



**Australian Government**  

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**Department of Health and Ageing**  
**Office of Chemical Safety**

## **TERMITE PROTECTION: AVAILABLE TREATMENTS AND HAZARD INFORMATION ABOUT TERMITICIDES**

### **Why are termites a problem in Australia?**

Termites (often incorrectly called 'white ants') feed on wood and serve an important function in nature by converting dead trees into organic matter. Unfortunately, the wood in buildings and other structures such as wooden power poles is equally appetising to termites, so they can cause serious damage which may be very expensive to repair. There are many species of termites in Australia, of which about 20 species can eat sound wood in buildings; those causing most damage to buildings are social insects that live in subterranean colonies that may contain up to 200, 000 individuals.

In order to maintain humidity and to protect themselves from extreme weather conditions, a colony (or nest) of subterranean termites may be up to 6–7 metres below the soil surface and have extensive tunnel networks that can extend up to 100 metres from the nest. In order to reach food sources above the soil surface, these subterranean termites construct mud tubes. These small mud tubes are often obscured by building structures and any infested wood is generally not obvious because termites take care to minimise damage to the outer surface in order to maintain the humidity of their tunnel networks.

Termites occur throughout Australia; however, they are most common in warmer areas of the country (the tropics, temperate coastal regions and inland) and somewhat less common in cooler regions (south eastern highlands and Tasmania).

### **How can buildings be protected against termites?**

Control techniques for termites can essentially be divided into two types, **prevention** and **treatment**. Preventative measures are easily applied during the construction of new buildings, but some (eg. stainless steel mesh, or a layer of granite chips) are not very suitable for existing buildings or structures.

#### **Prevention of infestation**

Building design can reduce the chances of termite damage. Important strategies include reducing the amount of timber used in buildings, a properly designed concrete slab with edges exposed for inspection for termite activity, or provision for easy under-floor inspections of timber floors. Installation of a reticulated system under the concrete slab can also to allow chemical barriers to be applied and re-applied whenever necessary.

Many local building authorities require that preventative measures against termites be incorporated during the construction of new buildings. Published Standards outline the

procedures that must be followed to protect new and existing buildings in Australia<sup>1</sup>. Since most termite-related damage to timber occurs from subterranean termites, preventative measures rely heavily on the establishment of barriers to stop the termites getting into the premises or timber from the underlying soil. Currently, two types of barriers are used, **chemical** or **physical**, often in combination.

### *Physical barriers*

**Metal shields, stainless steel mesh or granite chip barriers can all be used to stop termites getting into buildings.**

Termite shields (caps and strip shields) are installed on all substructures (isolated piers or posts and along walls, etc) to provide a continuous barrier.

Continuous sheets of fine stainless steel mesh can be installed under buildings during concrete slab construction. In certain situations, it may be adapted for service openings or wall cavities in existing structures.

Graded stone barriers are made up of a thick layer of small granite chips graded to a size and shape that cannot be transported by the termites and spaces between the particles are too small for termites to get through. Such stone barriers can be installed underneath a concrete slab or beneath a suspended floor. Such barriers are yet to be developed for tropical northern areas, which are inhabited by large termites (*Mastotermes darwiniensis*) that can make their way through the standard granite chip barrier.

### *Chemical barriers*

Chemicals that are used to kill termites are called **termiticides**. Termiticides have differing modes of action, and several methods are used to apply them.

For many new buildings, creation of a termiticide-treated layer of soil surrounding and under the building form an integrated barrier together with the physical methods described above. The termiticide is applied to the soil under the slab and around the footings, pipes, conduits and other structures of the house during construction to create a *vertical barrier*. Further loosened soil around the perimeter of the house, including around all pipes and service facilities, is treated during and after construction to form a *horizontal barrier*. Timber intended for use in the construction of houses, outbuildings, fences and other outdoor structures is often treated with chemicals by dipping and pressure or vacuum impregnation.

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<sup>1</sup> The Australian Standards relating to termite management are: AS 3660.1— 2000 *Termite Management – Part 1: New Building Work*; AS 3660.2 – 2000 *Termite Management – Part 2: In and Around Existing Buildings and Structures – Guidelines*; and AS 3660.3 – 2000 *Termite Management – Part 3: Assessment Criteria for Termite Management Systems*.

The termiticide used may repel termites, it may kill those that enter the treated area or attract the insects and then kill them, or it may be taken back to the nest on the bodies of the termites where it kills most of the colony by contact. With currently approved termiticides, an under-floor barrier may be effective for 4–10 years and an external barrier for 2–6 years, depending on climate, soil conditions and soil disturbance (protection lasts longer in the cooler southern regions).

To successfully complete termite barriers for existing buildings, strategic drilling through concrete slabs, porches, floors and wall footings may be needed, as well as under-floor treatment. This needs to be carefully worked out by a qualified pest control operator.

### ***Chemical baits***

Strategically placed bait stations can be used to attract termites with an attractive food such as dry wood or paper refuse. About 30 bait stations are needed for a typical house and they need to be inspected regularly to assess termite activity. Once the termites are attracted to the bait station, a particular termiticide is added. It is quickly spread through the colony by foraging termites due to their communal grooming activity and ultimately reaches and kills the queen. This external 'bait and treat' approach is not always possible in built-up areas as some space around a building is needed for placement of the bait stations.

### ***Some simple preventative measures***

Other simple prevention practices for existing buildings and other structures may also be helpful, such as:

- ensuring good under-floor ventilation, which discourages termite activity;
- not stacking timber against or near buildings;
- not building wooden in-ground structures (eg. untreated timber retaining walls) close to houses.

### **Treatment of infestation**

Treatment of a termite infestation in an existing structure also requires an integrated approach, including destruction of termites within the timber structures, measures to locate and destroy the termite nest, re-establishment of a chemical and/or physical barrier, and regular inspections to detect any ongoing or new termite activity. For existing buildings, where signs of infestation have been detected, chemical treatment is usually the only option for destroying termites and re-establishing a barrier.

Treatment with a termiticide directly into tunnels of the nest where termites are known to be active can reduce termite numbers but it rarely eliminates the colony altogether unless used in conjunction with another method.

