been reported by several investigators that isothiazolinones are mutagenic in ***Salmonella typhimurium*** strains (Ames test). Negative results were obtained in studies of the DNA-damaging potential of mixed isothiazolinones (Kathon) in mammalian cells ***in vitro*** and of cytogenetic effects and DNA-binding ***in vivo.*** The addition of rat liver S-9 (metabolic activation) reduced toxicity but did not eliminate mutagenicity. These compounds bind to the proteins in the S-9. At higher concentrations of Kathon the increase in mutagenicity may be due to an excess of unbound active compounds.
A study of cutaneous application of Kathon CG in 30 months, three times per week at a concentration of 400 ppm (0.04%) a.i. had no local or systemic tumourigenic effect in male mice. No dermal or systemic carcinogenic potential was observed.
Reproduction and teratogenicity studies with rats, given isothiazolinone doses of 1.4-14 mg/kg/day orally from day 6 to day 15 of gestation, showed no treatment related effects in either the dams or in the foetuses
Imidazole is structurally related to histamine and has been used as an antagonist to counteract the effects of excess histamine found in certain induced physiological conditions (it therefore acts as an antihistamine).
Imidazoles have been reported to disrupt male fertility through disruption of testicular function.
Certain imidazole fungicides provoke histamine release by a non-immunological mechanism, induce airway constriction in guinea-pigs and hence may be harmful to spray operators who might inhale fungicide aerosols used for plant protection.
Imidazole fungicides inhibit the cytochrome P450 (CYP) complex, including the 14alpha-demethylase (CYP51) enzyme required for ergosterol biosynthesis, in fungal cell membranes. In addition, intracellular accumulation of toxic methylated sterols occurs and the synthesis of triglycerides and phospholipids is altered. Disturbances in oxidative and peroxidative enzyme activities lead to an intracellular toxic concentration of hydrogen peroxide. As a result, intracellular organelle destruction then leads to cell necrosis.
2-Methylimidazole decreased luteinising hormone secretion and tissue interstitial fluid testosterone concentration two hours after injection into Sprague Dawley rats.
Imidazoles bind to cytochrome P450 haeme, resulting in inhibition of catalysis. However, 2-substituted imidazoles are considered to be poor inhibitors. Imidazole is probably an inducer of cytochrome P4502E1. In general, inducers of this isozyme stabilise the enzyme by preventing phosporylation of a serine which leads to haeme loss.
Several drugs containing an imidazole moiety were retained and bound in connective tissue when administered to laboratory animals. The bound material was primarily recovered from elastin (70%) and the collagen. It is postulated that reaction with aldehydes gives an aldol condensation pro
Delayed or long-term effects may result from the action of antineoplastic agents on rapidly dividing normal cells in the bone marrow, lymphoreticular tissue, gastrointestinal mucosa, skin, gonads and foetus. The most common of these is bone-marrow depression with leucopenia, anaemia, thrombocytopenia and bleeding. Immunosuppressant effects involve both antibody and cell-mediated immunity and may increase susceptibility to infection and increase the risk of haemorrhage, both potentially life-threatening. GI effects may include stomatitis, mouth ulcers, oesophagitis, abdominal pain, haemorrhage, diarrhoea and intestinal ulceration and perforation. Reversible baldness (alopecia) may result from the action of theses drugs on active hair follicles. Wound healing may be delayed. Long-term effects on the gonads may result in amenorrhoea and the inhibition of spermatogenesis.
Most antineoplastics are potentially mutagenic or teratogenic and coupled with the effects of prolonged immunosuppression may also be carcinogenic.

