

# Health-Based Investigation Level for Bifenthrin in Soil



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# Health-Based Investigation Level for Bifenthrin in Soil

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## 1 INTRODUCTION

Bifenthrin is a synthetic pyrethroid contact insecticide and acaricide used in a variety of crops, stored grain as a protectant and as a pre-construction termite barrier. It has some structural similarities to cypermethrin, tetramethrin and permethrin but is characterised by greater photostability and insecticidal activity. Bifenthrin affects the nervous system and causes paralysis in insects (Extoxnet, 1995).

## 2 PROPERTIES AND USES

### 2.1 PHYSICAL AND CHEMICAL PROPERTIES

Bifenthrin (C<sub>23</sub>H<sub>22</sub>CCF<sub>3</sub>O<sub>2</sub>) is a light brown substance with an aromatic odour. It is soluble in methyl chloride, chloroform, acetone, ether and toluene and is slightly soluble in heptane and methanol (Patty, 2001). It has a moderate to low vapour pressure and has very low solubility in water (Fecko, 1999).

The physical and chemical properties of bifenthrin are listed below.

CAS No	82657-04-3
Molecular weight	422.9
Melting point	68-70.6 °C
Flash point	65°C (open cup)
Vapour pressure (mmHg at 25 °C)	1.81 x 10 <sup>-7</sup>
Solubility water	0.1 mg/L
Octanol/water partition Coefficient (log Kow)	> 6.00

Source: Patty (2001)

## 3 EXPOSURE STANDARDS

### 3.1 ENVIRONMENTAL STANDARDS

There are currently no environmental standards for bifenthrin in Australia. Relevant tolerable intakes are listed below.

Organisation	Regulatory value	Dose (mg/kg/day)
Joint FAO/WHO Meeting on Pesticide Residues (JMPR)	Acceptable Daily Intake (ADI)	0.02
Therapeutics Goods Administration	ADI	0.01
US EPA	Reference Dose (RfD)	0.015

### **3.2 OCCUPATIONAL STANDARDS**

There is currently no exposure standard for any specific pyrethroid insecticide but the National Occupational Health and Safety Commission (NOSCH) has an exposure standard of 5 mg/m<sup>3</sup> for pyrethrum or pyrethrins.

### **3.3 OTHER APPLICABLE GUIDELINES**

An Australian drinking water guideline for bifenthrin or synthetic pyrethroids has not been set.

## **4 ENVIRONMENTAL BEHAVIOUR, OCCURRENCE AND DISTRIBUTION**

### **4.1 SOIL**

Bifenthrin is relatively immobile, as it binds strongly to soil. Its half-life in soil ranges from 122 to 345 days depending on the soil type and amount of air in the soil (US EPA, 1999). Photodegradation is not an important route of degradation; bifenthrin is the most persistent of the synthetic pyrethroids in field studies (US DA, 1999; US EPA, 1999). When released to soil, bifenthrin is expected to have no mobility based upon a range of K<sub>oc</sub> values from 131,000 to 302,000 (US DA, 1999).

### **4.2 WATER**

Bifenthrin is a non-polar molecule with a high octanol-water coefficient and therefore has a low solubility in water. It has a moderate to low reactivity towards aqueous photolysis with the photolytic half-life ranging from 5 days to 600 days. Volatilisation from water surfaces is thought to be attenuated by adsorption to suspended solids and sediment in the water column (Fecko, 1999).

### **4.3 AIR**

Bifenthrin has a vapour pressure of  $1.80 \times 10^{-7}$  mm Hg at 25°C and will exist in both the vapour and particulate phases in the ambient atmosphere. It is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals or photochemically-produced ozone with a half-life of between 13 hours and 7 days (US DA, 1999).

## **5 ABSORPTION, DISTRIBUTION, METABOLISM AND EXCRETION**

The metabolic pathways for the breakdown of the pyrethroids vary little between mammalian species but vary somewhat with structure (Hayes and Laws, 1991). In summary, pyrethrum and allethrin are broken down mainly by oxidation of the isobutenyl side chain of the acid moiety and of the unsaturated side chain of the alcohol moiety with ester hydrolysis playing an unimportant part. For the other pyrethroids, including bifenthrin, ester hydrolysis predominates. These reactions can take place in both the liver and plasma and are followed by hydroxylation and conjugation to glucuronides or sulfates, which are excreted in the urine.

In a number of studies in hens orally exposed to labelled bifenthrin, 40-50% of bifenthrin was excreted unchanged and 20-40% stored in the tissues in the forms of fatty acid conjugates (palmitic acid or oleic acid) and unconjugated metabolite hydroxylmethyl-bifenthrin (Tullman, 1987; Singer, 1987 as cited JMPR, 1992). Metabolism in hens is via

hydroxylation of the 2-methyl carbon of the cyclopropane ring followed by acid conjugation (Wu, 1987 as cited JMPR, 1992).

In the rat it has been demonstrated that after oral exposure to bifenthrin 90% is excreted in seven days, mainly via the faeces. In that study bifenthrin residues were detected in the liver, fat and skin with the total residue in tissues estimated to be at about 3% (El Nagger, 1983 as cited JMPR, 1992). Total absorption in rats is estimated to be 50% in females and 36% in males (El Nagger 1991, as cited in JMPR, 1992). The major metabolites in rats were the parent compound, the hydrolysis product 2-methyl-3-phenyl benzoic acid and the oxidised hydrolytic product 2 methyl-3-phenyl benzyl alcohol (El Nagger, 1991 as cited in JMPR, 1992).

