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REDUCED DOSE RATE INSECTICIDE USE IN CEREALS: EFFECTS ON INSECT PESTS AND PREDATORS.

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ABSTRACT

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REDUCED DOSE RATE INSECTICIDE USE IN CEREALS: EFFECTS ON INSECT PESTS AND PREDATORS

by David Edward Turner

Studies investigated the intrinsic toxicity of the insecticides deltamethrin, dimethoate and pirimicarb to *Rhopalosiphum padi* (Walk) (Homoptera: Aphididae) in the laboratory on a simple glass substrate. The dose-response data generated was used to predict reductions of cereal aphid populations in the field using reduced dose insecticide rates. The predicted reductions were initially tested under semi-field conditions and the results showed that above a certain dose threshold the predictions fitted well for deltamethrin. When predictions were tested in the field, predicted and observed population reductions were found to correlate well for deltamethrin and aphid control using reduced rate insecticide doses was not significantly different to a full field rate application.

Temperature was shown to influence the toxicity of topically applied λ-cyhalothrin and dimethoate to the cereal aphids *R. padi* and *Sitobion avenae* (Homoptera: Aphididae). The toxicity of λ-cyhalothrin was negatively and dimethoate positively correlated with temperature. *S. avenae* was more susceptible than *R. padi* to topical applications of both insecticides. Residual exposure to λ-cyhalothrin and dimethoate affected their toxicity to the aphids *R. padi* and *S. avenae* at different temperatures. A negative temperature coefficient was shown for λ-cyhalothrin whilst that of dimethoate was more complicated. Altering the route of exposure affected the susceptibility of the aphids to the insecticides. Laboratory exposure of aphids to deposits of λ-cyhalothrin applied in the field showed the same trends as λ-cyhalothrin applied in the laboratory.

The application of sub-lethal doses of λ-cyhalothrin to *R. padi* individuals affected measurements of their growth and development. Development time, adult weight, number of nymphs produced, mrgr and r_m values of the aphids were significantly different from untreated aphids when reared at an ambient temperature of 12°C. There were no differences between measurements of aphid growth and development at ambient temperatures of 18 and 25°C.

Substrate type affected the residual toxicity of laboratory applied doses of deltamethrin and dimethoate to carabid beetles (Coleoptera: Carabidae). Application of insecticide dose-rates similar to those used for aphid control were toxic on glass but relatively harmless on soil. The exposure to an insecticide by topical or residual application affected aphid consumption of carabid beetles in the laboratory. Presenting carabid beetles with insecticide contaminated aphids reduced the beetles consumption of the aphids compared to controls. When the contaminated prey were removed aphid consumption returned to that of the controls. The potential and limitations of reduced dose insecticide rates for IPM in cereals are discussed.

CONTENTS

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There is no such thing as a poisonous *substance* but only of a poisonous *dose*. It is possible to kill oneself with salt if one has the mind to do so.

Hassell, 1982

CHAPTER 1

INTRODUCTION AND LITERATURE REVIEW

One of the most important steps for man's success on Earth has been to secure adequate supplies of food. This was achieved through a transition from early hunter/gatherers to a settled farming situation (Spedding, 1992, Jones & Jones, 1984). The transition took place over thousands of years but despite the improvement of cultivation methods and crop yields that came with it, there was also an increased destruction of crops by both pests and diseases (Horsfall, 1956).

The growing of crops in a settled farming situation has led to the vast monocultures of todays modern farming techniques. This has favoured the multiplication and feeding of pests by providing more continuous and readily available food supplies. Even in the early stages of agricultural development there were examples of widespread devastation of crops by pests. For example, there are biblical accounts of famines caused by plagues of locusts consuming all plant material in their path...

By moving the wind had brought the locusts. They devoured all.....everything growing in the fields and the fruit on the trees. Nothing green remained on tree or plant in all the land of Egypt.

Exodus 10

Recently widespread destruction of crops has been witnessed in the potato crop in both Ireland (McBrien, 1964) and the United States (van Emden, 1989).

Estimates (Cranmer, 1967) have put crop losses up to harvest at over one-third of potential production. These losses were attributed to insect pests (13.8%), plant diseases (11.6%) and weeds (9.5%). If post-harvest losses are included in this assessment then the loss of potential production reaches 50%. The desire for a reduction in the level of these losses has meant that crop protection, using biological, chemical or cultural control, has become a vitally important area of agricultural production. The development, use and problems associated with insecticides in particular are reviewed in the following sections.

1.1. Insecticide Development

1.1.1. Early Inorganic Insecticides

The ancient civilisations of Athens and Rome were reported to have used soaps (made from olive oil and soda), smoke and to have burnt sulphur to ward off insect pests. In the centuries that followed some attempts were made to use plant extracts to discourage the presence of insects on crops, but these met with little success (Horsfall, 1956; Ordish, 1976; van Emden, 1989).

However two events, the establishment of *Leptinotarsa decemlineata* (Say) (Coleoptera: Chrysomelidae), the Colorado beetle and *Lymantria dispar* (L.) (Lepidoptera: Lymantridae), the Gipsy moth, in the USA, stimulated increased studies into methods of insect pest control (Hassell, 1982, Jones & Jones, 1984). This in turn led to the development of inorganic and eventually the vast range of organic insecticides that are available today.

Paris Green (impure copper arsenate) was probably the first successful insecticide and effectively controlled *L. decemlineata* and *L. dispar.* This was followed by other arsenical compounds including lead and calcium arsenate (Martin & Woodcock, 1983). One problem with Paris Green is its high mammalian toxicity, but the mass human mortality predicted from its application did not occur (van Emden, 1989). Arsenic compounds are also stomach poisons and therefore only effective against chewing insects and not those that feed from the phloem. Fumigation of fruit trees with hydrogen cyanide to control phloem feeding insects was developed in the latter half of the 19th century. However, by the end of the century the required dose had to be successively increased as control became less effective (Ordish, 1976). This observation was probably the first recorded case of the development of resistance in insects.

1.1.2. Early Organic Insecticides

Among the earliest organic insecticides were toxic extracts of plants used by primitive tribes to tip their hunting arrows or to bring fish to the surface of rivers and lakes (van Emden, 1989). The best known of these substances are pyrethrum (from *Tanacetum cinerariaefolium* (Composita: Astereae), rotenone (a root extract of *Derriis spp.* (Crucifera: Leguminosae) and nicotine (from tobacco). Although nicotine has contact and fumigant action against insects it also has a relatively high mammalian toxicity. In relation to aphids nicotine has been widely used to control *Aphis fabae* (Homoptera: Aphididae) on sugar beet. However in the case of pyrethrum and other

botanical extracts there was a high degree of specificity to insects. Differing activities among insect species were also revealed and this for the first time raised the prospect of selectivity for certain insecticides.

1.1.3. Organochlorine Insecticides

Organochlorine insecticides fall into two categories; chlorinated hydrocarbons e.g. DDT, HCH and cyclopentadiene derivatives e.g. aldrin, dieldrin (Jones & Jones, 1984) (see Table 1.1.). Undoubtedly the organochlorine insecticides have played a major role in the history of agrochemicals for pest control. The most famous (or infamous) organochlorine is DDT. It was first synthesised in 1874, but its insecticidal properties were only exploited during World War II and gained its discoverer, Dr Paul Muller a Nobel prize. The effective control of mosquitoes, lice and flies had a major impact on the epidemic diseases of malaria and typhus, resulting in their virtual eradication in many areas. When concern over the environmental effects of DDT (Carson, 1963) resulted in its restricted use in some areas, it was notable that the incidence of disease rapidly increased (Hutson & Roberts, 1985). The properties of DDT that made it so useful, cheap to manufacture, effective and safe to man and other warm-blooded animals, ultimately also lead to its downfall. The cost often meant application without regard to the environment (see Carson, 1963).

Table 1.1. Structure of the organochlorine insecticide DDT and related compounds with some of their important chemical properties (adapted from Graham-Bryce, 1987; Anon, 1995).

Common name	Synthetic chemical name	Type of action	Solubility in water (mg/l) at 20°C	K_{OW}*	vapour pressure (20°C) (mPa)
p,p'-DDT	1,1,1-trichloro-2,2-di-(4-chlorophenyl) ethane	Contact, stomach poison insecticide	insoluble	n.g.	0.025
Methoxychlor	1,1,1-trichloro-2,2-di-(4-methoxyphenyl) ethane	Contact, stomach poison insecticide	0.1	n.g.	n.g.
Dicofol	2,2,2-trichloro-1,1-di-(4-chloroxyphenyl) ethanol	Non-systemic acaricide	0.8	19000	0.053

* n.g. not given, value could not be found in literature

Its chemical stability resulted in unwanted persistence in the environment. This coupled with low water solubility and strong lipophilic character resulted in bioaccumulation along the food chain (Walker *et al.*, 1967; Matsumura, 1975; Walker, 1975). Therefore, when the less persistent organophosphorous and carbamate compounds were developed, organochlorine insecticide use became severely restricted. However, in relation to aphid control DDT is rather a poor aphicide due to its lack of mobility within and around the plant. A review of organochlorine development and use can be found by Brooks (1974).

1.1.4. Organophosphorous Insecticides

Organophosphorous compounds were spin-offs from the development of highly toxic nerve gases for potential use in warfare. Consequently many were highly toxic to man but were more easily broken down and less persistent in the environment than the organochlorines. Within the group there is a wide spectrum of physicochemical and biological properties. This can be seen by comparing some of the chemical properties of three insecticides in this group (Table 1.2.). Unlike the earlier organochlorine compounds the primary site of action of organophosphorous compounds is the enzyme acetylcholinesterase, which is responsible for hydrolysing acetylcholine, the substrate principally responsible for transmitting nerve impulses across synapses (Corbett *et al.*, 1984).

Table 1.2. Examples of organophosphorous insecticides and some of their important chemical properties (after Anon, 1995).

Common name	Synthetic chemical name (IUPAC)	Type of action	Solubility in water (mg/l) at 20°C	K_{ow}*	vapour pressure (° C) (mPa)
Malathion	S-1,2-bis(ethoxycarbonyl) ethyl 0,0-dimethylphosphoro dithioate	non-systemic contact insecticide	145	560	5.3 (30)
Dimethoate	0,0,dimethyl-S-methylcarbamoylm ethyl phosphorodithioate	systemic, contact insecticide	25	5	0.29 (20)
Dichlorvos	2,2-dichlorovinyl dimethyl phosphate	fumigant, contact insecticide	10	n.g.	290 (20)

* n.g. not given, value could not be found in literature

Many organophosphorous insecticides are effective aphicides because of their high mobility as compared to organochlorine compounds e.g. dimethoate, demeton-S-methyl. They are also considerably cheaper to apply than synthetic pyrethroids and carbamates recommended for aphid control (Oakley *et al.*, 1988). However they also have high insecticidal activity against non-target and beneficial insects e.g. pollinators, predators and parasitoids (Vickerman *et al.*, 1987a&b, Croft, 1990).