Apparent Lad Ultra Seed Dressing

TOXICITY	IRRITATION
Not Available	Not Available

imidacloprid

TOXICITY	IRRITATION
dermal (rat) LD50: >5000 mg/kg[2]	Not Available
Inhalation (Rat) LC50: >0.069 mg/L4h[2]	
Oral (Mouse) LD50; 131 mg/kg[1]	

tebuconazole

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

1,2-benzisothiazoline-3-one

TOXICITY	IRRITATION
dermal (rat) LD50: >2000 mg/kg[1]	Eye: adverse effect observed (irreversible damage)[1]
Oral (Rat) LD50: 454 mg/kg[1]	Skin (Human - man): 0.05%
	Skin (Human): 1%/1H
	Skin (Human): 5%/48H - Mild
	Skin: no adverse effect observed (not irritating)[1]

5-chloro-2-methyl-4-isothiazolin-3-one

TOXICITY	IRRITATION
dermal (rat) LD50: >1008 mg/kg[2]	Eye: adverse effect observed (irreversible damage)[1]
Inhalation (Rat) LC50: 1.23 mg/l4h[2]	Skin (Human - woman): 0.01%
Oral (Rat) LD50: 53 mg/kg[2]	Skin (Human): 0.01% - Severe
	Skin (Human): 0.1%/48H
	Skin: adverse effect observed (corrosive)[1]
	Skin: adverse effect observed (irritating)[1]

Legend: ***1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances***

IMIDACLOPRID

ADI 0.057 mg/kg bw. *
[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]

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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 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

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	(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. 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. 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. 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. 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Enhanced expression of enzymes of the cholesterol pathway has been reported in many cancer cell types. Furthermore, certain types of cancer exhibit CYP51 gene amplification. 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 and could not compete in efficiency with statins. 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. 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1,2-BENZISOTHIAZOLINE-3-ONE	The predominant fate of the thiazole ring is oxidative ring scission catalysed by cytochrome P450 (CYP) and formation of the corresponding alpha-dicarbonyl metabolites and thioamide derivatives. The well-established toxicity associated with thioamides and thioureas has led to the speculation that thiazole toxicity is attributed to ring scission yielding the corresponding thioamide metabolite. Ring opening has also been observed in benzothiazoles. For instance, benzothiazole itself is converted to S-methylmercaptoaniline. **Acute toxicity** data show that 1,2-benzisothiazoline-3-one (BIT) is moderately toxic by the oral and dermal routes but that this chemical is a severe eye irritant. Irritation to the skin from acute data show only mild skin irritation , but repeated dermal application indicated a more significant skin irritation response.

Continued...

