AC Emporium 500 Seed Treatment

AXICHEM Pty Ltd

Chemwatch Hazard Alert Code: **3**

Chemwatch: **5158-26**
Version No: **2.1.1.1**
Material Safety Data Sheet according to NOHSC and ADG requirements

Issue Date: **01/12/2014**
Print Date: **01/12/2014**
Initial Date: **Not Available**
S.Local.AUS.EN

SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	AC Emporium 500 Seed Treatment
Chemical Name	Not Applicable
Proper shipping name	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains fipronil)
Chemical formula	Not Applicable
Other means of identification	Not Available
CAS number	Not Applicable

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

Relevant identified uses	Agricultural insecticide for seed treatment.

Details of the manufacturer/importer

Registered company name	AXICHEM Pty Ltd
Address	18 Conquest Way Wangara 6065 WA Australia
Telephone	+61 8 9302 4666
Fax	Not Available
Website	www.axichem.com.au
Email	msds@axichem.com.au

Emergency telephone number

Association / Organisation	Not Available
Emergency telephone numbers	1800 039 008 (all hours)
Other emergency telephone numbers	1800 039 008 (all hours)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

HAZARDOUS SUBSTANCE. DANGEROUS GOODS. According to the Criteria of NOHSC, and the ADG Code.

Poisons Schedule	S6
Risk Phrases [1]	**R23/24/25** **Toxic** by inhalation, in contact with skin and if swallowed. **R50/53** Very toxic to aquatic organisms, may cause long-term adverse effects in the aquatic environment. **R48/25** Toxic: danger of serious damage to health by prolonged exposure if swallowed.
Legend:	*1. Classified by Chemwatch; 2. Classification drawn from HSIS ; 3. Classification drawn from EC Directive 1272/2008 - Annex VI*

Relevant risk statements are found in section 2

Indication(s) of danger	N, T

SAFETY ADVICE

S01	Keep locked up.
S07	Keep container tightly closed.

Continued...

S09	Keep container in a well ventilated place.
S13	Keep away from food, drink and animal feeding stuffs.
S20	When using do not eat or drink.
S23	Do not breathe gas/fumes/vapour/spray.
S28	After contact with skin, wash immediately with plenty of water
S29	Do not empty into drains.
S35	This material and its container must be disposed of in a safe way.
S36	Wear suitable protective clothing.
S37	Wear suitable gloves.
S38	In case of insufficient ventilation, wear suitable respiratory equipment.
S40	To clean the floor and all objects contaminated by this material, use water.
S45	In case of accident or if you feel unwell IMMEDIATELY contact Doctor or Poisons Information Centre (show label if possible).
S46	If swallowed, seek medical advice immediately and show this container or label.
S51	Use only in well ventilated areas.
S56	Dispose of this material and its container at hazardous or special waste collection point.
S57	Use appropriate container to avoid environmental contamination.
S61	Avoid release to the environment. Refer to special instructions/Safety data sheets.
S63	In case of accident by inhalation: remove casualty to fresh air and keep at rest.

Other hazards

	May produce discomfort of the eyes and skin*.
	Cumulative effects may result following exposure*.

SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
120068-37-3	30-60	fipronil
Not Available	30-60	Ingredients determined not to be hazardous

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	If this product comes in contact with the eyes: ▸ Wash out immediately with fresh running water. ▸ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▸ Seek medical attention without delay; if pain persists or recurs seek medical attention. ▸ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	If skin contact occurs: ▸ Immediately remove all contaminated clothing, including footwear. ▸ Flush skin and hair with running water (and soap if available). ▸ Seek medical attention in event of irritation.
Inhalation	▸ If fumes or combustion products are inhaled remove from contaminated area. ▸ Lay patient down. Keep warm and rested. ▸ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▸ Apply artificial respiration if not breathing, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▸ Transport to hospital, or doctor, without delay.
Ingestion	▸ If swallowed do **NOT** induce vomiting. ▸ If vomiting occurs, lean patient forward or place on left side (head-down position, if possible) to maintain open airway and prevent aspiration. ▸ Observe the patient carefully. ▸ Never give liquid to a person showing signs of being sleepy or with reduced awareness; i.e. becoming unconscious. ▸ Give water to rinse out mouth, then provide liquid slowly and as much as casualty can comfortably drink. ▸ Seek medical advice.

Indication of any immediate medical attention and special treatment needed

Treat symptomatically.
As in all cases of suspected poisoning, follow the ABCDEs of emergency medicine (airway, breathing, circulation, disability, exposure), then the ABCDEs of toxicology (antidotes, basics, change absorption, change distribution, change elimination).
For poisons (where specific treatment regime is absent):

BASIC TREATMENT

- Establish a patent airway with suction where necessary.
- Watch for signs of respiratory insufficiency and assist ventilation as necessary.
- Administer oxygen by non-rebreather mask at 10 to 15 L/min.
- Monitor and treat, where necessary, for pulmonary oedema.
- Monitor and treat, where necessary, for shock.