## **What chemicals are used as termiticides?**

For many years, the prevention and treatment of termites relied heavily on the use of the so-called organochlorine insecticides, such as dieldrin, chlordane and heptachlor (known collectively as cyclodienes, because of their particular chemical structure). These chemicals had some advantages in that they provided 20 to 30 years of protection against termites because of their chemical stability, were extremely effective Australia-wide, and had no immediate adverse health effects at the levels of exposure arising from the approved use. However, these chemicals were largely withdrawn from use in Australia from 1995 because of concerns about their environmental persistence, their tendency to accumulate in the fat of animals and humans, and the potential for them to exhibit toxic effects as their levels built up in the environment through continued use.

The elimination of these chemicals has presented a number of challenges because the replacement chemicals do not provide such long-term protection. The need for more regular applications of the newer, less-persistent chemicals means that there is an increased chance that both householders and the pest control operators will be more frequently exposed to the chemicals.

In recent years there has therefore been much research into chemicals that have suitable persistence, low mammalian toxicity and minimal environmental effects. However, integrated approaches using physical and chemical methods have proved the most successful for prevention or treatment of termite damage.

A number of chemicals are currently approved in Australia as active constituents for use in termite control products. Separate inserts to this brochure outline some basic hazard information about each of the chemicals. For greater effectiveness, two different chemicals may be used in combination.

In the formulated products that are available for termite control, these active chemicals may be mixed with solvents, emulsifying agents or other components to aid application. These formulated products are sold under brand names but the chemical/generic name of the active constituent should always be shown on the label.

## **Are termiticide chemicals safe?**

Termiticides must be approved and registered by the Australian Pesticides and Veterinary Medicines Authority (the APVMA). Before any agricultural or veterinary chemical can be used in Australia, it undergoes a rigorous approval process, including an assessment for possible effects on human health (both of the public and of pest control operators who are to apply the chemical), and on the environment.

Issues related to toxicity and public health are assessed by the Commonwealth Department of Health and Ageing, issues related to health of pesticide applicators by the National Occupational Health and Safety Commission (NOHSC), and issues related to environmental safety, by Environment Australia (EA). Together with the APVMA, which assesses chemistry and efficacy issues, the combined work of these agencies forms what is known as the National Registration Scheme (NRS).

Thus, chemical companies wishing to market a pesticide or other chemical must generate a large number of studies to demonstrate that the chemical can be used safely, without causing unacceptable risks to users or the general public. As for other pesticides, the toxicology database for a termiticide involves toxicity tests performed in animals, and is normally very extensive.

In interpreting the toxicity data obtained in animals, it should be noted that toxicity tests generally use doses that are quite high relative to likely human exposures. The use of such high doses increases the likelihood that potentially significant toxic effects will be identified. Toxicity tests also reveal doses at which toxic effects are not observed, known as 'no observable effect levels' (NOELs). They are used, together with the application of generous safety margins, to establish acceptable limits for exposure in humans at which no adverse health effects would be expected. These limits are called the Acute Reference Dose (ARfD) and Acceptable Daily Intake (ADI), and are set for most pesticides to which humans may potentially be exposed.

In addition to testing the 'pure' active ingredient, acute toxicity tests are also conducted on the formulated products containing the active termiticide chemical, solvents, emulsifiers, deodorants etc. Acute toxicity tests determine how poisonous the products are after a single dose by mouth, skin or inhalation, and whether they cause skin and eye irritation or skin sensitisation (allergies).

### **What about smells that remain after treatment?**

Many householders are concerned about the odour that remains after their house has been treated. In the case of chlorpyrifos this may be due to the active constituent itself, which has a distinctive odour that may be noticeable after treatment, depending on the airflow and humidity in the location where it was applied. Chlorpyrifos and pyrethroids (alpha-cypermethrin, bifenthrin and permethrin) are usually formulated with volatile organic or petroleum solvents that may smell as they evaporate during and after application. Consequently, some manufacturers have marketed "low odour" termiticides. For example, to minimise the 'petroleum odour', a new bifenthrin product has been produced containing a different solvent to that previously used.

Some water-based products are available which do not leave any solvent odour after under-floor treatment (eg. the active ingredients, imidacloprid and fipronil are available in products formulated in water). However, adequate ventilation is still recommended with all these products.

Air monitoring studies have shown that if adequate ventilation is used, the concentrations of termiticides in buildings after the initial spraying period are very low.

## **What about pets and wildlife?**

The potential for chemicals used in preventing or treating termite infestation to harm domestic animals or wild animals (including birds, fish and beneficial insects) is assessed by the National Registration Scheme for Agricultural and Veterinary Chemicals (the NRS). Because termiticides may have the potential to cause harm to domestic animals and wildlife in the environment, it is important not to unnecessarily expose the environment, including contaminating areas around treated premises, streams, rivers or waterways with excess unused chemical or with waste washings from chemical containers. Licensed pest control operators (PCOs) should be trained in the correct disposal procedures.

## **Hazard information on termiticides**

Information on chemicals approved in Australia for use as a termiticide is given in the enclosed data sheets. The sheets outline the intrinsic characteristics of the chemicals (ie. whether they are hazardous or not). To reduce any potential health risks from chemicals, the aim is to reduce the likelihood of exposure. Since termiticides are commonly used around homes, the correct volume and method of application are important considerations to minimise any exposure of applicators, householders, or bystanders. For this reason, registered pest control operators who have completed an approved training course relating to the use and safe application of termiticides normally perform termiticide application.

A number of the chemicals currently approved for the control of termites have relatively low toxicity (eg. imidacloprid and hexaflumuron), that is, they are not considered to be hazardous chemicals. In terms of the application method, hexaflumuron baits are likely to pose very low risk but the method requires a high level of vigilance over many years, with frequent monitoring of the baits.

Pyrethroids are widely used in household insecticides and have a good safety record when used as directed.

Organophosphorus compounds such as chlorpyrifos are widely used in agriculture as effective insecticides, but need to be handled with caution because of their acute neurotoxicity in animals and humans.

Arsenic trioxide is a well-known hazardous chemical but there is only a low probability that the public will become exposed to it. Small amounts are gently puffed into termite passages with a hand blower to reduce an active infestation. It must be used at least two weeks before any other treatment as most other products repel termites, so that they would not be exposed to the arsenic.