	The neurotoxicity observed in the rat acute oral toxicity study (piloerection and upward curvature of the spine at 300 mg/kg and above; decreased activity, prostration, decreased abdominal muscle tone, reduced righting reflex, and decreased rate and depth of breathing at 900 mg/kg) and the acute dermal toxicity study (upward curvature of the spine was observed in increased incidence, but this was absent after day 5 post-dose at a dose of 2000 mg/kg) were felt to be at exposures in excess of those expected from the use pattern of this pesticide and that such effects would not be observed at estimated exposure doses. **Subchronic oral toxicity** studies showed systemic effects after repeated oral administration including decreased body weight, increased incidence of forestomach hyperplasia, and non-glandular stomach lesions in rats. In dogs, the effects occurred at lower doses than in rats, and included alterations in blood chemistry (decreased plasma albumin, total protein, and alanine aminotransferase) and increased absolute liver weight. **Developmental toxicity** studies were conducted in rats with maternal effects including decreased body weight gain, decreased food consumption, and clinical toxicity signs (audible breathing, haircoat staining of the anogenital region, dry brown material around the nasal area) as well as increased mortality. Developmental effects consisted of increases in skeletal abnormalities (extra sites of ossification of skull bones, unossified sternebrae) but not external or visceral abnormalities. **Reproductive toxicity:** In a two- generation reproduction study, parental toxicity was observed at 500 ppm and was characterized by lesions in the stomach. In pups, toxic effects were reported at 1000 ppm and consisted of preputial separation in males and impaired growth and survival in both sexes. The reproduction study did not show evidence of increased susceptibility of offspring.
5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE	Considered to be the major sensitiser in Kathon CG (1) (1). Bruze etal - Contact Dermatitis 20: 219-39, 1989 Asthma-like symptoms may continue for months or even years after exposure to the material ends. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur after exposure to high levels of highly irritating compound. Main criteria for diagnosing RADS include the absence of previous airways disease in a non-atopic individual, with sudden onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. Other criteria for diagnosis of RADS include a reversible airflow pattern on lung function tests, moderate to severe bronchial hyperreactivity on methacholine challenge testing, and the lack of minimal lymphocytic inflammation, without eosinophilia. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. On the other hand, industrial bronchitis is a disorder that occurs as a result of exposure due to high concentrations of irritating substance (often particles) and is completely reversible after exposure ceases. The disorder is characterized by difficulty breathing, cough and mucus production. Based on laboratory and animal testing, exposure to the material may result in irreversible effects and mutations in humans. Formaldehyde generators (releasers) are often used as preservatives. The maximum authorised concentration of free formaldehyde is 0.2% and must be labelled with the warning sign "contains formaldehyde" where the concentration exceeds 0.05%.The use of formaldehyde-releasing preservatives ensures that the level of free formaldehyde in the products is always low but sufficient to inhibit microbial growth - it disrupts metabolism to cause death of the organism. However there is a concern that formaldehyde generators can produce amines capable of causing cancers (nitrosamines) when used in formulations containing amines. The material may be irritating to the eye, with prolonged contact causing inflammation. Repeated or prolonged exposure to irritants may produce conjunctivitis. The material may cause skin irritation after prolonged or repeated exposure and may produce on contact skin redness, swelling, the production of vesicles, scaling and thickening of the skin.
Apparent Lad Ultra Seed Dressing & 1,2-BENZISOTHIAZOLINE-3-ONE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE	The following information refers to contact allergens as a group and may not be specific to this product. Contact allergies quickly manifest themselves as contact eczema, more rarely as urticaria or Quincke's oedema. The pathogenesis of contact eczema involves a cell-mediated (T lymphocytes) immune reaction of the delayed type. Other allergic skin reactions, e.g. contact urticaria, involve antibody-mediated immune reactions. The significance of the contact allergen is not simply determined by its sensitisation potential: the distribution of the substance and the opportunities for contact with it are equally important. A weakly sensitising substance which is widely distributed can be a more important allergen than one with stronger sensitising potential with which few individuals come into contact. From a clinical point of view, substances are noteworthy if they produce an allergic test reaction in more than 1% of the persons tested. No significant acute toxicological data identified in literature search.
Apparent Lad Ultra Seed Dressing & TEBUCONAZOLE	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 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.
1,2-BENZISOTHIAZOLINE-3-ONE & 5-CHLORO-2-METHYL-4-ISOTHIAZOLIN-3-ONE	In light of potential adverse effects, and to ensure a harmonised risk assessment and management, the EU regulatory framework for biocides has been established with the objective of ensuring a high level of protection of human and animal health and the environment. To this aim, it is required that risk assessment of biocidal products is carried out before they can be placed on the market. A central element in the risk assessment of the biocidal products are the utilization instructions that defines the dosage, application method and amount of applications and thus the exposure of humans and the environment to the biocidal substance. Humans may be exposed to biocidal products in different ways in both occupational and domestic settings. Many biocidal products are intended for industrial sectors or professional uses only, whereas other biocidal products are commonly available for private use by non-professional users. In addition, potential exposure of non-users of biocidal products (i.e. the general public) may occur indirectly via the environment, for example through drinking water, the food chain, as well as through atmospheric and residential exposure. Particular attention should be paid to the exposure of vulnerable sub-populations, such as the elderly, pregnant women, and children. Also pets and other domestic animals can be exposed indirectly following the application of biocidal products. Furthermore, exposure to biocides may vary in terms of route (inhalation, dermal contact, and ingestion) and pathway (food, drinking water, residential, occupational) of exposure, level, frequency and duration.

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

Continued...