Continued...

- Anticipate seizures.
- **DO NOT** use emetics. Where ingestion is suspected rinse mouth and give up to 200 ml water (5 ml/kg recommended) for dilution where patient is able to swallow, has a strong gag reflex and does not drool.

ADVANCED TREATMENT

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

BRONSTEIN, A.C. and CURRANCE, P.L.
EMERGENCY CARE FOR HAZARDOUS MATERIALS EXPOSURE: 2nd Ed. 1994

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

	▸ Water spray or fog. ▸ Foam. ▸ Dry chemical powder. ▸ BCF (where regulations permit). ▸ Carbon dioxide.

Special hazards arising from the substrate or mixture

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

Advice for firefighters

Fire Fighting	▸ Alert Fire Brigade and tell them location and nature of hazard. ▸ Wear full body protective clothing with breathing apparatus. ▸ Prevent, by any means available, spillage from entering drains or water course. ▸ Use fire fighting procedures suitable for surrounding area. ▸ **Do not** approach containers suspected to be hot. ▸ Cool fire exposed containers with water spray from a protected location. ▸ If safe to do so, remove containers from path of fire. ▸ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	▸ Combustible. ▸ Slight fire hazard when exposed to heat or flame. ▸ Heating may cause expansion or decomposition leading to violent rupture of containers. ▸ On combustion, may emit toxic fumes of carbon monoxide (CO). ▸ May emit acrid smoke. ▸ Mists containing combustible materials may be explosive. Combustion products include:, carbon dioxide (CO_2), hydrogen chloride, phosgene, hydrogen fluoride , nitrogen oxides (NOx), sulfur oxides (SOx), other pyrolysis products typical of burning organic material

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

Minor Spills	▸ 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	▸ Clear area of personnel and move upwind. ▸ 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. ▸ Stop leak if safe to do so. ▸ Contain spill with sand, earth or vermiculite. ▸ Collect recoverable product into labelled containers for recycling. ▸ Neutralise/decontaminate residue (see Section 13 for specific agent). ▸ Collect solid residues and seal in labelled drums for disposal. ▸ Wash area and prevent runoff into drains. ▸ After clean up operations, decontaminate and launder all protective clothing and equipment before storing and re-using. ▸ If contamination of drains or waterways occurs, advise emergency services.

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

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	▸ Limit all unnecessary personal contact. ▸ Wear protective clothing when risk of exposure occurs. ▸ Use in a well-ventilated area. ▸ When handling **DO NOT** eat, drink or smoke. ▸ Always wash hands with soap and water after handling. ▸ Avoid physical damage to containers.

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	▸ Use good occupational work practice. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS.
Other information	▸ Store in original containers. ▸ Keep containers securely sealed. ▸ Store in a cool, dry, well-ventilated area. ▸ Store away from incompatible materials and foodstuff containers. ▸ Protect containers against physical damage and check regularly for leaks. ▸ Observe manufacturer's storage and handling recommendations contained within this MSDS.

Conditions for safe storage, including any incompatibilities

Suitable container	▸ Lined metal can, lined metal pail/ can. ▸ Plastic pail. ▸ Polyliner drum. ▸ Packing as recommended by manufacturer. ▸ Check all containers are clearly labelled and free from leaks.
Storage incompatibility	▸ Avoid reaction with oxidising agents

PACKAGE MATERIAL INCOMPATIBILITIES

Not Available

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
AC Emporium 500 Seed Treatment	Not Available	Not Available	Not Available	Not Available

Ingredient	Original IDLH	Revised IDLH
fipronil	Not Available	Not Available
Ingredients determined not to be hazardous	Not Available	Not Available

Exposure controls

Appropriate engineering controls	General exhaust is adequate under normal operating conditions.
Personal protection	
Eye and face protection	▸ Safety glasses with side shields ▸ Chemical goggles. ▸ Contact lenses may pose a special hazard; soft contact lenses may absorb and concentrate irritants. A written policy document, describing the wearing of lenses or restrictions on use, should be created for each workplace or task. This should include a review of lens absorption and adsorption for the class of chemicals in use and an account of injury experience. Medical and first-aid personnel should be trained in their removal and suitable equipment should be readily available. In the event of chemical exposure, begin eye irrigation immediately and remove contact lens as soon as practicable. Lens should be removed at the first signs of eye redness or irritation - lens should be removed in a clean environment only after workers have washed hands thoroughly. [CDC NIOSH Current Intelligence Bulletin 59], [AS/NZS 1336 or national equivalent]
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 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. 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. 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. ▸ Neoprene rubber gloves
Body protection	See Other protection below

Continued...