Structural timber may be treated with a wide variety of insecticides. In addition to pyrethroids, timber may be impregnated with metal salts or organo-metals (based on arsenic, copper, chromium, tin or boron), fluorine, or creosote (a heavy petroleum hydrocarbon mixture sometimes referred to as coal tar). Fungicides may also be present in timber treatment products to retard decomposition of wood. Under normal conditions, the chemicals remain within the treated timber and there is little potential for the public to become exposed to them. However, treated timber is unsuitable for use in situations where it could make contact with food or drinking water, and requires care in handling to avoid human and environmental toxicity from the release of chemicals. People should work with treated wood outdoors, while wearing a dust mask, goggles and gloves. Sawdust or scrap wood should never be disposed of by mulching or composting and must not be burned, as toxic combustion products may be released. Arsenic and other heavy metals are especially hazardous when inhaled in smoke.

The enclosed hazard profiles give information about some of the **active ingredients** approved for termite control in Australia. Further details about the toxicity of the **formulated products** that contain these chemicals can be found on the relevant product containers.

### **Poisons classification in Australia**

In Australia, drugs and poisons are classified according to the 'Standard for the Uniform Scheduling of Drugs and Poisons', or SUSDP<sup>2</sup>. All termiticides are classified in Poisons Schedule 5, 6 or 7 according to their availability and requirements for safe handling, as follows:

**Schedule 5** Poisons of a hazardous nature that must be readily available to the public but require caution in handling storage and use. Labels on containers of S5 chemicals must bear the signal heading, 'Caution'.

**Schedule 6** Poisons that must be available to the public but are of a more hazardous or poisonous nature than those classified in Schedule 5. Labels on containers of S6 chemicals must bear the signal heading, 'Poison'.

**Schedule 7** Poisons that require special precautions in manufacturing, handling, storage or use, or special individual regulations regarding labelling or availability. Labels on containers of S7 chemicals must bear the signal heading, 'Dangerous Poison'.

To accommodate dilute products that are less toxic than the pure active ingredient, some chemicals have a 'cut-off' concentration. If a product contains less than the 'cut-off' concentration of the active ingredient, then it is placed in a lower schedule, or even exempted from scheduling.

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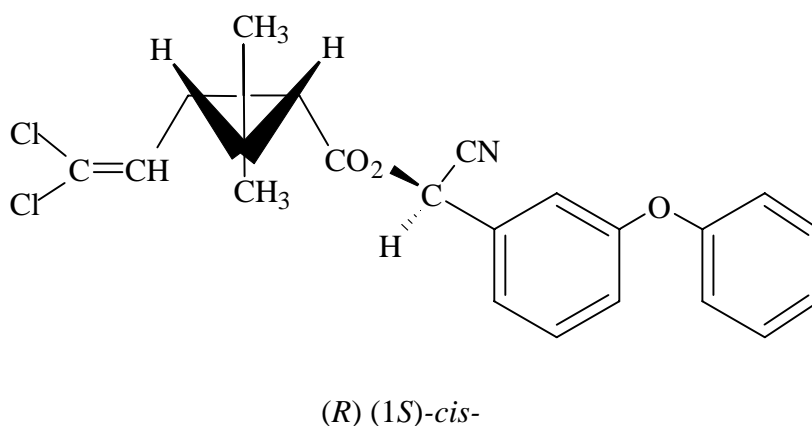
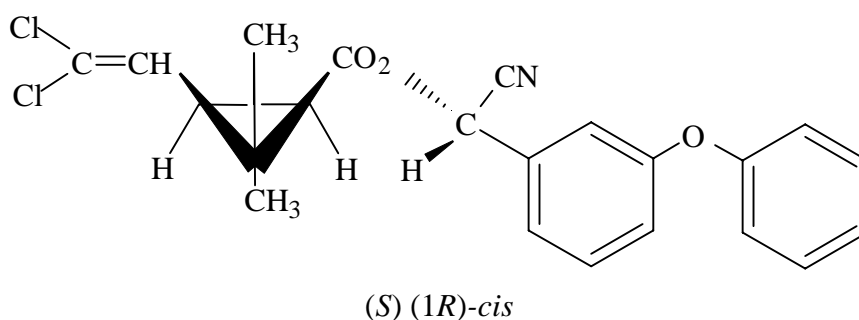
<sup>2</sup> This publication is a consolidation of recommendations made by the National Drugs and Poisons Schedule Committee, a national committee with State/Territory representation which meets on a regular basis.

## List of some chemicals approved in Australia for use in products to be used for controlling termites.

The following lists some chemicals (by their chemical, or generic name) which are used in Australia to control termites.

- Alpha-cypermethrin** a member of the pyrethroid class of chemicals which are synthetic analogues of the naturally occurring pyrethrums; it is used to form a barrier to repel or kill termites (see also deltamethrin, bifenthrin and permethrin).
- Deltamethrin** a synthetic pyrethroid similar to alpha-cypermethrin (see above); it is used in some termiticide products.
- Bifenthrin** another member of the pyrethroid class of chemicals; it is used to form a barrier to repel or kill termites.
- Permethrin** another synthetic pyrethroid, pyrethrin is commonly used as a barrier to repel or kill termites, and is also used for treatment of timber.
- Chlorpyrifos** a member of the organophosphorus class of chemicals that is used as a barrier to repel/kill termites.
- Chlorfluazuron** a member the benzoylurea class of chemical that inhibits the synthesis of chitin in insects. It is used in subterranean baiting stations to attract termites.
- Hexaflumuron** a member of the benzoylurea class of chemicals that inhibit chitin formation in insects. It is used in strategically placed bait stations to attract foraging termites, which transfer the chemical throughout the colony.
- Triflumuron** another benzoylurea insecticide, triflumuron is applied directly to termite nests.
- Imidacloprid** a member of the relatively new class of chemicals called chloronicotinyls. It is used to create a barrier or treated zone in the soil where it attracts termites, which die within the treated zone (partly from the effect of the chemical and partly from infection with fungi and other soil microorganisms).
- Fipronil** an extremely active insecticide belonging to the phenylpyrazole family, which has also been developed relatively recently. It is applied by spraying, trenching and soil rodding as a chemical soil barrier around existing structures, and may also be used to protect poles and fence posts.
- Arsenic trioxide** a compound used to directly kill termites in active passages (this method has variable effectiveness).