1.1.5. Carbamate Insecticides

The carbamate insecticides were discovered and developed by Gysin and co-workers in the latter 1940s (Gysin, 1941). The first successful carbamate was carbaryl developed in the USA by Union Carbide. Like the organophosphorous insecticides, carbamate insecticides have a wide range of chemical properties and modes of action (Table 1.3.). Carbamate insecticides are particularly useful for dealing with pests that have become resistant to organophosphorous insecticides. One of the most important carbamates for aphid control is pirimicarb. This was discovered in 1965 and is both fast acting and specific; selectively killing aphids. It has a moderately low mammalian toxicity and is safe to honeybees and most other beneficial insects (Zuriga & Suzuki, 1976). Pirimicarb's selectivity and short residual life are ideal for integrated control programmes at any crop growth stage. Its chemical structure gives it contact, fumigant and translaminar activity, it is also systemic by root uptake (Anon, 1994). Its main mode of action, through fumigant effects, makes it useful in cereals where the strong vapour phase activity is retained by a dense crop, giving effective aphid control at low rates

Table 1.3. Examples of carbamate insecticides and some of their important chemical properties (after Anon, 1995).

Common name	Synthetic chemical name (IUPAC)	Type of action	Solubility in water (mg/l) at 20°C	K_{ow}*	vapour pressure (° C) (mPa)
Methiocarb	4-methylthio-3,5-xyly methylcarbamate	non-systemic acaricide & insecticide	27	0.015	n.g.
Pirimicarb	2-dimethylamino-5,6-dimethylpyrimidin-4-yl dimethylcarbamate	fumigant, translaminar insecticide	2.7	5	4 (30)
Aldicarb	2-methyl-2-(methylyhio)propioaldehyde 0-methylcarbamoyl oxime	systemic insecticide	6	n.g.	13 (25)

* n.g. not given, value could not be found in literature

compared to the organophosphates. Although pirimicarb has only a limited residual life on plants, its persistence of effects are probably due to its selectivity towards to aphid predators and parasites. Preservation of these results in increased biological control of the aphids as pirimicarb breaks down, therefore giving the appearance of a longer residual life for the insecticide (ICI, 1989).

1.1.6. Synthetic Pyrethroids

The discovery and development of the synthetic pyrethroids has been one of the more recent advances in chemical pest control. As mentioned earlier, pyrethrum extract from *T. cinerariaefolium* has remarkably high knockdown properties. However limited stability in UV light has prevented the wide range use in agricultural of natural pyrethroids (Bullivant & Pattenden, 1976; Ruzo & Casida, 1981).

Table 1.4. Examples of pyrethroid insecticides and some of their important chemical properties (after Anon, 1995).

Common name	Synthetic chemical name (IUPAC)	Type of action	Solubility in water (mg/l) at 20°C	K_{ow} log P	vapour pressure (° C) (mPa)
Cypermethrin	(RS)-α-cyano-3-phenoxybenzyl (1RS,3RS;1RS,3SR)-3-(2,2-dichlorovinyl) 2,2-dimethylcyclopropane carboxylate	contact, residual insecticide	0.004	6.6	2.3×10^{-7} (20)
Deltamethrin	(S)-α-cyano-3-phenoxybenzyl (1R,3R)-3-(2,2-dibromovinyl)-2,2-dimethylcyclopropane carboxylate	contact, residual insecticide	< 0.0002	4.6	1.33×10^{-5} (25)
Cyhalothrin	(RS)-α-cyano-3-phenoxybenzyl (2)-(1Rs,3RS)-2-chloro-3,3,3-trifluoropropenyl)-2,2-dimethylcyclopropane carboxylate	contact, residual insecticide	0.004 ppb	6.8	c.0.001 (20)

Permethrin was the first photostable synthetic pyrethroid to be developed (Elliott *et al.*, 1973 a&b). By the late 1970's many more photostable compounds had been developed (Table 1.4) e.g. cypermethrin (Breese & Highwood, 1977), deltamethrin (Herve *et al.*,

1977), fenvalerate (Mowlam *et al.*, 1977). Further information may be found in many reviews of the synthetic pyrethroids i.e. Elliot & Janes, 1973; Crombie, 1980; Leahey, 1985; Ruigt, 1985.

The synthetic pyrethroids show a broad-spectrum of activity against a range of lepidopteran, coleopteran, homopteran, heteropteran, dipteran, orthopteran and thysanopteran pests (Cox, 1990; Hirano, 1989). Due to their high insecticidal activity, commercial formulations of some of the most active compounds such as deltamethrin, cypermethrin and λ-cyhalothrin are effective at very low field dosage rates, i.e. 5 to 25 g A.I./ha (Anon, 1993). This is often an order of magnitude lower than the organophosphorous and carbamate insecticides.

The precise mode of action of the synthetic pyrethroids is still not clear, although symptoms exhibited by affected organisms are indicative of a neuro-toxic effect. However it is generally accepted that the sodium channel of the nerve axon is the primary target for the neurotoxic action of pyrethroids (Soderland & Bloomquist, 1989). A high lipophilic nature and low volatility mean that the synthetic pyrethroids act as contact insecticides and have no systemic activity (Graham-Bryce, 1987). Therefore the main routes of intoxication are by direct contact during spraying, contact with residual deposits or consumption of contaminated substrates (Hirano, 1989).

1.2. Insecticide Use in Pest Control

Insecticides are one of the most powerful tools available for controlling pest outbreaks. They are highly effective, rapid in curative action, adaptable to most situations, flexible in meeting changes in agronomical and ecological conditions and economical. Of all the options available for pest management they are reliable for emergency action when a pest population approaches or exceeds the economic threshold (Metcalf, 1975).

However in the past the use of insecticides has often been ecologically unsound - the misuse, overuse and unnecessary use of insecticides have been important factors in the growth of interest in their more rational use (Hickman *et al.*, 1992). The application of an insecticide represents a purposeful contamination of the environment and should only be considered when the benefit:risk ratios are clearly tilted in favour of their use.

1.2.1. Problems of insecticide use

World agricultural productivity has increased with the development of high yielding crop varieties, disease and pest resistant plant varieties, and the production of fertilisers and highly effective synthetic pesticides (Yunlong & Smit, 1994). It has been estimated that without the use of pesticides, global crop yields could be reduced by as much as 30% (Finney, 1990). However, in todays society, where the general public are more aware of potential problems that pesticides may have on the environment, there is a need to manage their use to reduce adverse effects on the environment.

Public concern of the effects of pesticides on the environment were first raised by Rachel Carson in her book Silent Spring (1963), although previously Ripper (1944 & 1956) had expressed opinions on the undesirable side-effects of pesticides. The problems that have been observed were probably a result of misuse of toxic chemicals rather than their intrinsic toxicity. Other problems can be related to the method of application, e.g. less than 1% of a chemical that is applied may contribute to the mortality of the target pest (Graham-Bryce, 1987). Therefore in order to reduce undesirable side-effects it has become increasingly important to use compounds which demonstrate greater selectivity between target and non-target organisms, to improve pesticide application techniques (eg Hall, 1987 & 1991) and to further the understanding of the mechanisms that mediate undesirable side-effects (Jepson *et al.*, 1990; Jepson & Thacker, 1990).

1.2.2. Integrated insecticide use

Stern *et al.* (1959) defined the integrated control of insect pests as "*applied pest control which combines and integrates biological and chemical control. Chemical control is used as necessary and in a manner which is least disruptive to biological control.*" It could be argued that control failures during the mid 1950's when using pesticides resulted in a reluctance to use chemical controls within an integrated control strategy (van Emden, 1989). However with improvements in forecasting and predicting pest outbreaks, the dogma of integrated control today appears to be one of chemical control enhancing biological control (Poehling, 1987; Hickman *et al.*, 1992). The takeover of Bunting Biological Control by Ciba-Geigy is just one example of this. The new company Ciba-Bunting offers a complete integrated control programme, as opposed to the independent companies only selling chemical or biological control.

It must be pointed out that by definition of integrated pest control, optimal timing of the insecticide alone, using economic levels or economic thresholds will not

necessarily result in effective pest control with limited damage to biological control (Poehling, 1989). Whilst modern insecticides are less likely to accumulate in the environment, emphasis is now laid on the side-effects of the insecticides on non-target species especially beneficial insects. The desire today is for insecticide selectivity, stemming from the need to exploit naturally occurring beneficial species inconjuction with insecticides to bring about pest suppression (Poehling, 1986, 1988, 1989).

1.2.3. Pesticide selectivity

Pesticide selectivity has been placed into two categories - physiological and ecological (Hull & Beers, 1985). A physiological selectivity compound can be simply defined as "*one which discriminates in terms of its toxicity between organism to which it is applied.*" It has been shown that some insecticides posses elements of physiological selectivity or differential toxicities to entomophagous arthropod species (Lindgren & Ridgeway, 1967; Lasca, 1973; Lecorne & Smilowitz, 1980; Hsieh & Allen, 1986). Lecorne *et al.* (1980) compared the toxicities of carbaryl, metamidophos and pirimicarb. The latter was found to be less toxic to *Chrysopa oculata* (Say) (Neuroptera: Chrysopidae) and *Coleomegilla maculata* lengi (Coleoptera: Coccinellidae) (Timberlake), predators of the peach aphid (*Myzus persicae* (Sulz) Homoptera: Aphididae). The selectivity ratio (SR) of pirimicarb in favour of the natural enemies was *ca.* 4000:1 (SR is defined as the LD_{50} of the non-target organism divided by the LD_{50} of the pest. A ratio of greater than 1 indicates selectivity favouring the natural enemy and a ratio less than 1 disfavouring them).

Ecological selectivity attempts to minimise contact between the pesticide applied and beneficial organisms when controlling a particular pest. Ecological selectivity can be achieved via different routes; application technique (e.g. applying a systemic insecticide for phloem feeding pests), reducing the dose (Teetes,1972; Hull & Beers, 1985), limiting the area over which the insecticide is applied and the application timing (Jepson, 1989; Taye & Jepson, 1989; Croft, 1990). Application timing attempts to reduce the probability of hitting the vulnerable stages of the natural enemy whilst attacking the stage at which the pest is most susceptible.

1.3. Cereal aphids (Homoptera: Aphididae) as Pests of UK cereals

It has long been recognised that certain crops are more prone to attack by aphids than others (Blackman & Eastop, 1984). For instance, Hill (1568) wrote of "green fles o ye gardn"; Worlidge (1669) "of insects and creeping things offending"; and Evelyn (1693)

of "green fleas which fatten on the young shoots of peach and little black flies on artichokes in summer."

1.3.1. Aphid cereal pests

There are three common species of cereal aphids that colonise cereals at some time during their life-cycle. These are, the grain aphid (*Sitobion avenae* (Say)), the rose-grain aphid (*Metapolophium dirhodum* (Walk)) and the bird-cherry aphid (*Rhopalosiphum padi* (L.)). Several other species have also been recorded and may occasionally be common. Although cereal aphids have been recorded since the 18th Century (Marsham, 1798), they were not given pest status until the early 1950's when they were found to be vectors of Barley Yellow Dwarf Virus (BYDV) (Kendall *et al.*, 1984).

M. dirhodum is a host alternating species overwintering mainly as eggs on rose (*Rosa* spp.), its primary host, before spring migrants colonise cereals. On wheat, summer migrants may be induced from the milky-ripe stage onwards (Growth Stage (GS) 73, Zadoks *et al.*, 1974) in the absence of crowding, but at an earlier growth stage if the aphids are crowded. These summer migrants then disperse to colonise other grasses (Howard, 1988). In the autumn the aphids switch to producing sexual autumn migrants in response to short day lengths and low temperatures, these mate and lay their eggs on the primary host (Dixon, 1987).