Other protection	▸ Overalls. ▸ PVC Apron. ▸ PVC protective suit may be required if exposure severe. ▸ Eyewash unit. ▸ Ensure there is ready access to a safety shower.
Thermal hazards	Not Available

Recommended material(s)

GLOVE SELECTION INDEX

Glove selection is based on a modified presentation of the:
"Forsberg Clothing Performance Index".
The effect(s) of the following substance(s) are taken into account in the ***computer-generated*** selection:
AC Emporium 500 Seed Treatment Not Available

Material	CPI

* CPI - Chemwatch Performance Index
A: Best Selection
B: Satisfactory; may degrade after 4 hours continuous immersion
C: Poor to Dangerous Choice for other than short term immersion
NOTE: As a series of factors will influence the actual performance of the glove, a final selection must be based on detailed observation. -
* Where the glove is to be used on a short term, casual or infrequent basis, factors such as "feel" or convenience (e.g. disposability), may dictate a choice of gloves which might otherwise be unsuitable following long-term or frequent use. A qualified practitioner should be consulted.

Respiratory protection

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Viscous red liquid; mixes with water.

Physical state	Liquid	**Relative density (Water = 1)**	1.225
Odour	Not Available	**Partition coefficient n-octanol / water**	Not Available
Odour threshold	Not Available	**Auto-ignition temperature (°C)**	Not Applicable
pH (as supplied)	6.0-8.0	**Decomposition temperature**	Not Available
Melting point / freezing point (°C)	Not Available	**Viscosity (cSt)**	Not Available
Initial boiling point and boiling range (°C)	Not Available	**Molecular weight (g/mol)**	Not Applicable
Flash point (°C)	Not Applicable	**Taste**	Not Available
Evaporation rate	Not Available	**Explosive properties**	Not Available
Flammability	Not Applicable	**Oxidising properties**	Not Available
Upper Explosive Limit (%)	Not Applicable	**Surface Tension (dyn/cm or mN/m)**	Not Available
Lower Explosive Limit (%)	Not Applicable	**Volatile Component (%vol)**	Not Available
Vapour pressure (kPa)	Not Available	**Gas group**	Not Available
Solubility in water (g/L)	Miscible	**pH as a solution(1%)**	Not Available
Vapour density (Air = 1)	Not Available	**VOC g/L**	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

Inhaled	Inhalation of vapours or aerosols (mists, fumes), generated by the material during the course of normal handling, may produce toxic effects.

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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. The material may mimic the actions of the major inhibitory neurotransmitter of the brain, GABA, (gamma-aminobutyric acid) in inhibiting the electrical activity of certain elements of the nervous system. GABA is a putative amino-acid, produced within certain neurones (presynaptic cells) and is released into the synapse, between neurones, on the arrival of an action potential; GABA then interacts with post-synaptic neurones, slowing their rate of firing. Certain GABA congeners may produce lightheadedness, ataxia, mood elevation and muscle incoordination. Side-effects of uptake of GABA analogues and congeners (such as the isoxazole derivative, muscimol, isolated from hallucinogenic mushrooms), by neurones, may include dizziness, ataxia, euphoria, muscle twitches, and initial psychic stimulations followed by dream-filled sleep. More severe ingestions may produce visual disturbances, fever, confusion, myoclonus, mydriasis, seizures and coma. Residual headache may persist for several days. The congener muscimol is structurally related to GABA, crosses the blood-brain barrier easily, in contrast to GABA, and inhibits the firing of some central neurones. GABA, when introduced directly to the brain by injection (i.e. intrathecally), produces the same effect and similar outcomes to those produced by muscimol. Another amino-acid, with a similar structure to both GABA and muscimol, is ibotenic acid (also derived from mushrooms). Effects of ingestion are similar to those produced by muscimol. Ibotenic acid, however, binds to a different receptor, NMDA, which is normally activated by the putative neurotransmitter glutamic acid but now is inhibited by the action of ibotenic acid. NMDA receptors, in contrast to GABA receptors, when activated, normally cause neurones to fire. Systemic administration of ibotenic acid and muscimol to laboratory animals produces central inhibition of motor activity with little change to peripheral autonomic activity. Both compounds induce EEG changes in cats, rabbits and rats and thus within the central nervous system both compounds behave as false inhibitory neurotransmitters. GABA and its congeners inhibit the excitation of cells, of neurological origin, by allowing anions, such as chlorine, to enter the cell thus altering the electric potential of the cell. The GABA receptor acts as a gateway for influx of chloride ion. One subtype of receptor for GABA, the GABA-A receptor also contains binding sites for anxiolytic barbiturates, benzodiazepines, neurosteroids and, probably, ethanol. These anxiolytic groups potentiate the function of the chloride channels linked to the receptor. The whole receptor complex can be formed only by the interaction of several individual subunits, each of which is a membrane-spanning protein. Several different types of subunit have been identified and named the alpha-, beta-, and delta- subunits. The receptor may be made from any of up to five possible combinations of these subunits so that the number of possible subtypes of GABA-A receptor is huge and may, in part, explain their variable response to each anxiolytic agent. However, receptors made from any combination of two or three subunit types express much of the function of the native receptor.
Skin Contact	Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Skin contact with the material may produce toxic effects; systemic effects may result following absorption. 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	Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.
Chronic	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. Repeated or long-term occupational exposure is likely to produce cumulative health effects involving organs or biochemical systems.

