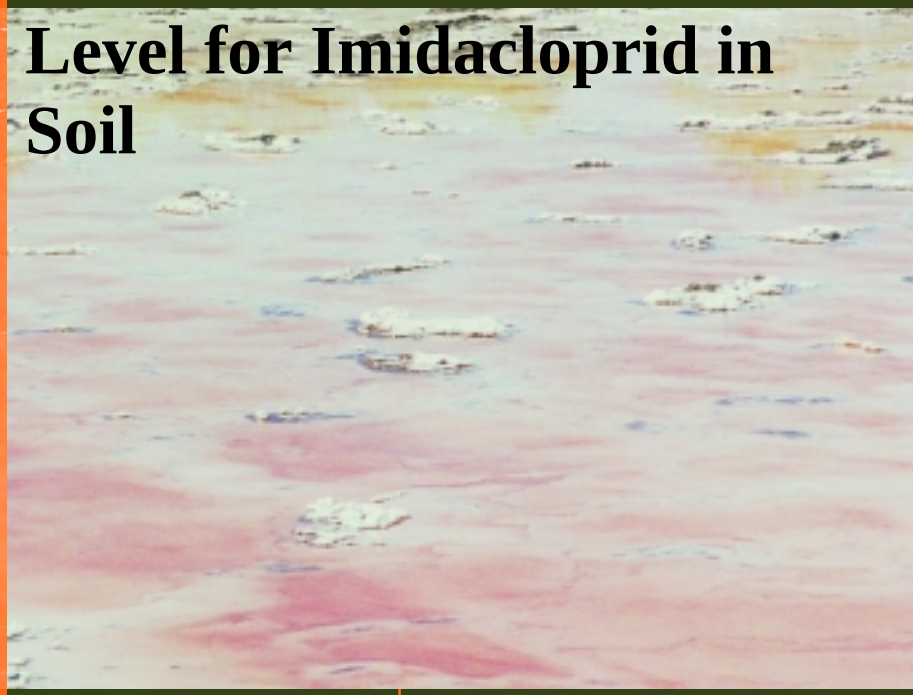
Health-Based Investigation Level for Imidacloprid in Soil



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Health Investigation Level for Imidacloprid in Soil

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1 INTRODUCTION

Imidacloprid is a broad-spectrum insecticide registered in Australia for the control of a wide range of insects. The use of imidacloprid as a termiticide, where it is applied directly to the soil, supports the establishment of a Health Investigation Level (HIL).

This paper describes the chemical and physical properties of imidacloprid, its uses in Australia and its occurrence and distribution in the environment. It reviews the toxicology of imidacloprid relevant to establishing the acceptable daily intake (ADI) and considers the potential for human exposure to occur. A HIL for imidacloprid in soil is derived.

2 PROPERTIES AND USES

2.1 PHYSICAL AND CHEMICAL PROPERTIES

Physical and chemical properties are described below:

Empirical formula	$C_9H_{10}ClN_5O_2$
Molecular weight	255.7 g/mol
Solubility in water (20°C at pH 7)	514 ppm
Partition coefficient Log P_{ow} (21°C)	0.57
Vapour pressure (20°C)	4×10^{-12} hPa

2.2 USES

Imidacloprid has developed into one of the main pest control agents for many crops. Imidacloprid is a broad spectrum insecticide that can be used in the control of a wide range of insects including aphids, various species of beetles and flies, leaf miners, termites and locusts (Bayer, 2000).

3 EXPOSURE STANDARDS

3.1 ENVIRONMENTAL STANDARDS

No environmental standards were identified.

3.2 OTHER APPLICABLE GUIDELINES AND REGULATIONS

ADI	National Registration Authority	0.06 mg/kg bw/day
ADI	World Health Organization	0.06 mg/kg bw/day

Australian Maximum Residue Levels (MRLs) in food range from 0.02 to 5.0 mg/kg (ANZFA, 2001).

4 ENVIRONMENTAL BEHAVIOUR, OCCURRENCE AND DISTRIBUTION

4.1 SOIL

Imidacloprid shows a medium adsorption to surface soil horizons (top 10 cm of the soil profile). However, leaching studies indicate that imidacloprid is not strongly bound to soils in lower horizons. The time for half the applied dose of imidacloprid to dissipate in soil has been determined to be 21 to 33 days in turf-covered soils (eg. golf courses and orchard settings), but in agricultural settings was found to be 1 to 2 years. The time for 90% of the applied dose to dissipate in turf-covered soils was found to be 1.2 to 2 years (Health Canada, 2001). These results suggest that imidacloprid has potential to persist in soil. In soils that are very porous or gravelly imidacloprid may move into ground water, otherwise, the potential for imidacloprid to contaminate ground water is considered low.

4.2 WATER

The half-life for imidacloprid has been reported to be less than 14 days (Bayer, 2000). Imidacloprid shows a high absorption rate of ultra-violet light in the wavelength range of natural sunlight. Therefore, a high rate of photolysis in aqueous solutions is expected. In turbid waters photochemical breakdown may be slow.

4.3 AIR

No information is available on the environmental behaviour, occurrence or distribution of imidacloprid in air.

4.4 PLANTS AND OTHER DIETARY SOURCES

No information is available on the extent that imidacloprid is taken up by plants or is present in food supplies.