S. avenae lives all year round on grasses moving between species during the course of each season. It overwinters mainly as eggs in northern Britain and pathenogenetically in the south (Newton & Dixon, 1988). The eggs hatch in March and give rise to spring migrants which along with aphids that overwinter parthenogenetically initiate the summer increase in abundance on cereals (Dixon, 1987). This aphid prefers to feed on the ear of cereals and is therefore much more conspicuous than *Metapolophium dirhodum*. The grain aphid can achieve very high rates of increase feeding on the ears and regularly becomes very abundant, i.e. this species exceeded the threshold of five aphids per tiller five times between 1977 and 1988 in Norfolk (Oakley *et al.*, 1988). The development of summer migrants is induced by crowding and poor host quality, this first slows down and then terminates population build-ups. The production of summer migrants enables this species to exploit grasses that flower and seed throughout the summer. Autumn migrants and sexual forms are again produced in response to short day lengths and low temperatures.

When *R. padi*, the principle BYDV vector, invades during the spring and autumn, transmission of the virus causes severe stunting of the infected plants. *S. avenae* and *M.*

dirhodum invading during the summer, cause damage by direct feeding and honeydew production (Vereijken, 1979; Rabbinge & Carter, 1983) which can reduce grain weight and quality (Lee *et al.*, 1979). A UK outbreak of *S. avenae* was first documented by Anglade (1969) and Fletcher & Bardner (1969) in 1968.

Cereals especially those sown in the spring are available for exploitation only for a short period of time. For instance during the 1976 aphid outbreak, population build-up and collapse on winter wheat lasted only five weeks. Thus there is often little time to monitor and respond to aphid population increases on cereals before they reach damaging levels. Approximately 2,000,000 ha of wheat are grown in the UK each year giving a yield of approximately 15,000,000 tonnes. Sunderland *et al.* (1983) calculated that a minor outbreak (eg a 12.5% yield loss) of cereal aphids would result in a loss of £144,000,000 if left unsprayed and £22,000,000 if the crop was sprayed on time.

Cereal aphids, therefore, have a high pest status in UK cereals. In outbreak years control measures are required to prevent or reduce the economic damage caused by their feeding.

1.3.2. The control of cereal aphids with insecticides

In economic terms prophylactic spraying would appear to be the best approach; a tank-mixed application of dimethoate can be applied for around £2 per hectare - the approximate cost of an aphid count by a crop consultant (D. Thackery, pers. comm.). The problem with spraying insecticides prophylactically is that it is uneconomical to spray when aphids are not causing economic damage. If a non-selective aphicide is used it may kill indigenous natural enemies which may lead to a later aphid outbreak if favourable conditions occur (Powell *et al.*, 1985). The development of resistance may also occur if the same aphicide is continuously prophylactically applied. However from the farmers point of view, prophylactic spraying acts as an insurance policy and does not have the risks of monitoring and forecasting.

The results of field trials measuring yield losses due to cereal aphids in the UK has enabled the Agricultural Development and Advisory Service (ADAS) to produce economic thresholds for spraying with aphicides. ADAS recommends spraying if aphid populations are increasing at flowering and exceed 5 per ear (George, 1975; George & Gair, 1979) or 66% of ears infested (Anon, 1988). George and Gair (1979) estimated 10-20% yield losses due to direct damage by *S. avenae* in winter wheat, whilst Vereijken (1979) concluded that in the absence of a fungicide treatment, losses caused by fungi growing on the aphids honeydew may cause up to a 50% yield loss.

Table 1.5. Common invertebrate natural enemies of cereal aphids that inhabit the temperate cereal ecosystem.

POLYPHAGOUS PREDATORS	APHID-SPECIFIC PREDATORS
Coleoptera: Carabidae	**Coleoptera: Coccinellidae**
Agonum dorsale (Pontoppidan)	*Adalia bipunctata* (L.)
Bembideon lampros (Herbst)	*Coccinella septempunctata* (L.)
B. obtusum (Serville)	*Propylea quadridecempunctata* (L.)
Calathus fuscipes (Goeze)	
Demetrius atricapillus (L.)	**Diptera: Syrphidae**
Harpalus rufipes (Degreer)	*Episyrphus balteatus* (Degeer)
H. affinis (Shrank)	*Metasyrphus corollae* (L.)
Nebria brevicolis (F.)	*Syrphus vitripennis* (Meig.)
Notophilus biguttatus (F.)	
Pterostichus melanarius (Ill.)	**Hemiptera: Anthocoridae**
P. madidus (F.)	*Anthocoris nemorum* (L.)
Trechus quadristriatus (Shrank)	*A. nemoralis* (F.)
	Neuroptera: Chrysopidae
Coleoptera: Staphylinidae	*Chrysopea carnea* (Stephens)
Philonthus cognatus (Stephens)	
Tachyporus hypnorum (F.)	PARASITOIDS
T. obtusus (L.)	
T. dispar (Paykull)	**Hymenoptera: Braconidae**
T. chrysomelinus (L.)	*Aphidius picipes* (Nees)
	A. rhopalosiphi (De Stefani-Perez)
Aranae: Linyphiidae	*A. ervi* (Haliday)
Erigone atra (Blackwall)	*Praon volucre* (Haliday)
Oedothorax apicatus (Blackwall)	*Toxares deltiger* (Haliday)
Aranae: Lycosidae	
Pardosa pullata (Clerck)	
Dermaptera	
Forficula auricularia (L.)	
Diptera: Empididae	
Empis livida (Meig.)	
Platypalpus minutus (Meig.)	

(Adapted from Moreton, 1969 and Wiles, 1992).

1.4. The Natural Enemies of Aphids Present in the Cereal Ecosystem

The natural enemies of aphids are wide-ranging and varied (Wratten, 1987; Wratten & Powell, 1991; Cilgi, 1994). Wratten (1987, 1989) reported that approximately 390 species of beneficial arthropods inhabit the cereal fields of the south of England (see Table 1.5.). They can be placed into four categories; i) aphid specific predators, ii) polyphagous predators, iii) parasitoids and iv) pathogens (Vickerman & Sunderland, 1975; Edwards *et al.*, 1979; Vickerman & Wratten, 1979; Carter *et al.*, 1980; Powell, 1980; Sunderland & Vickerman, 1980; Chambers *et al.*, 1983; Helenius, 1990; Wratten & Powell, 1991). Aphid specific predators have been shown to play a role in reducing aphid numbers within a season (Carter *et al.*, 1980). However the "background" predation of the polyphagous predators would appear to play a role in preventing rather than limiting aphid outbreaks (Sunderland *et al.*, 1987). The four categories are discussed further below.

1.4.1. Aphid specific predators

Aphid specific predators include the Coccinellidae (both larvae & adults), Chrysopidae (larvae) and Syrphidae (larvae) (Vickerman & Wratten, 1979). Their value as aphid control agents has been widely reported e.g. Rabbinge *et al.*, 1979; Chambers *et al.*, 1983; Sunderland *et al.*, 1986. They often require a certain threshold density of aphids in order to survive and propagate (Hodek, 1970; MacClean *et al.*, 1977), but can locate aphids at low densities. For example, syrphids may lay eggs at aphid densities of only 0.4-0.5 aphids per shoot (Chambers & Aikman, 1988). Aphid-specific predators often reproduce when aphids are abundant and well into the establishment phase of their population development (Chambers *et al.*, 1983), this ensures an adequate food supply for their offspring. Therefore aphid-specific predators may be important in controlling late season pest out breaks or may help in hastening population crashes (Sunderland, 1975; Vickerman & Wratten, 1979; Griffiths, 1982; Chambers *et al.*, 1983; Wratten, 1985, 87 & 88). Summaries of field studies providing evidence for the importance of aphid-specific and polyphagous predators are given in Table 1.6.

1.4.2. Polyphagous predators

The majority of aphid predators found in the cereal ecosystem are polyphagous (Sunderland *et al.*, 1985). To this category belong the Carabidae, Staphylinidae, Linyphididae, Forficulidae, Empididae and Dolichopodidae (Sunderland, 1975; Edwards *et al.*, 1979; Sunderland & Vickerman, 1980; Chambers *et al.*, 1983; Winder, 1990; Dennis & Wratten, 1991). Although polyphagous predators consume other prey than

Table 1.6. Summary of field studies investigating the effect of aphid-specific and polyphagous predators on populations of cereal aphids.

Author	Predators studied	Type of plot used	Methods	Comments
Edwards *et al.*, 1979	Carabidae	10 & 5 m^2 plots	Plots surrounded by polythene barriers and predators removed by pitfall traps or application of fonofos. Open field used for comparison.	The numbers of beetles caught in the enclosed and fonofos treated plots was less than control plots. Aphid populations were smaller in the control than manipualted plots. Earlier removal of predators resulted in higher aphid populations. An inverse relationship was found between polyphagous predators and aphid numbers. A positive relationship was found between aphid specific predators and aphid numbers.
Chambers *et al.*, 1983	Cocinellidae, Syrphidae, Chrysopidae	Open plots and plots covered by netting cages (1.8 x 3 x 1.5 m)	Nets were erected early and late in the season to protect aphid populations from predators	Predator numbers were found to be negatively associated with aphid abundance. Predation was considered to be the cause of differences in nimbers between caged and uncaged aphid populations.

Table 1.6. continued

Author	Predators studied	Type of plot	Methods	Comments
Chambers *et al.*, 1986	Coccinellidae, Syrphidae, Chrysopidae, Hymenopterous parasitoids	Open field	Sweep net samples taken from middle of crop and 5m from field boundary.	Observations suggested that aphid population growth was supreesed by aphid specific predators and parasitoids. Aphid specific predators were mainly responsible for population decreases.
Chiverton, 1986	Carabidae, Staphylinidae, Aranae	12.5 m^2 plots enclosed by 60 cm high polythene barrier. Open plots used for controls.	Polyphagous predators were removed from plots by pitfall trapping.	Trapping out of predators resulted in higher aphid populations in barriered compared to control plots. Earlier trapping out of predators resulted in higher aphid population levels. An inverse relationship was found between the number of pests and predators.
Winder, 1990	Carabidae, Staphylinidae, Coleopteran larvae, Lynphiidae, Syrphidae	$4m^2$ open plots	Pitfall traps were used to either trap out predators continuously (decreased plots) or trapping was restricted to three 5-day periods (intermediate plots). Control plots were free from pitfall traps. Aphid fall-off and climbing rates were also measured	Peak aphid densities were highest when polyphagous predator densities were lowest. Aphid fall-off rates were similar between plots, but climbing rates were highest when predator density was lowest.

Table 1.6. continued

Author	Predators studied	Type of plot	Methods	Comments
Dennis & Wratten, 1991	Staphylinidae; *Tachyporus spp.* & *Philonthus cognatus*	0.25 m^2 tepee exclusion/enclosure cages	Using total exclusion cages known numbers of predators were introduced.	With high numbers of aphids nly *P. cognatus* significantly influenced aphid popualtion numbers. At low aphid population densities *T. obtusus*, *T. chrysomelinus* and *P. cognatus* reduced population levels.

cereal aphids, they are present in the field during the crucial aphid establishment phase and some have been shown to consume aphids when they are at low densities (Sunderland & Vickerman, 1980). Consequently, obtaining evidence of their importance in regulating aphid populations in cereal crops has been the subject of both large and small scale field studies (see Table 1.6.).