	TOXICITY	IRRITATION
AC Emporium 500 Seed Treatment	Not Available	Not Available
fipronil	Dermal (Rabbit) LD50: 354 mg/kg	[** = Aventis]
	Dermal (Rat) LD50: >2000 mg/kg	Eye: slight *
	Dermal (rat) LD50: >2000 mg/kg **	Skin: non-irritating *
	Inhalation (Rat) LC50: >390 mg/m3	
	Inhalation (rat) LC50: 0.682 mg/M3/4h **	
	Oral (rat) LD50: 100 mg/kg *	
	Oral (Rat) LD50: 97 mg/kg	
	Not Available	Not Available

** Value obtained from manufacturer's msds*
unless otherwise specified data extracted from RTECS - Register of Toxic Effects of Chemical Substances

AC Emporium 500 Seed Treatment	For fipronil **Acute toxicity:** Clinical signs and symptoms reported after ingestion of fipronil by humans include sweating, nausea, vomiting, headache, abdominal pain, dizziness, agitation, weakness, and tonic clonic-seizures. Clinical signs of exposure to fipronil are generally reversible and resolve spontaneously Fipronil targets the nervous system. Signs of toxicity during an acute mouse feeding study with 87.4-97.2% fipronil included overactivity, irritability, convulsions, and death The primary metabolite of fipronil in army worms, mice, and humans is fipronil-sulfone, which binds to the GABA receptor with an affinity 6 times greater than the parent compound. Fipronil and its sulfone have similar toxicity in mammals; the mouse ip LD50 24 h after treatment is 41 and 50 mg/kg for fipronil and its sulfone, respectively. Fipronil-desulfinyl, the primary photoproduct in the environment, is 9-10 fold more potent and more acutely toxic than fipronil with an ip LD50 of 23 mg/kg in mice **Distribution:** After exposure fipronil is widely distributed in mammals and is found predominantly in fatty tissues. Rats given a single oral dose had the