– Data available to make classification

SECTION 12 Ecological information

Toxicity

Apparent Lad Ultra Seed Dressing	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	Not Available	Not Available	Not Available	Not Available	Not Available

imidacloprid	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	>10mg/l	2
	EC50	48h	Crustacea	0.001-0.011mg/L	4
	NOEC(ECx)	216h	Crustacea	<0.001mg/L	4
	LC50	96h	Fish	<0.001mg/L	4

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

1,2-benzisothiazoline-3-one	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.07mg/L	2
	EC50	48h	Crustacea	0.097mg/L	4
	NOEC(ECx)	72h	Algae or other aquatic plants	0.04mg/L	2
	LC50	96h	Fish	0.067-0.29mg/L	4

5-chloro-2-methyl-4-isothiazolin-3-one	**Endpoint**	**Test Duration (hr)**	**Species**	**Value**	**Source**
	EC50	72h	Algae or other aquatic plants	0.018-0.026mg/L	4
	EC50	48h	Crustacea	4.71mg/l	1
	EC50	96h	Algae or other aquatic plants	0.03-0.13mg/L	4
	NOEC(ECx)	504h	Crustacea	0.172mg/l	1
	LC50	96h	Fish	0.13-0.31mg/L	4

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

Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment.
Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.
Wastes resulting from use of the product must be disposed of on site or at approved waste sites.
for neonicotinoids:
The neonicotinoids are a class of insecticides with a common mode of action that affects the central nervous system of insects, causing paralysis and death. Some uncertainties have been identified since their initial registration regarding the potential environmental fate and effects of neonicotinoid pesticides, particularly as they relate to pollinators. Some of the compounds within this class have been demonstrated to persist for several years and residues have been detected in plants for several years following application.
Studies conducted in Europe in the late 1990s have suggested that neonicotinic residues can accumulate in pollen and nectar of treated plants and represent a potential risk to pollinators. Adverse effects on pollinators (beekill incidents) have also been reported in Europe that have further heightened concerns regarding the potential direct and/or indirect role that neonicotinic pesticides may have in pollinator declines. Data from studies conducted in Europe have supported concerns regarding the persistence of neonicotinoids; however, the translocation of residues into pollen and nectar of treated plants and the potential effect that these residues may have on bees remains uncertain.
For imidacloprid:
Solubility in water: 610 mg/l 920 C)
log Kow: 0.57 (21 C)
Vapour pressure: 4 x 10-10 pa (20 C
Environmental fate:
Due to the special pattern of substituents in the molecule imidacloprid does not show base-like properties under environmental conditions. That precludes pH-value dependent influences on other properties, e.g. the water solubility, which was found to be constant at a comparatively high level of 610 mg/L between pH 4 and 9 and at 20 °C. Substances with a water solubility in this range are termed as hydrophilic due to a tendency to undergo easily interactions with water. The hydrophilicity of imidacloprid is also reflected by its low partition coefficient in octanol and water. This indicates that it has no potential to accumulate in biological tissues and further enrichment in the food chain.
Laboratory degradation trials with artificial sunlight in distilled water indicated a rapid degradation with a half-life of 1 hour.
Imidacloprid is completely photo-degraded and a large number of products each occurring at low amounts are formed. Thus, the photo-degradation of imidacloprid is regarded as an important elimination process from all aqueous solutions which can be exposed to sunlight, i.e. surface water, spray solutions, aerosol and fog or rain water.
Imidacloprid is also degraded under anaerobic dark conditions. In a laboratory study performed with 14C labelled imidacloprid a DT50 of 27 days was determined for the entire anaerobic aquatic system, which is considerably shorter than determined in the aerobic tests. No other degradation products were found. However, in the reducing environment the first steps of degradation resulting in desnitro imidacloprid are promoted, while the subsequent steps are retarded. This is to be expected as under anaerobic conditions the oxygen necessary for most degradation steps is not available. Further degradation will occur as soon as the conditions change from anaerobic to aerobic ones. Therefore, an accumulation of desnitro-imidacloprid
is not to be expected since strict anaerobic conditions do not exist for a longer time period at locations treated by imidacloprid.
Fate in air: Imidacloprid has a very low vapour pressure of 4×10-10 Pa at 20 C which precludes dissipation by way of volatilisation.
Terrestrial fate: Imidacloprid undergoes mainly biotic degradation together with photochemical degradation and binding to soil matter . Since these processes are continuous though not very rapid (with the formation of carbon dioxide as the product of complete mineralisation), there is no risk of accumulation in soil, even if imidacloprid is applied repeatedly over several seasons. This has been found under laboratory conditions and in field studies on cropped or bare soil.
Koc values (average 225 ml/g) (a measure of soil binding) are in the range where translocation from soil is negligible under normal conditions. The degree of binding of imidacloprid to the soil is high and is not readily released. Thus the overall effect is that the compound remains in the upper root zone. The translocation to deeper-lying soil zones is negligible. This is confirmed by laboratory studies and three free-land Lysimeter trails
Ecotoxicity:
Bird Acute oral LD50: Japanese quail 31 mg/kg; bobwhite quail 152 mg/kg