Potts and Vickerman (1974) dispelled the myth that cereal fields were a sterile environment. They showed there were significant negative relationships between cereal aphid numbers and the proportion of predatory arthropods in different fields. These findings were verified by Chambers *et al.* (1982 & 1983) and Ekbom and Wiktelius (1985) from field experiments carried out in cereals (see Table 1.6.).

Manipulation studies using polythene barriers dug into the soil to exclude predators from experimental plots (Edward *et al.*, 1979; Chiverton, 1986; Winder, 1990) have shown that the absence of polyphagous predators results in aphid populations increasing at a faster rate than when predators are present. Therefore when aphid numbers are below thresholds for polyphagous predators to have a suppressive effect, the predators may be able to prevent aphid populations from reaching economic damage levels and so make spraying unnecessary. Their impact on aphid populations will be determined by a number of factors. These include prey density, predator density, dispersive capacity, climbing ability and prey consumption rate and will differ between species (Sunderland & Vickerman, 1980; Coombes & Sotherton, 1986; Pearson, 1980; Griffiths, 1983; Loughridge & Luff, 1983; Sopp & Wratten, 1986). Differences in the ability of polyphagous predators to control aphid population numbers were shown by Dennis & Wratten (1991) (see Table 1.6.).

1.4.3. Pathogens

Entomophagous fungi are effective at controlling aphids in the latter part of the season (Burn, 1987). Dedryver *et al.* (1980) reported three dominant pathogens in Europe; *Erynia neoaphidis*, *Conidiobolus obscurus* and *Entomophthora planchoniana*. The weather conditions and the time of year determine which is the predominant species. These fungi can contribute to the decline of aphid populations but they often occur too late to cause any substantial impact (Chambers *et al.*, 1983).

1.4.4. Parasitoids

Aphid populations in cereal crops may be reduced by a large array of parasitoids which belong to two hymenopteran families, the Aphelinidae and Aphidiidae (Powell,

1982). The activity of parasitoids in early winter-sown cereals may have significant effects on aphid populations, since parasitoids are abundant and mobile between fields (Powell, 1983; Vorley, 1986; Fougeroux *et al.*, 1988). The early attack of aphid populations by certain parasitoids (e.g. *A. rhopalosiphi* (De Stefani-Perez)) may be a strategy to reduce the loss of offspring due to aphid predation and hyperparasitism later in the season (Vorley, 1986). As a consequence of this they may not be very effective when late season aphid outbreaks occur. Initial growth rates of aphid populations may be slowed down by a high parasitoid:aphid ratio early in the season (Powell & Wratten, 1991). This could increase the likelihood of other natural enemies being able to keep aphids below economic damage levels later in the season, due to reductions in potential peak population levels (Powell & Wratten, 1991). Parasitoid numbers in late winter- and early-spring sown cereals are dependent on immigration from early-sown cereals and grassland (Vorley & Wratten, 1987). Using simulation modelling (Vorley & Wratten, 1985), it was demonstrated in the absence of parasitoid migrations from early- to late-sown winter cereals grain aphid populations would increase at flowering at a rate seven times greater than in the presence of such migrations.

1.4.5. Side-effects of pesticide applications on aphid natural enemies

The assessment of the side-effects of pesticide applications on predators and parasitoids ('non-target invertebrates') in cereals has recently become a major area of research interest (Brown, 1988; Jepson *et al.*, 1990; Barrett, 1992; Aldridge & Carter, 1992; Carter *et al.*, 1992; Dohmen, 1994). The actual benefit of these organisms is not always easy to determine (Hagen & van den Bosch, 1968; Oakley *et al.*, 1988; Sanderson *et al.*, 1992) but the importance of their ability to limit aphid pest populations demonstrates the need to understand what effect an insecticide application has on all aspects of their biology. Despite the widespread application of insecticides for controlling aphids during the past 25 years, there has been no increase in the incidence of aphid pest outbreaks that could be attributed to losses of natural enemies from cereal fields (Burn, 1987). It could be argued that growing cereals in monocultures is the cause of aphid outbreaks rather than a loss of natural enemies. Although localised extinction of natural enemies by insecticide applications does affect subsequent aphid population development (Duffield, 1992). Aphid population numbers increased at a faster rate in the centre of a treated plot compared to untreated areas of a wheat field. This was attributed to aphid population numbers increasing at a faster rate than natural enemies could re-invade into the treated plot from untreated areas in order to exert some population control on them.

There are excellent reviews of the side-effects of pesticide applications on non-target invertebrates e.g. Hill, 1985; Smith & Straatton, 1986; Inglesfield, 1989; Croft, 1990. The results generated from field studies of side-effects on non-target invertebrates indicate that predatory groups, such as the Carabidae, Staphylinidae, Syrphidae, Chrysopidae, Coccinellidae, Anthrocoridae, Arenae can be adversely affected by pesticide applications (Basedow *et al.*, 1985; Fischer & Cambon, 1987; Vickerman *et al.*, 1987a&b). In general the toxic affects of the insecticides applied could be ranked as follows; organophosphates > pyrethroids > carbamates. Carbamates were considered the least toxic because the specific aphicide, pirimicarb, is often applied.

The results generated from field studies such as those mentioned above may be difficult to interpret because they are primarily based on pitfall trap catches. Therefore detecting changes in predator numbers before and after treatment will be limited by the sampling method and experimental design. For example, pitfall trap capture efficiency is known to be species dependent (Curtis, 1980; Halsall & Wratten, 1988; Topping & Sunderland, 1992) and can also be affected by biotic and abiotic variables i.e. soil type (Adis, 1979) and vegetation cover (Speight & Lawton, 1976). Even if these limitations are minimised direct toxicological effects cannot always be inferred, since predator number reductions could also be due starvation rather than direct mortality from an applied insecticide (Vickerman *et al.*, 1987a&b).

Side-effects of pesticide applications on predators and parasitoids have been observed at recommended field rate applications (Vickerman *et al.*, 1987a&b). The next section considers the use of reduced dose insecticides in order to reduce undesirable side effects on predator and parasitoid populations whilst still bringing about the suppression of pest population numbers.

1.5. Reduced Dose Insecticides in Pest Control

The fundamental idea behind the concept of using reduced doses in pest control is to improve the natural enemy:pest ratio in favour of the natural enemies (Stern *et al.*, 1959). A reduction in the dose reduces the efficacy of the insecticide and a residual pest population will be left behind. However the residual pest population will provide food for the preserved natural enemy fauna of the crop, so that they will remain in the field and prevent any further pest resurgence (van Emden, 1988). Reasons for the preservation of natural enemies are discussed below.

1.5.1. Physiological selectivity through reduced doses

As discussed earlier, physiological selectivity refers to an insecticide having greater toxicity to a pest compared to a natural enemy when applied to populations of both. It involves differences in processes such as penetration, sequestration, excretion, detoxification and target site insensitivity between the different species. Few of these mechanisms have been studied in any great detail but they are summarised by Croft (1990).

Evidence for different degrees of selectivity that may be present within the cereal ecosystem has been provided by Brown *et al.* (1983). Laboratory studies investigated the responses of a guild of aphid predators and their prey *Metapolophium dirhodum* (Walk) to a range of insecticides. The position and slope of the dose-response curves indicated that the relative susceptibilities of the predators and aphid varied between the insecticides. The toxicity of the compounds to the aphid and its predators was as follows parathion-methyl > cypermethrin > pirimicarb. In relation to selectivity of the compounds at doses causing > 90% mortality of the aphid, pirimicarb exhibited selectivity to all six of the predators studied, parathion-methyl to three of the five predators and cypermethrin to none.

1.5.2. Management tactics for pest control using selectivity differences.

Application of selectivity differences in the field needs to be selected from at points along a dose-response curve where pest mortality is moderate to high and there is little or no mortality of a natural enemy (Bartlett, 1964; Hower & Davies, 1984). Croft (1990) has suggested several perspectives on selectivity for hypothetical pests and natural enemies (Fig. 1.1.) that could be used when considering the use of reduced doses within an IPM programme.

The ideal option would be *maximum selectivity*, the point where differences in pest and natural enemy mortality is greatest (P - NE_1). However, when choosing an insecticide rate, control of the pest is usually given more precedence over selectivity towards the natural enemy. Therefore *management selectivity* may be chosen to achieve adequate pest control but at the expense of greater natural enemy mortality. Management selectivity will be determined by the economics of pest control and the benefits of control provided by the pesticide verses the control by the natural enemy (Croft,1990). Figure 1.1. also illustrates the narrow range of differences in the dose-response curve of a pest and natural enemy. This range difference may not allow adequate control of the pest whilst conserving the natural enemy to a very large degree.

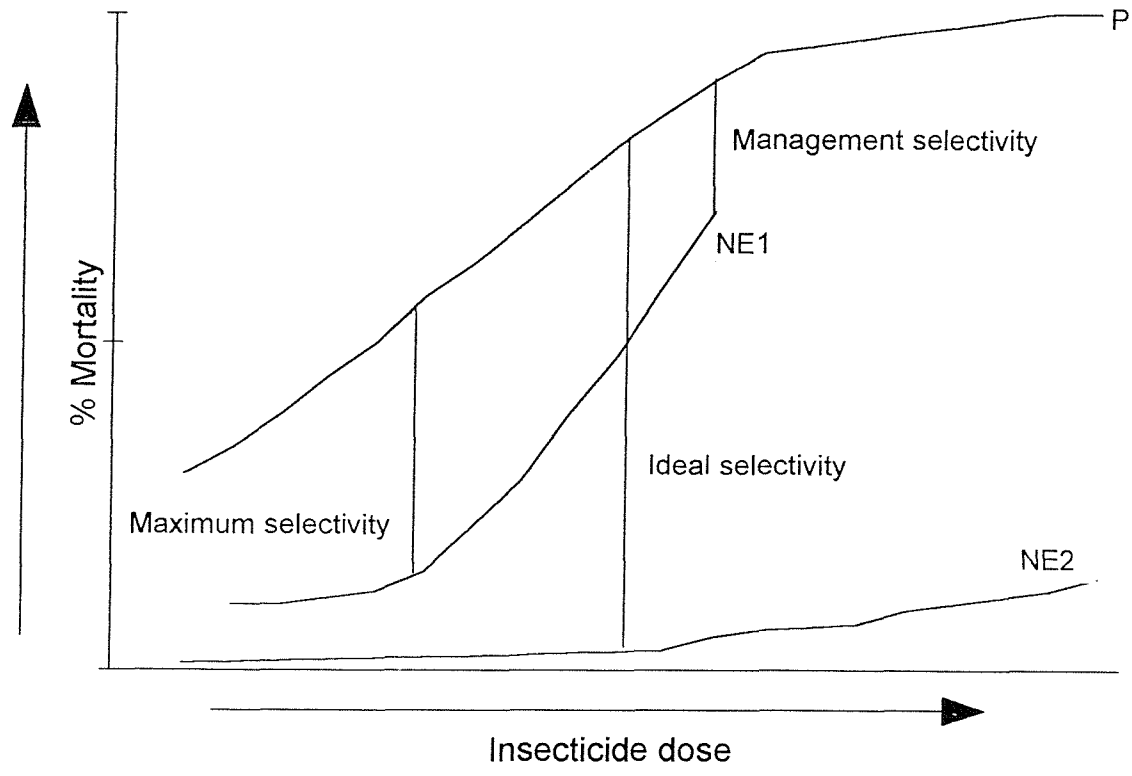


Figure 1.1. Relationship between maximum, management and ideal physiological selectivity of an insecticide to a natural enemy (NE) and its prey or host (P) (after Croft, 1990).