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	highest concentrations of fipronil in the stomach, GI tract, fat, and adrenals. Moderate levels were found in the liver, pancreas, thyroid, and ovaries. Low levels were present in the muscle, brain, heart, and cardiac blood . A spot-on treatment study with 14C-fipronil on dogs and cats reported radioactivity 2 months after treatment concentrated in the sebaceous glands, epithelial layers surrounding the hairs, and exposed part of the hair shaft, suggesting the passive diffusion of fipronil in the sebum covering hair and skin . Researchers applied a spot-on fipronil product to dogs and vigorously petted them for 5 minutes every day with cotton gloves to mimic normal exposures to treated animals. Residues transferred to the gloves peaked at 589+/-206 ppm fipronil 24 h after treatment, decreased steadily over time (448 +/- 118 ppm after 8 days) , and were undetectable after 36 days **Absorption:** In an *in vitro* study of 14C-fipronil absorption through human, rabbit, and rat epithelial membranes, researchers recorded penetration rates after 8 hours of 0.08% (rat), 0.07% (rabbit), and 0.01% (human) of the dose of 200 g/L fipronil solution. Researchers reported greater absorption from a 0.2 g/L solution of fipronil, with 0.9% (rat), 13.9% (rabbit), 0.9% (humans) of the dose being absorbed **Metabolism:** The whole-blood half-life of fipronil in rats ranged from about 6.2-8.3 days after a single 4 mg/kg oral dose and decreased significantly to 2.1-2.3 days after a single 150 mg/kg oral dose. The primary metabolite of fipronil in animals is the fipronil-sulfone derivative. Researchers injected mice with fipronil and detected the sulfone derivative in the brain, liver, kidney, fat, and faeces . Fipronil-desulfinyl, the primary photodegradate of fipronil, has been measured in the fat, brain, liver, kidney, skin, and feces of mice, rats and lactating goats after oral exposure or injection . **Excretion:** Rats given an oral dose of fipronil excreted 45-75% in the faeces and 5-25% in the urine. The parent compound and the oxidation product, fipronil-sulfone, were present in both. **Chronic toxicity:** Signs of toxicity during a chronic rat feeding study included reduced feeding, reduced body weight gain, seizures (including seizures resulting in death), alterations in thyroid hormones, and alterations in the mass and function of the liver, thyroid, and kidneys. No signs of systemic toxicity (NOEL) were observed in rats ingesting 0.5 ppm (0.019-0.025 mg/kg/day) during a 52-week chronic dietary study. The lowest dosage at which effects were observed (LOEL) was 1.5 ppm (0.059 mg/kg/day males, 0.078 mg/kg/day females), and included increased incidence of seizures and death, alteration in clinical chemistry (protein), and alterations in thyroid hormones **Carcinogenicity:** Mice given fipronil in their diet for 2 years showed no evidence of carcinogenicity at doses of 30 ppm .• Researchers administered fipronil in the diet of rats for 2 years. Carcinogenicity was observed at 12.68 mg/kg/day in males and 16.75 mg/kg/day in females based on an increased incidence of clinical signs and alterations in clinical chemistry and thyroid parameters. In one study, rats were fed 0, 0.5, 2, 6, and 10 ppm (0, 0.025, 0.098, and 0.050 mg/kg/day males, and 0, 0.032, 0.13, and 0.55 mg/kg/day females) fipronil-desulfinyl (the primary photodegradate), for 2 years. Male rats at 10 ppm and female rats at 2, 6, and 10 ppm developed clinical signs of toxicity with no evidence of carcinogenicity (13). The US EPA classified fipronil as a Group C (possible human) carcinogen, based on increased thyroid follicular cell tumors in both sexes of rats. **Mutagenicity**: Fipronil did not cause mutations in human lymphocytes, Chinese hamster V79 cells, salmonella (Ames test), or mouse micronuclei **Reproductive and developmental toxicity:** In one study with rats, no observable effects were recorded at 30 ppm (2.54 mg/kg/day in males, and 2.74 mg/kg/day in females; route of exposure not included). The lowest dosage at which reproductive effects were recorded was 300 ppm (26.0 mg/kg/day in males and 28.4 mg/kg/day in females; route of exposure not included) based on clinical signs of toxicity, decreased litter size, decreased body weights, decrease in percentage of animals mating, reduction in fertility index, reduced post-implantation survival and offspring postnatal survivability, and delay in physical development. Other experimental studies with ingestion of fipronil have not reported significant alterations on animal development. There were no observable adverse effects within the limits of two studies performed using rats and rabbits. The Lowest Observable Adverse Effect Levels (LOAELs) were the highest doses tested: .20 and .1.0 mg/kg/day in rats and rabbits, respectively