5 ADSORPTION, DISTRIBUTION, METABOLISM AND ELIMINATION

Imidacloprid is rapidly and almost completely absorbed (>92%) from the gastrointestinal tract of rats and is widely and uniformly distributed to organs and tissues. In rats, imidacloprid was extensively metabolised with 10-16% of the parent compound eliminated unchanged. Elimination was rapid with 96% of the administered dose recovered in the urine and faeces within 48 hours (JMPR, 2001). As results from toxicological studies indicate that inhalation and dermal exposure to imidacloprid does not result in appreciable toxicity (JMPR, 2001) absorption through these routes of exposure is expected to be low.

Biological monitoring for imidacloprid in body tissues and fluids will only establish whether exposure to imidacloprid has recently occurred as it is rapidly eliminated from the body. Monitoring of the level of imidacloprid in urine may be useful in occupational settings where regular exposure to imidacloprid is more likely to occur.

6 TOXICOLOGY

Imidacloprid acts by interfering with the transmission of impulses in the nervous system. Like acetylcholine, the common nerve transmitter, imidacloprid stimulates certain cells by binding to the nicotinic receptor (Bayer, 2000 and NRA, 1994). Signs of neurotoxicity include reduced muscle tone, tremors, and in extreme cases muscle cramps and difficulty in breathing due to effects on the muscles associated with respiration.

Recent reviews of the toxicology of imidacloprid by the World Health Organization (JMPR, 2001) and the National Registration Authority (NRA, 1994) were reviewed for the purpose of establishing the HIL for imidacloprid in soil.

Limited information is available on the toxicity of the plant and animal metabolites of imidacloprid.

For the purpose of this discussion, toxicological considerations will be limited to the health effects reported in humans and toxicological studies considered by regulatory agencies in setting the ADI for imidacloprid.

6.1 HUMANS

No epidemiological studies of the effects of imidacloprid or information on symptoms of poisoning or clinical signs were available. A 4-year-old child who ingested about 10 mg/kg bw of a veterinary preparation of imidacloprid showed no signs of poisoning or adverse health effects (JMPR, 2001).

6.2 ACCEPTABLE DAILY INTAKE

Both the WHO and NRA set an ADI of 0.06 mg/kg bw/day for imidacloprid. This is based on a no observed effect level (NOEL) identified from a combined chronic and carcinogenicity dietary study in rats and the application of an uncertainty factor of 100 to account for inter- and intra-species variability. In this study, the NOEL was 5.7 mg/kg/day (100 ppm) with effects to the thyroid (increased mineralisation in the colloid of thyroid follicles) occurring at the next highest dose level (300 ppm). The effects to the thyroid were thought to represent a premature ageing process. Thyroid function as assessed by blood hormone levels, was not affected. At levels of 300 ppm, brain and plasma and erythrocyte cholinesterase activities were not affected by treatment (NRA, 1994 and JMPR, 2001).

7 BACKGROUND EXPOSURE

In assessing the potential for exposure to imidacloprid the main exposure pathway considered was ingestion through the intake of residues in food and water.

7.1 FOOD

No information was available on the level of imidacloprid residues in the food supply. However, on the basis of information on MRLs set for imidacloprid in different foods (ANZFA, 2001) and the daily consumption of these foods by a 2-year old (ANZFA, 2000) it was estimated that a 2-year old would consume 0.144 mg of imidacloprid a day. This equates to approximately 18% of the ADI for a 13.2 kg child.

7.2 WATER

No information was available on the level of imidacloprid in drinking water supplies. However, for the purpose of this assessment it is assumed that a 2-year old child would ingest 0.0792 mg/day (estimate based on imidacloprid residue in drinking water contributing 10% to the ADI for a 13.2 kg child).

8 DERIVATION OF HEALTH INVESTIGATION GUIDELINE VALUE

As young children are considered the most likely to encounter significant residues of imidacloprid in soil in a residential setting, the HIL for imidacloprid accounts for the exposure that a 2-year old child could potentially experience in such a setting. The HIL derived for imidacloprid in soil is considered conservative, as it is unlikely that a person's daily intake of imidacloprid from soil would be maintained over a lifetime at the level estimated for a 2-year old.

For the purpose of establishing a HIL for imidacloprid in soil it is assumed that metabolites of imidacloprid in soil would be of similar toxicity as imidacloprid. Where possible the total level of imidacloprid residues in the soil should be compared to the HIL.

8.1 RECOMMENDED SOIL INVESTIGATION GUIDELINE FOR IMIDACLOPRID

The derivation of the HIL is based on the following:

ADI	0.06 mg/kg bw/day
Bioavailability from soil	100%
Amount of soil ingested	100 mg/day

The amount of soil ingested was based on a default soil ingestion value for a 2-year old (ANZECC & NHMRC, 1992).

Conservative estimates of background exposure to imidacloprid residues in food and water suggest that background exposure to imidacloprid would not be expected to contribute more than 30% of the ADI. However, as there is no data on background levels of imidacloprid in food and water it is considered reasonable that exposure to imidacloprid in soil should not contribute more than 20% of the ADI.

For a 2-year old weighing 13.2 kg, an intake of imidacloprid at 20% of the ADI (0.06 mg/kg bw/d) would equate to a daily intake of imidacloprid of 0.1584 mg. If a 2-year old were to ingest 100 mg of soil the concentration in soil required to achieve an intake of 0.1584 mg would be 1584 mg/kg. Accordingly, the HIL established for imidacloprid and its residues in soil is 1600 mg/kg.

This value applies when a structure is removed from a site leaving an exposed termiticide area or where a spill or disposal of the product has occurred.

9 FURTHER RESEARCH

Further research should be considered to more accurately estimate the background exposure to imidacloprid from food, water and air.

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