Ideal selectivity could be achieved if moderate to high mortality of the pest was possible, whilst the natural enemy was unaffected over a range of insecticide doses (P - NE_2). Pirimicarb would be an example of this and surviving natural enemies would contribute greater biological control (P - Ne_2) compared to a less selective insecticide (P - NE_1).

1.5.3. Specific cases of reduced dose insecticide use in pest control.

The use of reduced doses has been successfully applied to pest control in orchards and crops such as sorghum, rice and cereals (Smith *et al.*, 1985; Kiritani, 1976; Wertzel *et al.*, 1987; Poehling, 1989; Cross & Berrie, 1990). Stern *et al.* (1959) controlled the organophosphorous resistant spotted alfalfa aphid (*Therioaphis trifolii* (Homoptera: Aphididae) by applying insecticides at reduced rates. Part of the reason for the improved control was the increased natural enemy:pest ratio; achieved through the reduced mortality of aphid natural enemies. Hoyt (1969) demonstrated that azinophos-methyl applied at reduced rates, down to 50-60% of the standard rates, still provided control of the codling moth (*Cydia pomonella* (L.) (Lepidoptera: Tortricoidea)) on apple, whilst allowing the survival of moderately high numbers of the predatory mite *Typholodromus occidentalis* (Nesbitt) (Acarina: Phytoseiidae). In New Zealand, Collyer (1976), demonstrated similar selectivity to the above using reduced doses of azinophos-methyl again. The reduced doses allowed substantial survival of *T. pyrii* (Acarina: Phytoseiidae), rendering the treatment for *Panomychus ulmi* (Koch) (Acarina: Phytoseiidae) unnecessary. The results of Hull and Starner (1983) indicated that low rates of pyrethroids applied over a three year period could select in favour of the predator *Stethorous punctum* without compromising control of *Cydia pomonella* (F.) and *P. idaeusalis (Wlk.)* compared to the control provided by standard organophosphorous insecticides.

Poehling (1987, 1988 & 1989) , Taye (1988) and Taye & Jepson (1988) investigated the prospects for enhanced levels of natural enemies and adequate pest control in reduced rate pesticide regimes. They all found that reduced doses resulted in reduced kills of natural enemies. Poehling (1989) studied cereal aphids and a few selected natural enemies (*Coccinella septumpunctata*, *Episyrphus balteatus* and *Chrysoperla carnea*). The results demonstrated that reduced-doses could generate increasing differences in effects between pests and predators. For instance, at a dose which is still toxic to the pest, syrphid mortality could be dramatically reduced by lowering the dose of pirimicarb from 100 g a.i./ha to 25 g a.i/ha. The effect was even more evident with fenvalerate. On the other hand, no selective effects could be achieved using coccinelid larvae and fenvalerate. These observations illustrated that selectivity is often

species specific and how small a range of selectivity broad-spectrum insecticides can have.

Currier & Witkowski (1988) determined the efficacy of chlorpyrifos 4E at reduced rates when compared to the highest recommended rate for control of the European Corn Borer (ECB), *Ostrinia nubilalis* (Hbn.) (Lepidoptera: Pyralidae), larvae in whorl stage corn. Single and double applications of the insecticide were applied, i.e. the double application was half the rate of the single application, applied twice. No significant differences between plots receiving 1.12 kg a.i./ha and those receiving lower insecticide treatments were found. However, there were significantly fewer ECB cavities in plots receiving 0.56 kg a.i./ha than those receiving 0.28 kg a.i./ha.

1.6. Insecticide Resistance in Insects

Resistance to pesticides is a worldwide problem and has been documented in more than 440 species of insects and mites (Georghiou, 1986). In consequence the response to resistance is often an increase in the concentration or frequency of the pesticide applied (Tabashnik & Croft, 1982; Tabashnik, 1986 & 1989; Georghiou & Taylor, 1986). However, there is little doubt that pests have developed resistance rapidly when frequently treated with insecticides i.e. bioassays showed that resistance to pyrethroids in the pear psylla (*Psylla pyricola* (Coleoptera: Psylliodes) was correlated with the extent of pyrethroid use (Croft *et al.*, 1989). The same was also true for the cotton aphid, *Aphis gossypii* (Homoptera: Aphididae) Glover from different Hawaiian Islands (Hollingsworth *et al.*, 1994)

1.6.1. Insecticide resistance in aphids

Currently in the UK there are only three important species of aphids that have developed resistance to organophosphorous, carbamate or pyrethroid insecticides. These are the damson-hop aphid, *Phorodon humuli* (Schrank), the cotton aphid *Aphis gossypii* (Glover), and the peach-potato aphid *Myzus persicae* (Sulz.) (Homoptera: Aphididae) (Perrin, 1983). Resistance to an insecticide can be conferred by three general processes; i) reduced penetration, ii) increased detoxification or iii) altered site of action.

Decreased penetration has not been found in aphids although it is present in many other resistant insects (McCaffery & Holloway, 1992). Detoxifying mechanisms can be conferred by hydrolases, mixed function oxidases, glutathione-S-tranferases and DDT-dehydrochlorinase (Oppenoorth, 1985). In the case of *M. persicae* the evolution of

resistance to organophosphorous, carbamate and pyrethroid insecticides was through the overproduction of a carboxylesterase (E4) that both degrade and sequester these insecticidal esters (Devonshire, 1977; Devonshire & Moores, 1982 & 194). The increased amount of E4 is due to the amplification of the genes that code for this enzyme (Devonshire & Field, 1991). Recently however the third mechanism of resistance has also been found in this aphid. This is an altered form of acetylcholinesterase which confers resistance to the specific aphicide, pirimicarb (Moores, Devine & Devonshire, 1994). Currently there are no documented cases of resistance for the common cereal aphids and the less intensive use of insecticides in cereals compared to glasshouses or other field crops is likely to reduce the pressure on the development of resistance within these species.

1.6.2. Insecticide Resistance and the Use of Reduced Doses

Resistance development, could in theory, be avoided by ensuring that all the treated insects are killed. However, this is easier said than done (Crow, 1952). Modelling studies (Tabashnik & Croft, 1982) have shown in theory that the combination of immigration by homozygous susceptible (SS) individuals and use of a dose sufficiently high to kill the heterozygous (RS) individuals would suppress resistance development (Taylor & Georghiou, 1979; Tabashnik & Croft, 1982). The basic idea behind this is that the few homozygous (RR) resistant survivors in the treated population would mate with the SS immigrants and produce RS offspring that can be killed by the insecticide.

Is it possible to introduce reduced dose rates into the scenario described above? Reduced doses are often considered to encourage resistance to develop in insect pests at a faster rate compared with more standard doses. It is thought that reduced doses will kill only susceptible individuals and part of the heterozygous resistant population, allowing large populations of the homozygous resistants to persist and reproduce. Higher doses would not only kill all of the SS individuals but also more of the RS & RR individuals. However, this may not always be the best solution for resistance management, since a low pesticide use strategy may reduce the potential for pest resistance while maximising the potential for biological control (Tabashnik & Croft, 1982). In principle a low-pesticide use strategy will reduce the rate at which the S genes are removed from the population, thereby reducing the rate of resistance evolution. Biological control potential may be maximised because reduced doses may help to conserve natural enemies by maintaining their food supply. The major drawback of the low-pesticide-use strategy is that reductions in the rate of resistance development are achieved by reducing pesticide kill, which may cause inadequate control if effective alternative control methods are not available.

The effective management of pesticide resistance involves not only the judicious use of existing compounds but also the discovery and development of new and novel chemical control agents. However, no management strategy can prolong the useful life of a single insecticide indefinitely.

1.7. Aims of this Study

This study was initiated to consider the effect of reduced rates of commonly applied insecticides to the summer cereal ecosystem. The thesis falls into two broad sections: Chapters 2, 3, 4 and 5 investigate the effect of reduced rate insecticides on aphids and Chapters 6 and 7 consider the effect of actual or comparable rates used in Chapter 2 on a range of beneficial predators that inhabit the cereal ecosystem .

In Chapter 2, I adopted a step-wise procedure to study the effect of reduced rate insecticide applications of deltamethrin, dimethoate and pirimicarb against cereal aphid pests. The dose-response curves generated from the laboratory results were used to predict potential aphid population reductions under semi-field conditions. I was then able to adopt a more mechanistic approach to select insecticide rates to be used in the field, that were intended to give control of aphids comparable to currently recommended field rates.

In Chapter 3, I investigated the effect that post-treatment temperature had on the topical toxicity of λ-cyhalothrin and dimethoate in the laboratory. I did this as insecticides from different classes are known to have positive and negative temperature coefficients and I wanted to investigate the potential for enhancing the toxicity of reduced rate insecticides by considering the effect that temperature had on the two insecticides used in this chapter.

In Chapter 4, I again investigated the effect that post-treatment temperature had the toxicity of λ-cyhalothrin and dimethoate, this time however I increased the realism of the situation by investigating the toxicity coefficients of the insecticides when aphids were residually exposed to the insecticide λ-cyhalothrin both in the laboratory and field.

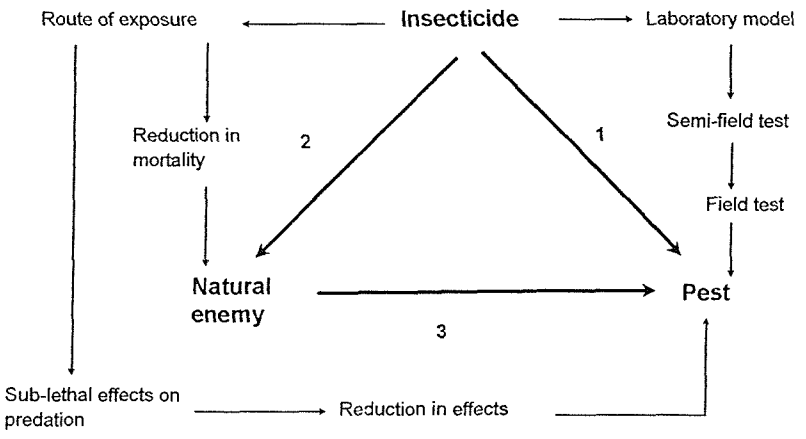
In Chapter 5, I investigated the effect that sublethal doses of λ-cyhalothrin had on individual measurements of aphid growth and development. I did this because a residual pest population may be left after a reduced rate insecticide application and it is important to know what affect this may have on subsequent aphid population growth. The

measurements of aphid growth and development are known to have effects on aphid population growth.

In Chapter 6, a group investigation of the residual toxicity of three insecticides, applied at a number of different rates, against a range of beneficial predators, necessitated the need to test actual or comparable doses of insecticides used in Chapter 2, against an important beneficial predator that inhabit the cereal ecosystem, on two different substrates. Residual toxicity was investigated because it was considered to be the route that beneficial insects are most likely to be exposed to an applied insecticide. Repeating the study on different substrates allowed predictions to be made as to the likely effect of applying a reduced dose rate of an insecticide on these insects.

In Chapter 7, I investigated the effect that exposure to reduced rate insecticides through topical, residual and dietary routes may have on the predatory potential of beneficial predators. This was considered necessary because the application of a reduced rate insecticide is intended to preserve beneficials in order to prevent further pest resurgence therefore any sub-lethal effect on their predatory potential may have consequences for pest control.