FIPRONIL	For fipronil **Acute toxicity:** Clinical signs and symptoms reported after ingestion of fipronil by humans include sweating, nausea, vomiting, headache, abdominal pain, dizziness, agitation, weakness, and tonic clonic-seizures. Clinical signs of exposure to fipronil are generally reversible and resolve spontaneously Fipronil targets the nervous system. Signs of toxicity during an acute mouse feeding study with 87.4-97.2% fipronil included overactivity, irritability, convulsions, and death The primary metabolite of fipronil in army worms, mice, and humans is fipronil-sulfone, which binds to the GABA receptor with an affinity 6 times greater than the parent compound. Fipronil and its sulfone have similar toxicity in mammals; the mouse ip LD50 24 h after treatment is 41 and 50 mg/kg for fipronil and its sulfone, respectively. Fipronil-desulfinyl, the primary photoproduct in the environment, is 9-10 fold more potent and more acutely toxic than fipronil with an ip LD50 of 23 mg/kg in mice **Distribution:** After exposure fipronil is widely distributed in mammals and is found predominantly in fatty tissues. Rats given a single oral dose had the highest concentrations of fipronil in the stomach, GI tract, fat, and adrenals. Moderate levels were found in the liver, pancreas, thyroid, and ovaries. Low levels were present in the muscle, brain, heart, and cardiac blood . A spot-on treatment study with 14C-fipronil on dogs and cats reported radioactivity 2 months after treatment concentrated in the sebaceous glands, epithelial layers surrounding the hairs, and exposed part of the hair shaft, suggesting the passive diffusion of fipronil in the sebum covering hair and skin . Researchers applied a spot-on fipronil product to dogs and vigorously petted them for 5 minutes every day with cotton gloves to mimic normal exposures to treated animals. Residues transferred to the gloves peaked at 589+/-206 ppm fipronil 24 h after treatment, decreased steadily over time (448 +/- 118 ppm after 8 days) , and were undetectable after 36 days **Absorption:** In an *in vitro* study of 14C-fipronil absorption through human, rabbit, and rat epithelial membranes, researchers recorded penetration rates after 8 hours of 0.08% (rat), 0.07% (rabbit), and 0.01% (human) of the dose of 200 g/L fipronil solution. Researchers reported greater absorption from a 0.2 g/L solution of fipronil, with 0.9% (rat), 13.9% (rabbit), 0.9% (humans) of the dose being absorbed **Metabolism:** The whole-blood half-life of fipronil in rats ranged from about 6.2-8.3 days after a single 4 mg/kg oral dose and decreased significantly to 2.1-2.3 days after a single 150 mg/kg oral dose. The primary metabolite of fipronil in animals is the fipronil-sulfone derivative. Researchers injected mice with fipronil and detected the sulfone derivative in the brain, liver, kidney, fat, and faeces . Fipronil-desulfinyl, the primary photodegradate of fipronil, has been measured in the fat, brain, liver, kidney, skin, and feces of mice, rats and lactating goats after oral exposure or injection . **Excretion:** Rats given an oral dose of fipronil excreted 45-75% in the faeces and 5-25% in the urine. The parent compound and the oxidation product, fipronil-sulfone, were present in both. **Chronic toxicity:** Signs of toxicity during a chronic rat feeding study included reduced feeding, reduced body weight gain, seizures (including seizures resulting in death), alterations in thyroid hormones, and alterations in the mass and function of the liver, thyroid, and kidneys. No signs of systemic toxicity (NOEL) were observed in rats ingesting 0.5 ppm (0.019-0.025 mg/kg/day) during a 52-week chronic dietary study. The lowest dosage at which effects were observed (LOEL) was 1.5 ppm (0.059 mg/kg/day males, 0.078 mg/kg/day females), and included increased incidence of seizures and death, alteration in clinical chemistry (protein), and alterations in thyroid hormones **Carcinogenicity:** Mice given fipronil in their diet for 2 years showed no evidence of carcinogenicity at doses of 30 ppm .• Researchers administered fipronil in the diet of rats for 2 years. Carcinogenicity was observed at 12.68 mg/kg/day in males and 16.75 mg/kg/day in females based on an increased incidence of clinical signs and alterations in clinical chemistry and thyroid parameters. In one study, rats were fed 0, 0.5, 2, 6, and 10 ppm (0, 0.025, 0.098, and 0.050 mg/kg/day males, and 0, 0.032, 0.13, and 0.55 mg/kg/day females) fipronil-desulfinyl (the primary photodegradate), for 2 years. Male rats at 10 ppm and female rats at 2, 6, and 10 ppm developed clinical signs of toxicity with no evidence of carcinogenicity (13). The US EPA classified fipronil as a Group C (possible human) carcinogen, based on increased thyroid follicular cell tumors in both sexes of rats. **Mutagenicity**: Fipronil did not cause mutations in human lymphocytes, Chinese hamster V79 cells, salmonella (Ames test), or mouse micronuclei **Reproductive and developmental toxicity:** In one study with rats, no observable effects were recorded at 30 ppm (2.54 mg/kg/day in males, and 2.74 mg/kg/day in females; route of exposure not included). The lowest dosage at which reproductive effects were recorded was 300 ppm (26.0 mg/kg/day in males and 28.4 mg/kg/day in females; route of exposure not included) based on clinical signs of toxicity, decreased litter size, decreased body weights, decrease in percentage of animals mating, reduction in fertility index, reduced post-implantation survival and offspring postnatal survivability, and delay in physical development. Other experimental studies with ingestion of fipronil have not reported significant alterations on animal development. There were no observable adverse effects within the limits of two studies performed using rats and rabbits. The Lowest Observable Adverse Effect Levels (LOAELs) were the highest doses tested: .20 and .1.0 mg/kg/day in rats and rabbits, respectively ***[* The Pesticides Manual, Incorporating The Agrochemicals Handbook, 10th Edition, Editor Clive Tomlin, 1994, British Crop Protection Council]*** Negative in Ames and chromosome aberration tests. * ADI: 0.0002 mg/kg/day NOEL: 0.02 mg/kg/day technical fipronil