## ALPHA-CYPERMETHRIN



Alpha-cypermethrin

### Description and mode of action

Alpha-cypermethrin is a synthetic pyrethroid insecticide with a range of agricultural uses. It is also used to form a barrier to repel or kill termites (see also bifenthrin).

The synthetic pyrethroids are man-made chemicals similar in structure to the naturally occurring pyrethrums. Like other pyrethroids, alpha-cypermethrin kills insects by affecting the salt balance (sodium channels) in nerve cells. It has a broad spectrum of activity against insects with the main toxic effect on the nervous system.

### Toxicity

In mammals, alpha-cypermethrin is very toxic if swallowed. It is a type II pyrethrum, which means that it affects the central nervous system (brain and spinal cord) causing writhing, salivation and clonic convulsions (muscle spasms). These effects are reversible and pyrethroids have a good safety record. Alpha-cypermethrin is less likely to cause poisoning after contact with the skin, although facial skin contact may cause temporary facial numbness.

In laboratory animals, ingested alpha-cypermethrin is absorbed from the gastrointestinal tract. Most of the absorbed cypermethrin is broken down into other chemicals, and rapidly excreted in urine, with a small percentage (1%) stored in fat and excreted more slowly. It is poorly absorbed through the skin (only about 20% of the applied dose was absorbed when alpha-cypermethrin was applied to the skin of animals for four days).

Like other pyrethroids, alpha-cypermethrin can produce nerve damage. This can result in tremors, abnormal gait, agitation and difficulty in standing or walking. It may also cause vomiting and diarrhoea. If it is present in a water-based solution it is less toxic than when it is present in oils. It is less toxic by exposure on the skin, but signs of poisoning can include numbness or an itching or burning sensation on the skin. Alpha-cypermethrin is a mild eye irritant but minimally irritating to the skin. Some products containing alpha-cypermethrin may cause allergic reactions when applied on the skin (skin sensitisation).

Long term exposure to cypermethrin at high doses has produced changes in the liver and kidneys of experimental animals. These effects were not seen in animals fed lower doses. In some studies using very high doses in mice, alpha-cypermethrin produced benign (that is non-malignant, or unlikely to spread) tumours in the lungs. These effects were not seen in other species.

Alpha-cypermethrin was negative in a range of genotoxicity tests (that is, tests to assess its potential to damage the genetic material of cells and cause mutations or cancer). Alpha-cypermethrin did not cause birth defects or other reproductive problems in laboratory animals.

### **Poisons scheduling in Australia**

Alpha-cypermethrin products with a high concentration (more than 10% alpha-cypermethrin) are included in Schedule 7 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). Products with 1.5 to 10% alpha-cypermethrin are included in Schedule 6, while products at less than 1.5% are in Schedule 5. Schedule 5 chemicals are available for use in the home garden. Alpha-cypermethrin products for termiticide treatment around the home can only be applied by licensed pest-control operators who have been trained in their handling and use.

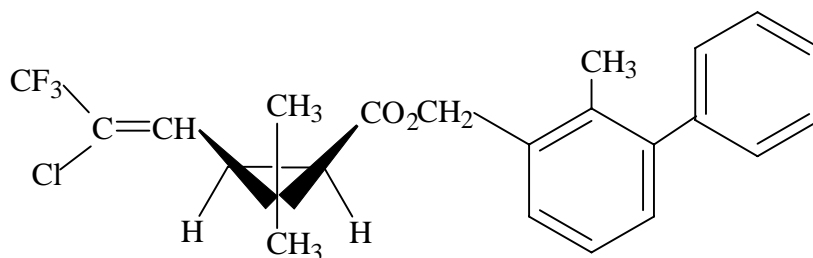
### **Ecological and environmental effects**

Alpha-cypermethrin is not soluble in water, and tends to bind to the soil meaning that it is unlikely to contaminate ground water. It breaks down more rapidly in sandy soils than in clay soils, and is also broken down by bacteria and light.

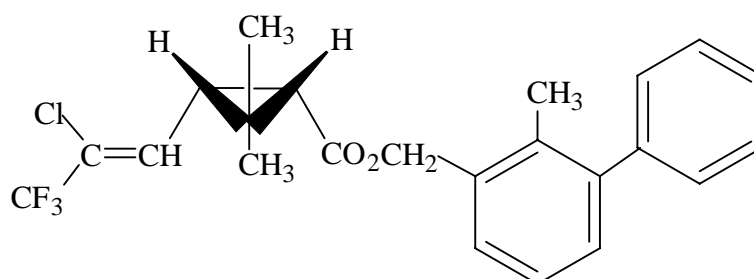
Alpha-cypermethrin is of low toxicity to birds, but is very toxic to fish and invertebrates which live in the water. Fish are more sensitive to alpha-cypermethrin because they break down and excrete the chemical much more slowly than birds or mammals. Alpha-cypermethrin is also highly toxic to bees.

**Note:** another type II pyrethrum chemical occasionally used in termiticide products is **deltamethrin**. Its physical and toxicological characteristics closely resemble those of alpha-cypermethrin. Deltamethrin is also in Schedule 7 with Schedule 6 and 5 classifications for some medium and low strength products.

## BIFENTHRIN



(Z)-(1R)-*cis*-



(Z)-(1S)-*cis*-

Bifenthrin

### Description and mode of action

Bifenthrin is a synthetic pyrethroid insecticide with a range of agricultural uses. It is also used to form a barrier to repel or kill termites (see also alpha-cypermethrin).

The synthetic pyrethroids are synthetic chemicals similar in structure to the naturally-occurring pyrethrums. Like other pyrethroids, bifenthrin kills insects by affecting the salt balance (sodium channels) in nerve cells. It has a broad spectrum of activity against insects with the main toxic effect on the nervous system.

### Toxicity

In mammals, bifenthrin is very toxic if swallowed. It is a type I pyrethrum (as compared with type II pyrethrums such as alpha-cypermethrin), which means that it mainly affects the peripheral nervous system causing tremors; it may also cause difficulties in walking. These effects are reversible and pyrethroids have a good safety record.