In Chapter 8, I draw together the results that have been generated consider the consequence of applying reduced dose rates not only from a farmers point of view but also from a biocontrol one. The limitations of the study are also considered as are suggestions for further research. A flow chart indicating the experimental framework of the study is shown in Figure 1.2.



Goals from applying reduced dose compared with conventional field rate applications

1 Control of pest population

2 Reduction in predator mortality

3 Increase in comparison to conventional application

Figure 1.2. Interaction triangle showing aims and approaches of using reduced dose compared to conventional field rate applications

CHAPTER 2

THE USE OF REDUCED RATE INSECTICIDES TO CONTROL APHID PESTS OF CEREALS.

INTRODUCTION

The cereal aphids *Sitobion avenae* (F.), *Rhopalosiphum padi* (Wlk) and *Metopolophium dirodum* (L.) (Homoptera: Aphididae) are sporadic pests of spring and winter-sown cereals in Great Britain (Carter *et al.*, 1980). The use of insecticides to control these aphids in the summer and autumn has increased over recent years (Sly, 1986; Rands *et al.*, 1988). During 1992, 3,422,188 ha of cereals were grown in Great Britain, although this was a 5% reduction compared to the area grown in 1990, there was only a 1% reduction in the total pesticide area treated (Davies *et al.*, 1993). Aphids, especially as transmitters of BYDV, were the principle pest of the 2,057,534 ha of wheat grown and 65% of the insecticide sprayed area, approximately 205,753 ha, was treated before the end of December. The most extensively used insecticides were cypermethrin (applied to 14% of the insecticide treated area), dimethoate (14%), gamma-HCH (9%, principally as a seed treatment), deltamethrin (7%) and fenvalerate (7%) (Davies *et al.*, 1993).

At present, few pesticides, with the exception of pirimicarb (ICI, 1989; Anon, 1995), demonstrate genuine physiological selectivity[1] between an aphid pest and its predators. Therefore options for obtaining any selectivity are related more to decisions about timing, targeting and dose, than the intrinsic selectivity of the insecticide (Taye & Jepson, 1988). Reducing the dose, achieved by reducing the quantity of active ingredient while maintaining spray volumes, has economic and environmental advantages. Labels accompanying pesticides often prescribe doses which may be unnecessarily high under many field situations (Metcalf, 1980). Dosage reduction may permit a greater survival of natural enemies while still offering satisfactory levels of pest control. This will arise through reduced exposure of all the arthropods in the crop environment. Selectivity may however be assisted by differences in the forms of the tolerance distribution exhibited by predators in comparison with their herbivorous prey (Hull & Beers, 1985; Poehling, 1987; van Emden, 1989; Wratten & Powell, 1991). These differences might lead to enhanced protection of predators, relative to their prey, if dosage is reduced.

[1]one which discriminates in terms of its toxicity between organisms to which it is applied (Hull & Beers, 1985).

Table 2.1. Table showing the degree of control over the experimental variables at each step in the progression of testing from the laboratory to the field. (+++ good control; ++ fair control; + poor control; - no control).

Experimental variables	Laboratory	Polytunnel	Open field
Aphid population structure[1]	+++	+	+
Substrate complexity and position of aphids[2]	+++	++	++
Control over exposure route to active ingredient[3]	+++	+	+
Dose impinging on aphids[4]	++	+	+
Temp/RH/L:D[5]	+++	++	-
Presence/absence of predators	+++	++	+

[1] numbers of aphids in each age class used in the experiment

[2] aphids are positioned on the plant were the insecticide may not directly impinge upon them i.e. under the flag leaf

[3] the applied insecticide may exert an effect through topical, residual, systemic or fumigant action, in consequence the actual route of exposure may not be known.

[4] dependent on spraying equipment used and aphid position on the target substrate

[5] dependent on ambient conditions of experimental area

Reduced doses may also maintain residual pest populations because of reduced efficacy as the rate gets progressively lower. This residual pest population may then provide food resources for the conserved natural enemy fauna of the crop. These natural enemies might then, remain within the crop and prevent any pest resurgence. Lower input of active ingredient will also reduce variable costs and provide an economic benefit. Pesticides represent a component of a farmers' variable costs, and reducing the application rate of costly active ingredients may be one of the few options for lowering costs of crop production (Poehling, 1990; Mann *et al*, 1991; Gren, 1994).

Large scale field experiments are expensive and time consuming. It would therefore be advantageous to know any relationship between the toxicity of a compound in the laboratory and its performance in the field. Developing methods that permit extrapolation between laboratory and field situations, might indicate the potential degree of pest control that could be achieved using reduced dose rate insecticide applications without the need for large scale field experiments. The degree of control over experimental variables decreases as experiments progress from laboratory through semi-field to field experiments however (Table 2.1). The increase in control benefits laboratory experiments, because the intrinsic toxicity of an insecticide can be quantified precisely under controlled conditions. There are however many advantages to the realism of the field situation.

This chapter describes the results of a simple step-wise progression from laboratory through semi-field to field experiments to investigate the potential for using reduced dose rates of currently recommended insecticides to control aphid pests of UK cereals.

MATERIALS AND METHODS

All aphids used in the experiments were cultured on barley (cv. Halcyon) in an insectary (18-22°C, 55-70% RH, 16:8 L:D). It had been intended to use *S. avenae* throughout, however, the culture did not provide enough individuals for all of the experiments, and *R. padi* was used as an alternative. Susceptibility to insecticides is often dependent on body mass (Busvine, 1971). When the aphids were weighed, the average weight of 10 individuals for both aphid species was found to be $2x10^{-4}$ g ($\pm$ $1x10^{-4}$ g). Since there is little evidence to the contrary, the two species were therefore considered to have similar susceptibility to the insecticides, permitting extrapolation of the results between species in the different experiments.

Laboratory bioassays;

Aphids from single instars (i.e. 4th instar) were used in the bioassays to minimise differences in susceptibility that could be attributed to changes in anatomy, physiology, size and behaviour of individual aphid instars (Busvine, 1971). All the laboratory insecticide treatments were delivered via Potter laboratory spray tower (Burkard Manufacturing Co. Ltd). The machine was calibrated to deliver insecticides at a volume rate of 200 l/ha (equivalent to conventional field rate applications in the UK).

In all the experiments distilled water was used as a control treatment and as a diluent for the insecticides. When assessing the effects of the treatments, organisms were scored as **alive** (moving as normal) or **knocked down** (moved when stimulated with a fine paint brush) or **dead** (no response after being stimulated with a fine paint brush).

The insecticides chosen were deltamethrin, a synthetic pyrethroid that has recently been approved for summer aphicide use, pirimicarb, a carbamate insecticide that selectively kills aphids and dimethoate, a broad-spectrum organophosphorous insecticide. The compounds are members of different insecticide classes with differing modes of action. Deltamethrin has contact and residual toxicity and its target site is the sodium channel of the nerve axon. Pirimicarb has contact, fumigant and translaminar activity, and dimethoate contact and systemic activity. The target site of both compounds is the enzyme acetylcholinesterase. This breaks down the neurotransmitter acetylcholine, responsible for carrying nerve impulses across the synapse (Hassell, 1982; Anon, 1990b).

Ten *R. padi* were placed on damp filter papers inside glass Petri dishes (9.5 cm in diameter) that had 'Fluon'-coated sides (Poly(tetrafluroethylene), ICI). For each of seven doses, five Petri dishes were sprayed making a total of 50 aphids per dose. The dishes were sprayed in sequential order with the lowest dose sprayed first. Control dishes were sprayed with distilled water. After spraying, the aphids were transferred to clean Petri dishes and covered with a ventilated chamber (Wiles, 1992). This consisted of a plastic pot (9 cm in diameter and 5 cm high) with the base removed and replaced by a piece of gauze. This was then inverted and placed over the Petri dish. Aphid mortality was recorded at 24 h.

Probit analysis was carried out on the 24 h dose-response data from the laboratory bioassays in order to obtain dose-response statistics (Finney, 1971). Abbott's formula was used to correct for control mortality (Abbott, 1925). Only dead aphids were

included in the calculations, however after 24 h few individuals remained knocked down. All statistical analyses were carried out using the MLP package (Ross, 1987). Contact toxicity ratios were calculated as follows; the doses (in g a.i./ha) giving 30 to 70% mortality were estimated in 5% mortality steps from the 24 h dose-response statistics for each compound. The values were transformed to percentages of the field rate application of the compound, and a ratio was calculated by dividing the dose (as a percentage of the field rate) at each mortality step by each other for the compounds being compared. Mean ratios of these values and the confidence limits around them was then calculated.

Semi-field experiments;

The laboratory work used a filter paper disc as the substrate for aphids from a single instar. The toxic route of exposure to the insecticide was through direct contact, any vapour activity of the compounds was minimal and systemic or translaminar modes of action did not have any effect and temperature and relative humidity were controlled. These are all factors which vary from a field situation and are known to affect insecticide toxicity.

Beyond the laboratory, an aphid may also be located between the grains on the ear or on the underside of the flag leaf of the cereal plant, where exposure to insecticides may be greatly reduced. In the present study it was considered necessary to carry out experiments under more realistic conditions than the laboratory but more controlled than the field situation: this was achieved by using mature plants in a polythene tunnel or glasshouse. Aphids were located on the ear and flag leaf and the population was made up of individuals from all instars. The aphids were exposed to contact, residual, systemic and translaminar routes of exposure of the insecticide and variations in dose caused by stratification of the applied spray through the crop. Carrying out the experiment in a polythene tunnel or glasshouse enabled some control over temperature, rainfall, daylength and presence or absence of predators.

Spring wheat (cv Alexandria) was sown (at an equivalent field density of 450 plants per m^2) in a 2:1 mixture of potting compost and sharp sand. The seeds were grown in wooden boxes (300 x 300 x 150 mm) in a glasshouse to growth stage (GS) 60 (Zadoks *et al.*, 1974; Tottman, 1987). At GS 60 the plants were transferred to a polythene tunnel and infested by draping leaves from a laboratory culture of *S. avenae* over the ears and flag leaves of the plants. Seven days later the plants were sprayed with one of five doses of deltamethrin (6 boxes per dose, see Table 2.2.). Control treatments were sprayed with water alone. The plants were removed from the tunnel for

spraying and placed on a concrete path. For each treatment, the plants for each dose were arranged in a line and sprayed using an Oxford precision sprayer fitted with a wet boom and four Lurmarck 02-F80 nozzles (BCPC Nozzle Code F80/0.80/3) spaced 50 cm apart and operated at 2 bar pressure. The sprayer was calibrated to deliver spray at a volume rate equivalent to the recommended field rate in UK cereals (200 l/ha). After spraying the plants were returned to the tunnel and arranged in a fully randomised layout once more. This design randomised any differences in temperature, light intensity or humidity in the tunnel on a treatment by treatment basis. The number of aphids on the ears and flag leaves of ten marked plants were recorded before spraying and for a further 7 days after. All the marked plants had 20 or more aphids before spraying. On each occasion the number of apterous and alate adults and nymphs (instars I-IV), was recorded. Temperature in the tunnel averaged 18 ± 5 °C, with a mean daylength of 10 h. Statistical analysis by ANOVA was carried out on $\log_{10}$ (X+1) transformed data using the SPSS statistical package (Version 6.0., SPSS corporation).