Acute Toxicity	✔	**Carcinogenicity**	⊘

Continued...

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

Legend: ✔ *– Data required to make classification available*
✘ *– Data available but does not fill the criteria for classification*
⊘ *– Data Not Available to make classification*

CMR STATUS

Not Applicable

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

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 fipronil
Solubilities Water (pH = 5): 0.0024 g/l (pH = 9): 0.0022 g/l
Melting Point (Technical Grade) : 195.5-203 C
Vapor Pressure : 3.7 x 10-4 mPa @ 25 C
Henry's Law Constant : 3.7 x 10-5 (Pa m3/mol)
log Kow = 4.01
Koc (average value) = 803
Hydrolysis Half-lives : @ 22 C: (pH 9.1) = 1,100 days, (pH 7.1) = 1,390 days : @32°C: (pH 9.1) = 11.3 days, (pH 7.1) = 15.6 days
Aqueous Photolysis : 4.1 Hours (pH 5.5)
Aerobic Aquatic Half-Life : 14.5 days
Soil Photolysis : 34 days
Field Dissipation Half-Life : 102-160 days
Aerobic Soil Half-life : 630-693 days
Anaerobic Soil Half-life : 123 days
Environmental fate:
Due to its very low vapor pressure and Henry's Law constant, fipronil is not likely to be found in the air. Fipronil is readily transformed into its desulfinyl photodegradate when exposed to sunlight. This photoproduct has a high affinity for insect GABA regulated chloride channels. Consequently, the photoproduct is neurotoxic toward insects.
Water: Laboratory data indicate that fipronil is much more susceptible to breakdown through photolysis rather than hydrolysis in water. Under environmental pHs fipronil is stable to hydrolysis with a half-life of 1390 days at pH 7.1 (22 C). The laboratory photolytic halflife was 4.1 hours; suggesting that photolysis is a more important pathway for the degradation of aqueous fipronil. Fipronil degrades rapidly in water when exposed to UV light to form fipronil-desulfinyl. Under these conditions, fipronil has a half-life of 4 to 12 hours . Fipronil-desulfinyl photodegrades in aerated and static water with recorded half-lives of 120+/-18 and 149 +/- 39 hours, respectively. Fipronil is stable to hydrolysis at pH 5 and pH 7. Hydrolysis of fipronil is only important at a very basic pH. The hydrolysis half-lives for pH of 12 and 9 in aqueous solutions were 0.1 and 32 days, respectively. Fipronil-amide is the primary residue formed from hydrolysis
A reported field half-life of fipronil under aerobic aquatic conditions was 14.5 days. In an aerobic metabolism study fipronil readily partitioned from the aqueous layer into the sediment, with most of the fipronil reaching the sediment layer within seven days after application.
Soil: The half-life of fipronil has been measured at 122-128 days in aerobic soils. Under aerobic conditions, naturally occurring soil organisms break down fipronil to form fipronil sulfone.
Fipronil can also be hydrolysed to form fipronil-amide Fipronil tends to dissipate by soil binding along with gradual microbial breakdown; however, on the soil surface photolysis may also be important. The major metabolite was the sulfide degradate. The extractable radioactive fipronil decreased from 99.46% of the applied dose to 4.07% at 60 days of incubation to non-detectable at 12 months. The major metabolite in anaerobic aquatic conditions was the sulfide degradate while both the amide and sulfone were products of aerobic soil conditions. Fipronil degrades on soil surfaces from ultraviolet radiation (i.e. sunlight) to form fipronil-desulfinyl, and has a measured half-life of 34 days in loamy soil. However, soil particles may prevent light from penetrating any significant depth under field conditions and increase residence time. There was no evidence of volatility of fipronil and its metabolites .
Fipronil has low mobility in soil and is not expected to leach into groundwater. After soil treatment, fipronil usually does not travel further than the upper 6 inches of soil, and significant lateral movement is not expected .Koc values for fipronil range from 427-1248 in sandy loam but will vary depending on clay and organic carbon content. The Koc is 3946 +/- 2165 for fipronil-sulfide and 2010 +/- 1370 for fipronil-desulfinyl
Fipronil and fipronil-desulfinyl are less volatile than water and can concentrate under field conditions
Air: The vapour pressure for fipronil is 3.7 x 10-4 mPa (25 C). Photodegradation studies in soil found no evidence of volatility of fipronil or its metabolites
Bioaccumulation and Bioconcentration: Fipronil accumulates in fish with a bioconcentration factor of 321 for whole fish, 164 for edible tissue, and 575 for non-edible tissue. Fish eliminated fipronil completely 14 days after being transferred to clean water. The primary metabolites in fish are fipronil-sulfone and -sulfide
When applied to water, fipronil varies greatly in its toxicity and potential to bioaccumulate in aquatic arthropods depending, on the species
Plants: Fipronil is not well absorbed by plants after soil treatment (about 5%) and partially degrades in plants to the sulfone and amide derivatives. Fipronil applied to foliage partially photodegrades to form fipronil-desulfinyl
Ecotoxicity:
Fipronil is highly toxic to bobwhite quail and pheasants.
Fipronil is highly to very highly toxic to marine and freshwater fish
Fipronil is highly toxic to honeybees by contact and ingestion when applied to plant foliage
Non toxic to earthworms.
Fish LC50 (96): rainbow trout 0.248 mg/l; bluegill sunfish 0.085 mg/l; sheepshead minnow 0.13 mg/l; Japanese carp 0.34 mg/l
In one study, male copepods reared in a 0.63 ug/L fipronil solution had a 75-89% decrease in reproductive success. Carry-over effects were significant for males (but not females) moved to clean seawater three days before mating
Mysid Shrimp LC50 (96 h): 140 ng/l
Exposure to less than 5.0 ng/L fipronil affected mysid growth, reproduction, and survival
Daphnia LC50 (48 h): 0.19 mg/l
Birds Acute LD50 mallard duck >2150 mg/kg; bobwhite quail 11.3 mg/kg; pheasants 31 mg/kg
Bird dietary LC50 (5-d): mallard duck >5,000 mg/kg; bobwhite quail 49 mg/kg
Chronic Toxicity
Invertebrate (Daphnia) Life Cycle NOEC: 0.0098 ppm
Mallard Reproduction NOEC: 1000 ppm
Bobwhite Reproduction NOEC: 10 ppm
Fish (Rainbow Trout) Early Life Stage NOEC: 0.0066 ppm
Fish (Rainbow Trout) Early Life Stage LOEC: 0.015 ppm
The degradation products of fipronil are high to highly acutely toxic to rainbow trout, bluegill sunfish, and freshwater invertebrates. The sulfone degradate is 6.3 times more toxic to rainbow trout, 3.3 times more toxic to bluegill sunfish, and 6.6 times more toxic to freshwater invertebrates. The sulfide degradate is 1.9 times more toxic to freshwater invertebrates. The sulfone degradate is very highly toxic to upland game birds and moderately toxic to waterfowl on an acute oral basis.
DO NOT discharge into sewer or waterways.