Continued...

Fish LC50 (96 h): golden orfe 237 mg/l; rainbow trout 211 mg/l
Daphnia magna EC50 (48 h): 85 mg/l
Harmful to honeybees by foliar application but no problems expected when used as a seed treatment.
For antineoplastics:
Ecotoxicity:
Because antineoplastics are genotoxic, mutagenic and carcinogenic concerns are warranted for their potential effect in the environment. There are a number of known mammalian toxic and nausea effects associated with antineoplastic treatment, which could indicate that similar effects, might be expected in non-target mammals, and possibly also in non-target species other than mammals. Total dosage over a whole therapy protocol is approximately 150 mg /kg body weight. Approximately 14-53% of the administered pharmaceutical is excreted unmetabolised into urine.
Antineoplastics as a class of drugs are of potential concern for environmental impacts, not just for their acute toxicity but perhaps more for their ability to effect subtle genetic changes, the cumulative impact of which over time can lead to more profound ecologic change. Hospitals are the major source of genotoxic drugs. publicly-owned waste-water treatment works (POTWs) that service hospitals, especially multiple hospitals, are likely candidates for releasing these chemicals into surface waters.
Antineoplastics are highly [geno]toxic compounds, primarily from hospitals, with poor removal from sewage treatment plants (STWs). Antineoplastic agents, antitumour agents primarily used only within hospitals for chemotherapy, are found sporadically and in a range of concentrations, probably because only small amounts are introduced to STWs via domestic sewage because of their long-lived physiologic retention.
These compounds act as nonspecific alkylating agents (i.e., specific receptors are not involved) and therefore have the potential to act as either acute or long-felt stressors (mutagens carcinogens/ teratogens/ embryotoxins) in any organism.
Using well-established QSAR modelling techniques almost 1/5 of the commonly used antineoplastics were predicted to be very toxic to algae, and close to 1/3 were predicted to be non-toxic to plants. A third of the compounds were predicted to be very toxic to daphnids, and almost half were predicted to be non-toxic to daphnids. Slightly more than 1/5 were predicted to be very toxic to fish, and 47% were predicted to be non-toxic to fish.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
imidacloprid	HIGH	HIGH
tebuconazole	HIGH	HIGH
5-chloro-2-methyl-4-isothiazolin-3-one	HIGH	HIGH

Bioaccumulative potential

Ingredient	Bioaccumulation
imidacloprid	LOW (LogKOW = 0.57)
tebuconazole	LOW (LogKOW = 3.7)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (LogKOW = 0.0444)

Mobility in soil

Ingredient	Mobility
imidacloprid	LOW (Log KOC = 5048)
tebuconazole	LOW (Log KOC = 20660)
5-chloro-2-methyl-4-isothiazolin-3-one	LOW (Log KOC = 45.15)

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. ▸ **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. ▸ 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.

Continued...

- 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.

SECTION 14 Transport information

Labels Required



Marine Pollutant



HAZCHEM •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 imidacloprid)
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 imidacloprid)
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 imidacloprid)

Continued...