## 5.1 MODE OF ACTION

Bifenthrin delays the closure of sodium channels of nerve cell endings. Bifenthrin holds the activation gate that depolarises the presynaptic terminals in the open position, effectively paralysing the organism by severely limiting neurotransmission (Extoxnet, 1995). However according to Hayes and Laws (1991) interference with sodium channels is not the only mechanism of action proposed for the pyrethroids. Observed effects on the central nervous system (CNS) have led researchers to suggest actions such as antagonism of gamma-aminobutyric acid (GABA)-mediated inhibition, modulation of nicotinic cholinergic transmission, enhancement of noradrenaline release, and actions on calcium ions (Hayes and Laws, 1991). Since neurotransmitter specific pharmacological agents offer only poor or partial protection against poisoning, it is unlikely that any one of these effects represents the primary mechanism of action of the pyrethroids and most neurotransmitter release is secondary to increased sodium entry. Specific information on other effects of bifenthrin on the CNS was not available.

## 6 TOXICOLOGY

Bifenthrin has a moderate acute toxicity and is classified as moderately hazardous (JMPR, 1992). Predominant clinical signs in animals are clonic convulsions, tremors and oral discharge.

Species	Sex	Route	LD <sub>50</sub> mg/kg/bw	Reference
Mouse	M, F	Oral	43	Freeman (1983)
Rat	M, F	Oral	56	Freeman <i>et al.</i> (1982)
Rabbit	M, F	Intraperitoneal Dermal	799	Freeman <i>et al.</i> (1983a)
			> 2000	Freeman <i>et al.</i> (1983b)

Source: JMPR (1992)

There is no evidence of carcinogenicity, reproductive toxicity or genotoxicity (JMPR, 1992).

Bifenthrin is not a dermal irritant and does not cause delayed neurotoxicity in rats or hens (Algate *et al.*, 1985; Roberts *et al.*, 1984; as cited JMPR, 1992).

There is very limited information available on the toxicity of bifenthrin in humans (Kreger, 2001). The diagnosis of toxicity in humans from bifenthrin is often confused with

toxicity from other pesticides or solvents for two reasons: firstly, bifenthrin toxicity has similarities to anticholinesterase pesticide intoxication; and secondly, the most common formulations of bifenthrin are with solvents.

The relative resistance of mammals to the pyrethroids is almost wholly attributable to their ability to hydrolyse the pyrethroids rapidly to their inactive acid and alcohol components, since direct injection into the mammalian CNS leads to susceptibility similar to that seen in insects (Hayes and Laws, 1991).

### 6.1 ACCEPTABLE DAILY INTAKE (ADI)

The Therapeutic Goods Administration has set an ADI of 0.01 mg/kg/day for bifenthrin. The ADI was established based on the no observed effect level (NOEL) of 1.5 mg/kg/day and the application of the uncertainty factor of 100 to account for interspecies and intraspecies differences. The critical study relevant to the establishment of the ADI was a year long dietary study on dogs by Serota *et al.* (1985, as cited in JMPR, 1992). In this study dose-related tremors appeared at 3 and 5 mg/kg/day after 15 weeks of treatment. The toxicological endpoints used are consistent with the adverse effects observed in a number of animals after exposure to bifenthrin.

Like the TGA ADI, JMPR established an ADI of 0 - 0.02 mg/kg body weight on the basis of the NOEL of 1.5 mg /kg body weight/day from the study of Serota *et al.* (1985, as cited in JMPR, 1992).

JMPR (1992) identified the following studies in which a NOEL was established (Table 2).

Table 2: Summary of pertinent studies of bifenthrin.

Species	NOEL mg/kg/day	Type of study	References
Mouse	86	28 day dietary	Rand <i>et al.</i> (1983a)
Mouse	7.6	lifetime dietary	Gieger <i>et al.</i> (1986)
Rat	11	28 day dietary	Rand <i>et al.</i> (1983b)
	5	90 day dietary	Rand <i>et al.</i> (1984)
	4	2 year feed	McCarty <i>et al.</i> (1986)
	1	teratogenicity	Freeman <i>et al.</i> (1984)
Rabbit	3	Reproductive	McCarty <i>et al.</i> (1986)
	100	21 day dermal	De Prosopo <i>et al.</i> (1984)
	2.7	teratogenicity	Freeman <i>et al.</i> (1984a)
Dog	2.5	13 week dietary	Serota <i>et al.</i> (1985)
	1.5	52 week dietary	Serota <i>et al.</i> (1985)

Source: JMPR (1992)

## 7 EXPOSURE ASSESSMENT

According to the 19<sup>th</sup> Australian Total Dietary Survey (ANZFA, 2001) synthetic pyrethroids are biodegradable and therefore tend not to persist in the environment. Bifenthrin was not a pesticide residue reported in this dietary study. However, as part of the Belgian Total Diet Study between 1991-1993, the average daily intake of bifenthrin from food commodities was estimated at 0.02 mg/kg person per day (Dejonckheere *et al.*, 1996, as cited TOXNET, 2002).

## 7.1 SETTING GUIDELINE VALUE

The most important exposure route for the setting of a HIL in soil for bifenthrin will be based on the target group of a two year old child.

## 7.2 DERIVATION OF HIL

The derivation of HIL is based on the following:

Soil ingestion	100 mg/ day
ADI	0.01 mg/kg/day
Bioavailability from the soil	100%
Weight of child	13.2 kg
Background exposures	

In the absence of Australian data, a default assumption is that equivalent portions (20%) of the ADI can be assigned to water, food, air, consumer products and soil.

A Health-based Investigation Level (HIL) can then be derived:

$$\begin{aligned} \text{HIL(mg/kg)} &= \frac{\text{ADI} \times 0.2 \times 13.2 \times 10^6 \times 1}{100} \\ &= 264 \\ &= 300 \text{ mg/kg} \end{aligned}$$

Accordingly a HIL of 300 mg/kg is recommended for bifenthrin.

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