Table 2.2. Table showing the doses used in the semi-field and field experiments

Experiment	Insecticide	Dose applied as fraction of field rate
Semi-field	deltamethrin	1, 0.25, 0.13, 0.06, 0.03
	dimethoate	1, 0.33, 0.1, 0.03
Field	deltamethrin	1, 0.33, 0.2
	pirimicarb	1, 0.17, 0.1

The experiment was repeated using dimethoate (4 doses, 7 replicates, see Table 2.2.), this time however the plants were sown in 9 inch pots at an equivalent field density and moved to a glasshouse at GS 60. Aphid numbers were recorded on the ears of 10 randomly selected plants over the 7 days following spraying.

Field experiments;

Several years may pass before sufficiently large aphid populations develop to permit field experimentation to take place. Populations were therefore established within field cages to create sufficiently large aphid populations for experimentation. The advantages of this technique include improved microclimate, single species pest populations and reducing effects of predation. A 0.9 x 0.9 x 0.9 m field cage was constructed from Tygan (1 mm mesh) supported at the corners by a wooden stake (2.5 x 2.5 cm x 1.2 m).

Field trials were carried out at the Leckford Estate, Hampshire. In a field of winter wheat (c.v. Galahad) 35 0.9 m x 0.9 m plots were marked out and the wheat within each plot enclosed by a field cage. Aphid infested ears and flag leaves were collected from around the field site and used to inoculate the cages. Laboratory cultured *S. avenae* were also used to inoculate the cages by draping infested leaves over the ears and flag leaves of the plants. The cages were then assigned one of three doses of the insecticides deltamethrin or pirimicarb (5 replicates per dose, see Table 2.2). The plots were sprayed using the Oxford precision sprayer fitted with a wet boom and four Lurmarck 02-F80 nozzles (BCPC Nozzle Code F80/0.80/3) spaced 50 cm apart and operated at 2 bar pressure. The sprayer was calibrated to deliver spray at a rate equivalent to the recommended field rate in UK cereals (200 l/ha). Control treatments were sprayed with water only.

Aphid numbers were counted on 10 marked and 10 unmarked ears within each plot before and after spraying. Statistical analysis by ANOVA was performed on $\log_{10}$ X transformed data using the SPSS statistical package (Version 6.0., SPSS corporation).

Observed and Predicted aphid mortality in the semi-field and field experiments;

The 24 h dose-response equation generated from the laboratory data were used to predict reductions in aphid numbers following treatment with full and reduced dose rates of the insecticides in the semi-field and field experiments. The volumetric distribution of spray over wheat plants in the field was used to provide estimates of likely dose rate and permit extrapolation between laboratory and field situations.

The concentration (in g a.i./ha) for each dose to be applied was expressed in ng/ml and substituted into the data set of volumetric deposition at G.S. 73 (Cilgi & Jepson, 1992; Table 2) to calculate a deposition rate in cm^2 on the ear of a wheat plant. Using a density of 450 ears/m^2, the deposition rate was converted to g a.i./ha and inserted into the 24 h dose-response equation obtained in the present study. The calculated probit response was transformed back to a percentage mortality, and this 'predicted mortality' was compared with the results of the semi-field and field experiments. Calculating a 'predicted mortality' by this method assumed that the level of exposure, expressed as dose/unit area was equivalent to that which would land on the equivalent area of an aphid, that the aphids were fully exposed to the insecticide and not concealed within the crop canopy, and that the exposure any applied insecticide was through direct contact alone.

To compare the 'predictions' of aphid mortality generated from the dose-response data at each dose applied in the semi-field and field experiments, the 'observed percentage reduction' (OPR) in aphid numbers was calculated from the aphid counts of the semi-field and field experiments as follows;

$$OPR = ((Y - X)/Y) \times 100$$

where;

Y = Total number of aphids in all plots of treatment pre-spray

X = Total number of aphids in all plots of treatment on post-spray assessment day

When calculating the OPR by the above method it was assumed that a reduction in aphid numbers was solely a result of treatment with the insecticide, and that comparing aphid numbers on each assessment day with the pre-spray numbers would negate continued reproduction in the surviving population.

Comparison between 'observed percentage reductions' and 'predicted mortality' were made at 24 h, to compare data generated in the laboratory, and semi-field and field experiments after the same time periods. The 'predicted mortality' was also compared to 'maximum mortality', which was defined as the largest OPR calculated on any one post-treatment assessment day.

RESULTS

Laboratory bioassay;

For all three insecticides, aphid mortality was related to the dose applied 24 hours after treatment (Fig. 2.1a-c.). The 24 h dose-response data was subjected to probit analysis (Table 2.3.) and the LC_{50} values corresponded to 0.3, 1 and 0.9 % of the field rates for dimethoate, pirimicarb and deltamethrin respectively.

Since the lines were parallel it was possible to calculate contact toxicity ratios for the compounds based on their LC_{50} values as a percentage of their field rate applications (Table 2.4). The value of the contact ratios for dimethoate and pirimicarb and pirimicarb and deltamethrin were close to 1, indicating their toxicities at the same percentage of field rate application were very similar. The contact ratio between deltamethrin and dimethoate was greater than 1, indicating for the same 'percentage' of their respective

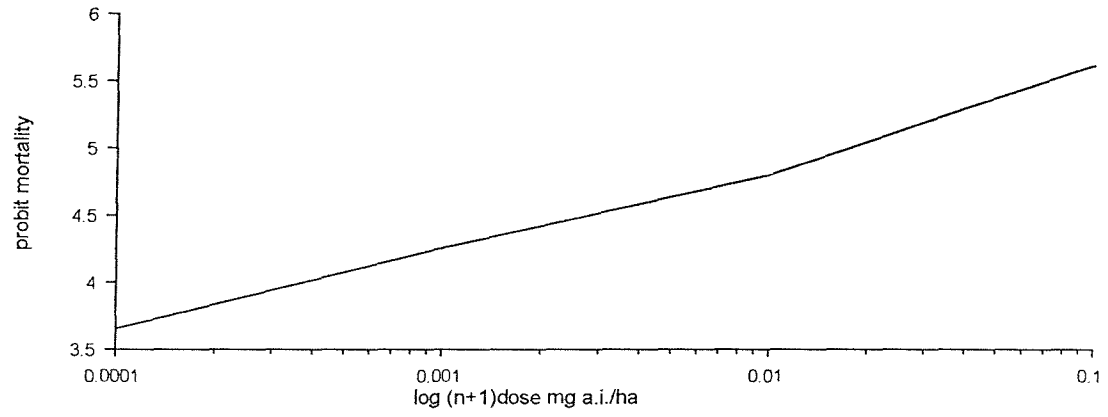


Figure 2.1a. Graph of 24 h probit mortality for aphids treated with deltamethrin.

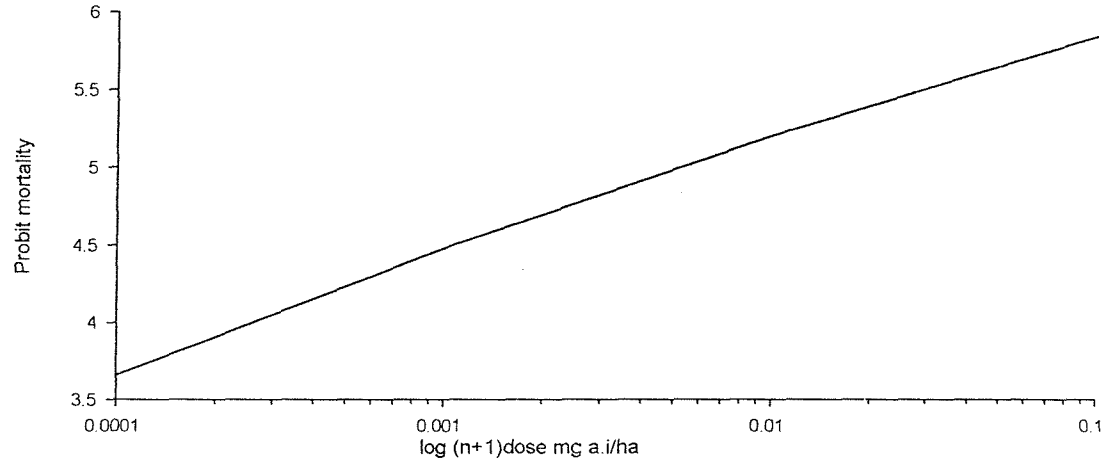


Figure 2.1b. Graph of 24 h probit mortality for aphids treated with dimethoate.

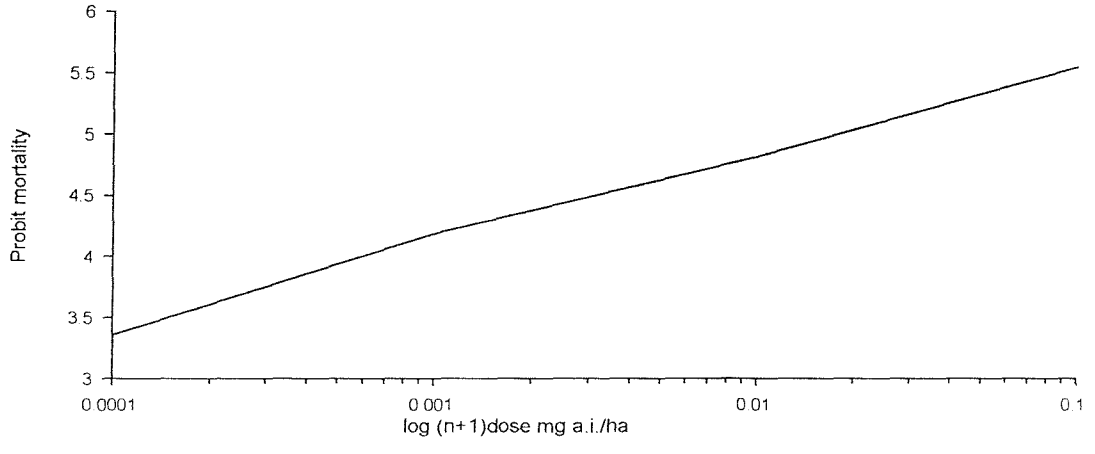


Figure 2.1c. Graph of 24 h probit mortality for aphids treated with pirimicarb.

field rates dimethoate was more toxic, Deltamethrin is however intrinsically more toxic than both dimethoate and pirimicarb. This is revealed by the LC_{50} values (Table 2.3.) and its application rates which is over fifty times less than dimethoate and twenty times less than pirimicarb (Table 2.2).

Table 2.3. 24 h probit statistics of response of *R. padi* to deltamethrin, dimethoate and pirimicarb.

Insecticide	Probit slope	LC_{50} (fiducial limits) (detransformed) (g a.i./ha)	Heterogeneity C^2 (d.f.) Significance[a]
deltamethrin	0.93	0.06 (0.03 - 0.15)	5.54 (5) ns
dimethoate	1.03	1.02 (0.54 - 1.84)	5.56 (5) ns
pirimicarb	0.94	1.40 (0.64 - 4.05)	10.55 (5) ns

Test of Parallelism; X^2 = 0.306 (2) ns
[a] Significance level (p=0.05): ns = not significant

The next step in the progression for extrapolating laboratory studies to a field situation was semi-field experimentation. The insecticide rates used in the semi-field experiment were chosen by inserting a range of rates into the 24 h dose-response equation to obtain a 'predicted mortality'. The relationship between performance in the laboratory and 'field' would then be verified by comparing the 'predicted mortality' with OPR. The correlation would then be used to select the insecticide rates used in the field experiment.