In laboratory animals, ingested bifenthrin is poorly absorbed from the gastrointestinal tract and is excreted unchanged in the faeces. It does not accumulate in the body after repeated doses. When applied to the skin, bifenthrin is of low toxicity, is not irritating and does not cause allergic reactions (skin sensitisation). However, bifenthrin is a slight eye irritant. Bifenthrin products are typically of low toxicity by inhalation.

After long-term exposure to high levels in the diet, mice developed benign (that is non-malignant, or unlikely to spread) tumours of the urinary bladder. These tumours are thought

to result from chronic inflammation of the bladder wall, and were not seen in other species, or at lower doses. Bifenthrin was negative in most genotoxicity tests (that is, tests to assess its potential to damage the genetic material of cells and cause mutations or cancer). Bifenthrin did not cause birth defects or other reproductive problems in laboratory animals.

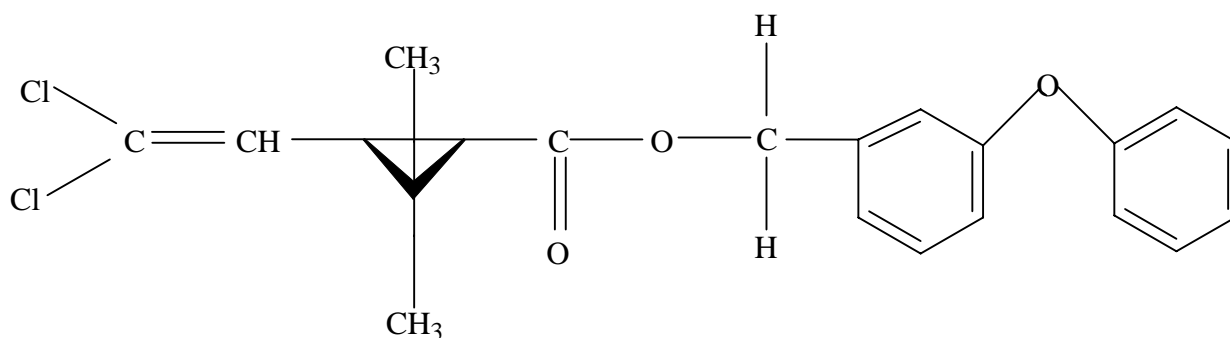
### **Poisons scheduling in Australia**

Bifenthrin is included in Schedule 7 of the Standard for the Uniform Scheduling of Drugs and Poisons, with products containing less than 10% included in Schedule 6. Products containing less than 0.5% bifenthrin are exempt from scheduling. Bifenthrin products for termiticide treatment around the home can only be used by licensed pest control operators who have been trained in their handling and use.

### **Ecological and environmental effects**

Bifenthrin is poorly soluble in water and exhibits strong soil binding properties and low mobility in soils. Neither plants nor animals metabolise bifenthrin extensively. These characteristics tend to make bifenthrin very stable in the environment. The chemical is highly toxic to fish, aquatic invertebrates and bees, but only slightly toxic to birds.

## PERMETHRIN



Permethrin (one of four isomers)

Permethrin is a synthetic pyrethroid insecticide, which has the same mode of action as other pyrethroids including alpha-cypermethrin and bifenthrin. Technical grade permethrin is a mixture of four isomers, known as trans-(*R*), trans-(*S*), cis-(*R*) and cis-(*S*) permethrin. The proportions of the isomers vary with the method of synthesis. The chemical has found widespread use in agricultural and veterinary settings, and forms the active constituent of numerous products that are applied as a barrier treatment to kill or repel termites. Often combined with anti-bacterial agents and fungicides, permethrin is also used in preservatives for timber, which may be treated by dipping or vacuum impregnation.

### Toxicity

Permethrin is classified as a type I pyrethrum that affects the central and peripheral nervous systems, causing tremors, hyper-excitability, salivation and paralysis. In rodents, a single high dose exposure to permethrin has been shown to cause degeneration in the peripheral nerves. However, these effects are reversible and pyrethroids have a good safety record. In mammals, permethrin is of moderate to low acute oral toxicity, influenced by the ratio of the trans and cis isomers in the chemical. The cis isomer is more toxic than the trans isomer. Permethrin has low dermal toxicity, is of very low toxicity by inhalation, is slightly irritating to the skin and eyes of rabbits and causes skin sensitisation in guinea pigs. Permethrin-based products tend to be of low toxicity but their irritation potential varies, ranging from nil to moderate depending on their strength and on the properties of other chemicals in the product. Similarly, some permethrin products cause allergic skin reactions whereas others do not. Information on human toxicity of permethrin is limited, but paraesthesia (numbness, itching, tingling and burning sensations) has been reported.

Following oral administration, permethrin is rapidly absorbed, distributed and excreted. The trans isomer is metabolised and excreted in the urine more readily than the cis isomer, which is more stable and tends to be eliminated in the faeces. A variety of metabolic products are formed. The trans isomer may deposit in adipose tissue and milk fat, although the extent of accumulation is very low. Permethrin is absorbed across the skin of experimental animals.

Short-, medium and long-term studies in laboratory species have revealed the liver is the main target organ of permethrin at moderate oral doses. Enlargement of the liver was seen, sometimes with activation of metabolic enzymes. Some studies showed that the effects were reversible if treatment was withdrawn. Neurological abnormalities occurred in dogs fed permethrin at high doses for at least a year. However, low doses of permethrin did not cause

any toxic effects. Permethrin does not damage genetic material and does not cause cancer, have adverse effects on reproduction, or cause deformities in the developing foetus.

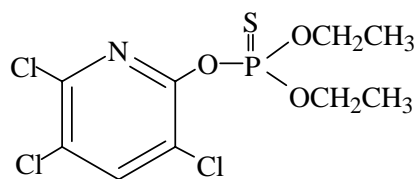
### **Poisons scheduling in Australia**

Permethrin is in Schedule 6 for preparations containing more than 25% of permethrin, Schedule 5 for preparations containing between 2 and 25% permethrin, and exempt from Scheduling when present at concentrations of 2% or less.