Persistence and degradability

Continued...

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

Bioaccumulative potential

Ingredient	Bioaccumulation
fipronil	MEDIUM (LogKOW = 4.0887)

Mobility in soil

Ingredient	Mobility
fipronil	LOW (KOC = 30930)

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	▸ Recycle wherever possible or consult manufacturer for recycling options. ▸ Consult State Land Waste Authority for disposal. ▸ Bury or incinerate residue at an approved site. ▸ Recycle containers if possible, or dispose of in an authorised landfill.

SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	
HAZCHEM	2X

Land transport (ADG)

UN number	2902
Packing group	III
UN proper shipping name	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains fipronil)
Environmental hazard	No relevant data
Transport hazard class(es)	Class: 6.1 Subrisk: Not Applicable
Special precautions for user	Special provisions: 61 223 274 Limited quantity: 5 L

Air transport (ICAO-IATA / DGR)

UN number	2902
Packing group	III
UN proper shipping name	Pesticide, liquid, toxic, n.o.s. * (contains fipronil)
Environmental hazard	No relevant data
Transport hazard class(es)	ICAO/IATA Class: 6.1 ICAO / IATA Subrisk: Not Applicable ERG Code: 6L
Special precautions for user	Special provisions: A3A4 Cargo Only Packing Instructions: 663 Cargo Only Maximum Qty / Pack: 220 L Passenger and Cargo Packing Instructions: 655 Passenger and Cargo Maximum Qty / Pack: 60 L Passenger and Cargo Limited Quantity Packing Instructions: Y642 Passenger and Cargo Limited Maximum Qty / Pack: 2 L

Sea transport (IMDG-Code / GGVSee)

UN number	2902

Continued...

Packing group	III
UN proper shipping name	PESTICIDE, LIQUID, TOXIC, N.O.S. (contains fipronil)
Environmental hazard	No relevant data
Transport hazard class(es)	IMDG Class: 6.1 IMDG Subrisk: Not Applicable
Special precautions for user	EMS Number: F-A , S-A Special provisions: 61 223 274 Limited Quantities: 5 L

SECTION 15 REGULATORY INFORMATION

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

fipronil(120068-37-3) is found on the following regulatory lists	"International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs","Australia Hazardous Substances Information System - Consolidated Lists"

SECTION 16 OTHER INFORMATION

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.
A list of reference resources used to assist the committee may be found at:
www.chemwatch.net/references

The (M)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.