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
imidacloprid	Not Applicable
tebuconazole	Not Applicable
1,2-benzisothiazoline-3-one	Not Applicable
5-chloro-2-methyl-4-isothiazolin-3-one	Not Applicable

14.7.3. Transport in bulk in accordance with the IGC Code

Product name	Ship Type
imidacloprid	Not Applicable
tebuconazole	Not Applicable
1,2-benzisothiazoline-3-one	Not Applicable
5-chloro-2-methyl-4-isothiazolin-3-one	Not Applicable

SECTION 15 Regulatory information

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

imidacloprid 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
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

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

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

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

5-chloro-2-methyl-4-isothiazolin-3-one 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 6
Australian Inventory of Industrial Chemicals (AIIC)

Additional Regulatory Information

Not Applicable

National Inventory Status

National Inventory	Status
Australia - AIIC / Australia Non-Industrial Use	No (imidacloprid; tebuconazole)
Canada - DSL	No (imidacloprid; tebuconazole)
Canada - NDSL	No (imidacloprid; tebuconazole; 1,2-benzisothiazoline-3-one; 5-chloro-2-methyl-4-isothiazolin-3-one)
China - IECSC	Yes
Colombia - Colombia National Industrial Chemicals Inventory	No (imidacloprid; tebuconazole; 1,2-benzisothiazoline-3-one; 5-chloro-2-methyl-4-isothiazolin-3-one)
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	Yes
Korea - KECI	Yes
New Zealand - NZIoC	Yes

Continued...

National Inventory	Status
Philippines - PICCS	Yes
USA - TSCA	TSCA Inventory 'Active' substance(s) (1,2-benzisothiazoline-3-one; 5-chloro-2-methyl-4-isothiazolin-3-one); No (imidacloprid; tebuconazole)
Taiwan - TCSI	Yes
Mexico - INSQ	Yes
Vietnam - NCI	Yes
Russia - FBEPH	No (imidacloprid)
UAE - Control List (Banned/Restricted Substances)	No (1,2-benzisothiazoline-3-one; 5-chloro-2-methyl-4-isothiazolin-3-one)
Legend:	***Yes = All CAS declared ingredients are on the inventory*** ***No = One or more of the CAS listed ingredients are not on the inventory. These ingredients may be exempt or will require registration.***

SECTION 16 Other information

Revision Date	14/10/2026
Initial Date	14/10/2026

Other information

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

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

Definitions and abbreviations

- PC—TWA: Permissible Concentration-Time Weighted Average
- PC—STEL: Permissible Concentration-Short Term Exposure Limit
- IARC: International Agency for Research on Cancer
- ACGIH: American Conference of Governmental Industrial Hygienists
- STEL: Short Term Exposure Limit
- TEEL: Temporary Emergency Exposure Limit。
- IDLH: Immediately Dangerous to Life or Health Concentrations
- ES: Exposure Standard
- OSF: Odour Safety Factor
- NOAEL: No Observed Adverse Effect Level
- LOAEL: Lowest Observed Adverse Effect Level
- TLV: Threshold Limit Value
- LOD: Limit Of Detection
- OTV: Odour Threshold Value
- BCF: BioConcentration Factors
- BEI: Biological Exposure Index
- DNEL: Derived No-Effect Level
- PNEC: Predicted no-effect concentration
- MARPOL: International Convention for the Prevention of Pollution from Ships
- IMSBC: International Maritime Solid Bulk Cargoes Code
- IGC: International Gas Carrier Code
- IBC: International Bulk Chemical Code

- AIIC: Australian Inventory of Industrial Chemicals
- DSL: Domestic Substances List
- NDSL: Non-Domestic Substances List
- IECSC: Inventory of Existing Chemical Substance in China
- EINECS: European INventory of Existing Commercial chemical Substances
- ELINCS: European List of Notified Chemical Substances
- NLP: No-Longer Polymers
- ENCS: Existing and New Chemical Substances Inventory
- KECI: Korea Existing Chemicals Inventory
- NZIoC: New Zealand Inventory of Chemicals
- PICCS: Philippine Inventory of Chemicals and Chemical Substances
- TSCA: Toxic Substances Control Act
- TCSI: Taiwan Chemical Substance Inventory
- INSQ: Inventario Nacional de Sustancias Químicas
- NCI: National Chemical Inventory
- FBEPH: Russian Register of Potentially Hazardous Chemical and Biological Substances

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