### **Ecological and environmental effects**

Permethrin is moderately stable in soil, to which it binds strongly and degrades with a half-life of 28 days or less. The decline of permethrin residues from crops is moderately fast, and it disappears rapidly from foliage and the soil surface when exposed to sunlight. Permethrin is of low mobility and shows little tendency to accumulate in the environment. Permethrin is of low oral toxicity to birds but highly toxic to insects, fish and aquatic arthropods such as crabs. However, permethrin's toxicity in the field is limited by its tendency to bind to sediment and degrade in sunlight.

## CHLORPYRIFOS



Chlorpyrifos

### Description and mode of action

Chlorpyrifos is an organophosphate insecticide that has widespread agricultural uses. It is also found in a number of insecticide products that are used in or around homes and gardens, including use as a termiticide. It has a mild mercaptan (sulphurous) odour, sometimes noticeable after treatment of buildings.

Like other organophosphate insecticides, chlorpyrifos kills insects by interfering with the activity of an enzyme (acetylcholinesterase) in the nervous system. This interference causes an increase in levels of the nerve transmitter chemical, acetylcholine, leading to over-stimulation of the nervous system and rapid twitching and paralysis of muscles.

### Toxicity

In mammals, the main signs of organophosphate poisoning are increased swallowing, excessive saliva, rapid breathing, pinpoint pupils, loss of coordination, excitement, twitching and rapid contractions of the neck and jaw muscles, coarse generalised body tremors, secretion of tears, urination, defecation, depression, prostration, convulsions, respiratory failure and death. The severity of signs increases with the amount of exposure, but there is an effective antidotal treatment for chlorpyrifos poisoning. Regardless of the route of exposure (oral, dermal or inhalation), the toxic effects of chlorpyrifos are similar.

In laboratory animals, ingested chlorpyrifos is rapidly absorbed from the gastrointestinal tract, but does not remain for long periods in the tissues or organs. It is broken down into other chemicals and excreted relatively quickly from the body, mostly in urine. Inhaled chlorpyrifos is also absorbed but relatively little is absorbed through skin. Chlorpyrifos is a very slight eye and skin irritant but does not cause allergic reactions when applied on the skin (skin sensitisation).

Long-term exposure to low concentrations of chlorpyrifos in the diet was without serious consequences in animal studies, although high concentrations caused similar symptoms to those listed above after single high doses. Both chlorpyrifos and its main metabolite gave negative results in a range of genotoxicity tests (that is, tests to assess its potential to damage the genetic material of cells and cause mutations or cancer). Similarly, exposure to chlorpyrifos did not cause cancer, reproductive problems or birth defects in experimental animals.

The possibility of chronic neurological effects after repeated exposures to low levels of organophosphate insecticides has been investigated. Health studies in workers producing and

packaging chlorpyrifos products have not shown any differences in the levels of illness or diseases compared with a matched control group not exposed to chlorpyrifos.

### **Air monitoring studies**

Air monitoring studies have shown that if adequate ventilation is used, the concentration of termiticides in buildings after the initial spraying period is very low. For example, in 1992 the WorkCover Authority of New South Wales monitored chlorpyrifos concentrations in the air of seven Sydney houses that had been sprayed in the under-floor area (the treatment procedure most likely to cause the highest concentrations of the termiticide). Average chlorpyrifos concentrations in the houses did not exceed 1 microgram per cubic metre ( $\mu\text{g}/\text{m}^3$ ). Breathing air at this concentration is calculated to give an exposure that is approximately 200-fold less than the lowest dose of chlorpyrifos that causes significant reduction in plasma cholinesterase activity in humans (the most sensitive marker of exposure).

### **Poisons scheduling in Australia**

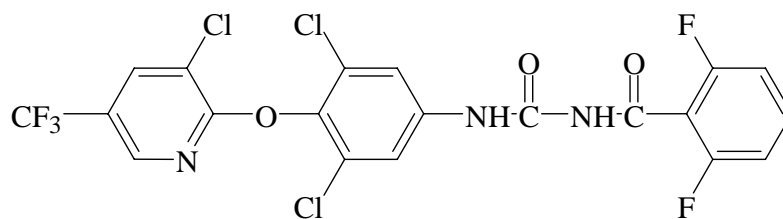
Chlorpyrifos products with high concentrations of the active ingredient are listed in schedule 6 of the Standard for the Uniform Scheduling of Drugs and Poisons (SUSDP). Termiticide treatments around the home are restricted to licensed pest-control operators who have been trained in the handling and use of these products. A schedule 5 classification exists for preparations containing 5% or less of chlorpyrifos, controlled release granular preparations containing 10% or less, and for microencapsulated chlorpyrifos when present at 20% or less in aqueous preparations. Potting or soil mixtures containing  $100 \text{ g}/\text{m}^3$  or less are exempt from scheduling.

### **Ecological and environmental effects**

Chlorpyrifos does not dissolve easily in water. It is strongly adsorbed by most soils and is relatively immobile in the soil. The half-life of chlorpyrifos in the soil has been shown to range from 11 to 141 days depending on the soil type; it is thus considered to be moderately persistent. Chlorpyrifos was least persistent in soils with high pH values. Soil micro-organisms break down chlorpyrifos. Chlorpyrifos is hydrolysed at a moderate rate. Based on data from available studies, chlorpyrifos is unlikely to leach into ground water in measurable quantities under most typical use scenarios.

Chlorpyrifos is moderately to very highly toxic to birds and bees when exposed to direct treatment. Terrestrial non-food application of chlorpyrifos to sites such as turf represents an acute hazard to birds. Run-off from such applications could be hazardous to fish and aquatic invertebrates, as chlorpyrifos is highly toxic to aquatic organisms. Various uses of chlorpyrifos may pose a risk to small birds and small mammals.

## CHLORFLUAZURON



Chlorfluazuron

### Description and mode of action

Chlorfluazuron is a benzoyl phenyl urea insecticide, which requires ingestion by target pests to be efficacious. It is an insect growth regulator inhibiting the synthesis of chitin, a vital part of the exoskeleton of insects. Chlorfluazuron disrupts the formation of chitin at the time of moulting, resulting in the failure of the termite to complete the moulting process, and eventually kills the affected termites. Chlorfluazuron is found in several insecticide products used in subterranean termite baiting stations.

### Toxicity

In laboratory animals, chlorfluazuron had very low acute toxicity if ingested, inhaled or exposed on the skin. It was slightly irritating to the eyes but did not cause skin irritation or allergic reactions when applied to the skin. Chlorfluazuron was absorbed only to a limited extent when swallowed and excreted mainly in the faeces.

Short and long-term exposure to low concentrations of chlorfluazuron in the diet was without serious consequences in animal studies. The only consistent finding was an increase in cholesterol levels. Chlorfluazuron did not cause birth defects in laboratory animals.

There is evidence to indicate that chlorfluazuron persists and bio-accumulates in the body. In rats, high levels of chlorfluazuron residues were deposited in fat and depletion was slow, with a depletion half-life of approximately 42 days. Goats and hens secreted chlorfluazuron into milk and egg yolk, respectively. If given in the diet of food-producing animals, there is a significant potential for accumulation and retention of residues in the fat and for excretion in milk.

As chlorfluazuron is used in subterranean termite baiting stations, public exposure is unlikely to occur. However, due to its persistent and bio-accumulative nature, care needs to be taken not to misuse the chemical.

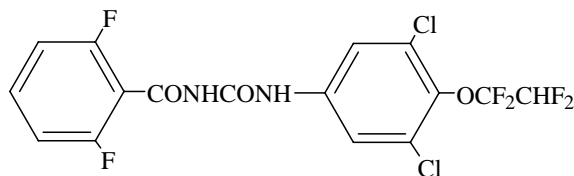
### Poisons schedule in Australia

Chlorfluazuron has been considered by the National Drugs and Poisons Schedule Committee and is currently exempt from poisons scheduling due to its low toxicity.

## **Ecological and environmental effects**

Chlorfluazuron partitions primarily to the soil/sediment compartments where it adsorbs strongly to soil organic matter. It is moderately persistent in soil and movement to watercourses in runoff must be avoided. It is highly toxic to aquatic arthropods such as daphnia due to its chitin inhibiting properties, but is of low toxicity to mammals, birds, bees, fish, soil micro-organisms and the earthworms.

## HEXAFLUMURON



Hexaflumuron

### Description and mode of action

Hexaflumuron is a chemical of the benzoylurea class, which regulates insect growth by inhibiting chitin (outer skeleton) formation.

### Toxicity

In laboratory animals, hexaflumuron has very low acute toxicity if ingested, inhaled or exposed on the skin. It is not irritating to the skin or eyes and does not cause allergic reactions when applied on the skin (skin sensitisation). Hexaflumuron is absorbed only to a limited extent when swallowed, is metabolised extensively, and excreted rapidly via the urine and faeces.

Short- and long-term exposure to low concentrations of hexaflumuron in the diet was without serious consequences in animal studies. High doses produced some damage to the liver, oxidation of haemoglobin, and elevated red blood cell production in the spleen. It did not cause birth defects.

As hexaflumuron is mainly used in bait stations, significant public exposure is unlikely to occur.

### Poisons scheduling in Australia

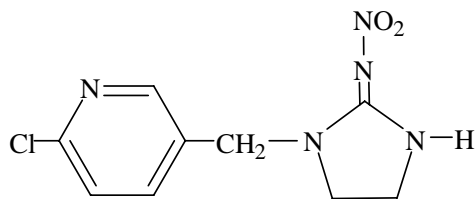
As a reflection of its low toxicity, hexaflumuron is currently exempt from poisons scheduling.

### Ecological and environmental effects

Hexaflumuron is strongly adsorbed by a wide range of soils. It is highly toxic to aquatic insects under field conditions, but is of low toxicity to bees and birds.

**Note: Triflumuron**, another benzoylurea chemical, has closely similar toxicological characteristics to hexaflumuron, and is applied directly to termite nests when in a powder formulation. Triflumuron is in Poisons Schedule 5.

## IMIDACLOPRID



Imidacloprid

### Description and mode of action

Imidacloprid belongs to a relatively new class of nitromethylene chemicals called chloronicotinyls, which kill insects by blocking nerve impulses.

### Toxicity

In mammals, imidacloprid is broken down to a compound that has toxic effects on the nervous system. This compound is either broken down and excreted, or converted to a protein that is used in the body. The toxic effects on the nervous system may include apathy, reduced muscle tone, tremors and, in extreme cases, muscle cramps and difficulties in breathing due to effects on the muscles associated with respiration.

In laboratory animals, ingested imidacloprid is rapidly absorbed from the gastrointestinal tract. Around 96% of an ingested dose is excreted within two days (predominantly in the form of metabolites) in urine, with some excretion in the faeces. Ingested imidacloprid is moderately toxic but even large doses are of low toxicity when applied to skin. Dust formulations of imidacloprid are of low toxicity when inhaled. Imidacloprid is not irritating to the eyes or skin and does not cause allergic reactions when applied to the skin (skin sensitisation). Some imidacloprid products may contain clay as a binding agent, which can irritate the eyes.

Long-term exposure to low concentrations of imidacloprid in the diet was without serious consequences in animal studies. At high doses, bodyweight loss, tremors and evidence of liver toxicity were seen. There was also premature ageing in the thyroid of rats.

Imidacloprid did not cause cancer in mice or rats. Similarly, it did not affect reproduction or cause birth defects. It was negative in most genotoxicity tests (that is, tests to assess its potential to damage the genetic material of cells and cause mutations or cancer), although it caused chromosome damage in cultured cells. However, tests for chromosome damage in animals were negative.

### Poisons scheduling in Australia

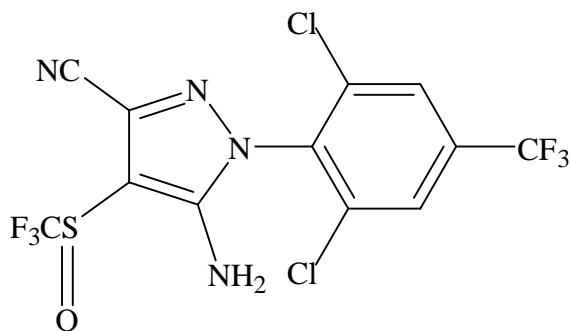
Imidacloprid is in Schedule 6 of the Standard for the Uniform Scheduling of Drugs and Poisons. Products containing 20% or less of imidacloprid are included in Schedule 5, and those containing 5% or less are exempt from scheduling.

## **Ecological and environmental effects**

Imidacloprid has quite a long half-life in soil (from about 6 weeks up to about 6 months). It is broken down more quickly in soils with plant cover than in bare earth. Imidacloprid is moderately soluble in water with a half-life of greater than 31 days, and can bind to organic matter in soils. In some soils that are very porous or gravelly, there may be potential for imidacloprid to move into ground water.

Imidacloprid is toxic to birds and highly toxic to bees by direct application. In studies where birds were allowed to eat seed treated with imidacloprid, they had short-term toxic effects including retching and loss of coordination. They recovered rapidly, and learnt to avoid treated seed. Imidacloprid is of moderately low toxicity to fish, but may be highly toxic to aquatic invertebrates. It is unlikely that there will be significant effects on birds and non-target insects from soil treatment if care is taken during application.

## FIPRONIL



Fipronil

### Description and mode of action

Fipronil belongs to a relatively new class of chemicals called phenylpyrazoles, which kill insects by blocking nerve impulses. The chemical has a stronger binding affinity within the nervous system of insects than in animal nerves, and therefore has enhanced toxicity to insects.

### Toxicity

In laboratory animals, there is moderately extensive absorption of ingested fipronil from the gastrointestinal tract. Once absorbed, fipronil is metabolised rapidly but is excreted slowly, as the metabolism products tend to become deposited in the body fat. The major route of excretion is the faeces. Fipronil is moderately toxic by ingestion and inhalation, and in laboratory animals causes abnormal gait and posture, ruffled fur, lethargy, tremors and convulsions. However, fipronil is poorly absorbed across the skin and is therefore of low toxicity when applied dermally. Fipronil does not cause skin or eye irritation, but some fipronil-based products are skin and eye irritants due to the presence of other chemicals in the formulations. Fipronil may cause skin sensitisation in sensitive individuals.

In long-term feeding studies with fipronil, the main effects in laboratory animals were decreased weight gain and degenerative lesions in the liver. Convulsions and other signs of nervous system toxicity occurred at high doses. In rats, fipronil caused a disturbance of thyroid hormone regulation, leading to an increased incidence of benign tumours of the thyroid gland. However, rats are highly sensitive to this condition and fipronil is very unlikely to pose a carcinogenic hazard to humans when used as a termiticide. Fipronil did not cause mutations, damage genetic material or have adverse effects on foetal development, but did impede the growth and survival of rat pups at high doses.

### Poisons scheduling in Australia

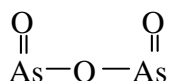
Fipronil is included in Schedule 6 of the SUSDP, with cut-offs to Schedule 5 when present in preparations at 10% or less, and is unscheduled in products containing 0.05% or less.

## **Ecological and environmental effects**

Fipronil is an extremely active insecticide and is highly toxic to birds, fish and aquatic invertebrates. It is stable in water in the dark but rapidly breaks down in the presence of light. Laboratory and field studies have demonstrated that fipronil is also broken down by light when on plant and soil surfaces. The main degradation products retain high insect and non-target organism toxicity. Fipronil and its degradation products are relatively immobile in soil, and therefore the risk of residues leaching into groundwater is considered to be low.

## ARSENIC TRIOXIDE

Arsenic trioxide



### Description and mode of action

Arsenic is highly toxic following both short-term exposure to high doses and long-term exposure to lower doses. However, for termite control, arsenic is generally used in bait stations, or in direct application to termite nests. In both of these situations, the potential for exposure of the public is limited.

### Toxicity

Arsenic is very poisonous if ingested in large doses, with signs including vomiting, diarrhoea and stomach cramping.

In laboratory animals arsenic is quite well absorbed from the gastrointestinal tract following ingestion and is mostly excreted in the urine (up to 80%), with less than 20% in the faeces. The arsenic is combined with other compounds by the body to aid in urinary excretion. On long-term administration, arsenic can build up in the liver, kidneys and lungs.

There is some evidence that a certain level of arsenic is required for normal body functioning. However exposure to increased levels can cause a range of problems. Long-term exposure to increased arsenic levels has occurred due to naturally high arsenic levels in the water supply or contamination following mining activities. Long-term ingestion of relatively high levels of arsenic causes thickening of the skin and changes in the pigmentation. Arsenic has also been associated with increased cancer of the liver, lung, skin, bladder and kidneys. It is believed that exposure to arsenic after exposure to an agent which is known to cause cancer (such as ultraviolet light or cigarette smoke) can increase the chance of developing cancer.

Arsenical chemicals are genotoxic in cultured bacterial and mammalian cells and in insects. Mutagenic effects have been identified, together with damage to chromosomes, inhibition of DNA repair and interference with cell division. These chemicals can also increase the effect of other DNA-damaging agents. Workers exposed to arsenic trioxide in a smelter showed increased mortality from tuberculosis, respiratory cancer, heart disease and emphysema.

Arsenic is teratogenic (causes birth defects) in experimental animals. In a number of human studies where there was enough arsenic exposure to cause changes in the skin, there was some evidence of increased numbers of spontaneous abortions and babies being stillborn. These effects were only seen at these relatively high levels, with lower levels of arsenic producing no problems with mammalian development.

### Poisons scheduling in Australia

Like all arsenic-containing chemicals, arsenic trioxide is a very hazardous chemical. Only very small amounts (several grams) are used by licensed pest control operators. It is classified as a Schedule 7 poison.

## **Ecological and environmental effects**

Inorganic arsenic compounds are moderately toxic to fish and aquatic invertebrates. However, they are highly toxic to algae, and low concentrations may have considerable impact on aquatic ecosystems. Inorganic arsenic trapped in sediments may be released into ground water over long periods of time. Biological transformations result in the production of less toxic organic arsenic compounds, but soil pollution resulting in ground water contamination has proved to be a serious human health problem in some parts of the world. Although persistent and taken up by plants and other organisms, inorganic arsenic is not subject to significant biomagnification in food chains.

Information prepared for the Department of Health and Ageing by the Chemicals Review and International Harmonisation Section, Office of Chemical Safety, Therapeutic Goods Administration, and by Dr Janet Salisbury of Biotext, Canberra.