Table 2.4. Contact toxicity ratios (when expressed as a percentage of their respective field rates) for the insecticides used in the laboratory experiments.

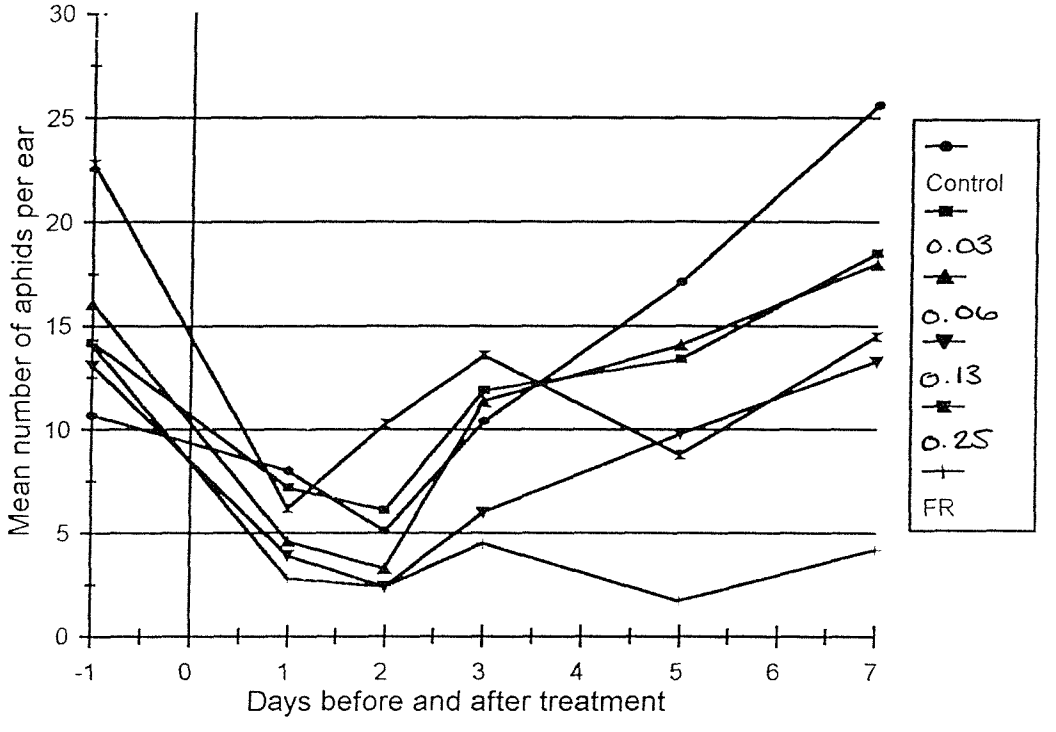
Insecticides	Mean ratio* (± 95% C.I)
deltamethrin vs dimethoate	1.41 (± 0.18)
dimethoate vs pirimicarb	0.81 (± 0.51)
deltamethrin vs pirimicarb	1.01 (± 0.53)

* Ratio calculations are explained in the text.

Semi-field Experiment;

Prior to spraying aphid densities in all treatments exceeded the economic threshold of five aphids per ear (George & Gair, 1975) and there were no significant differences in aphid numbers between replicates (d.f.=5, F-ratio=1.17, p>0.05). In all treatments mean numbers of aphids on each plant declined until two days after spraying, and on the final assessment day mean numbers of aphids in each replicate were

Figure 2.2. Mean number of aphids per plant before and after treatment with deltamethrin in the semi-field experiment. The treatments are explained in table 2.2.



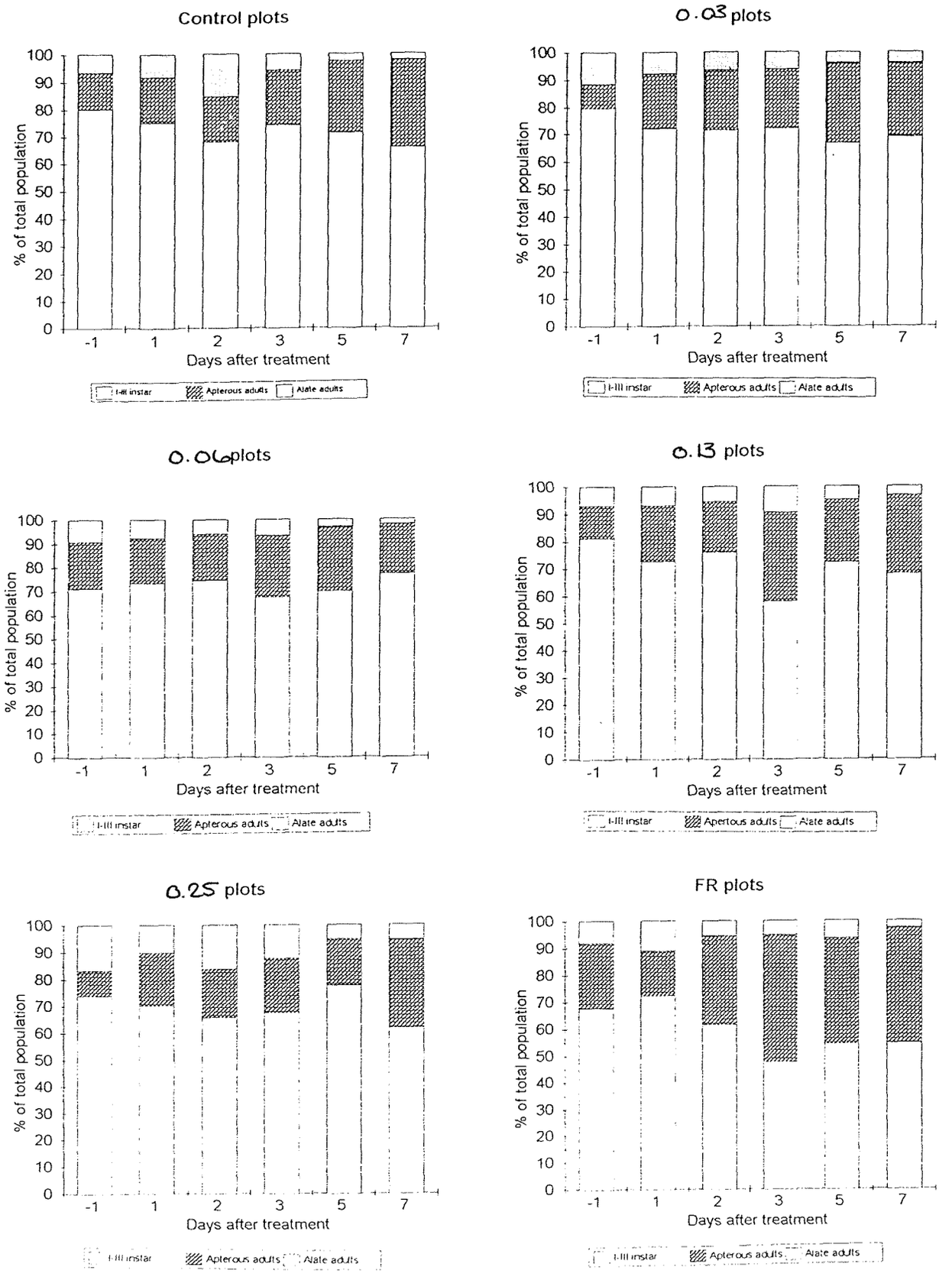


Figure 2.3. Percentage of individuals in each aphid class before and after treatment with deltamethrin in the semi-field experiment.

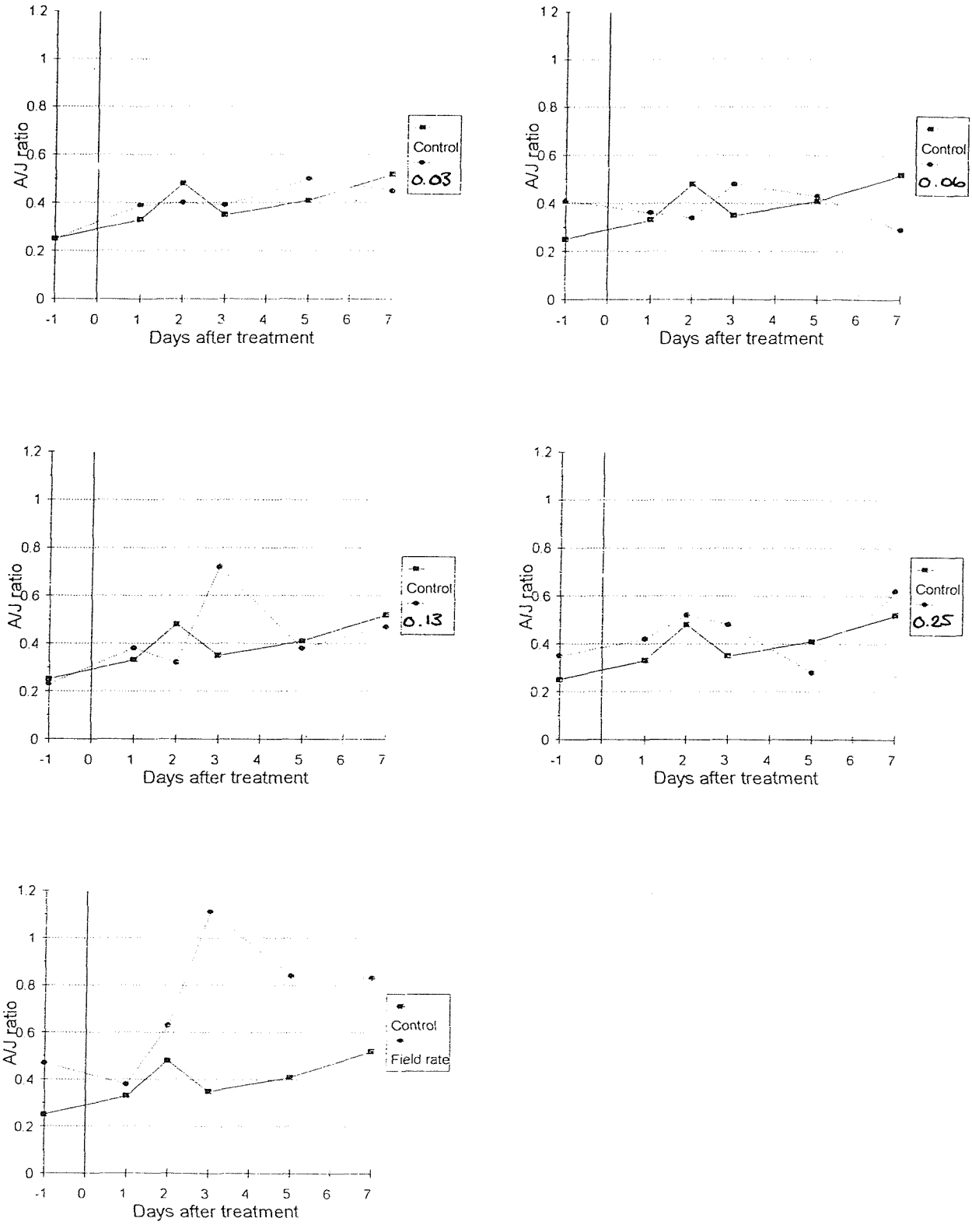


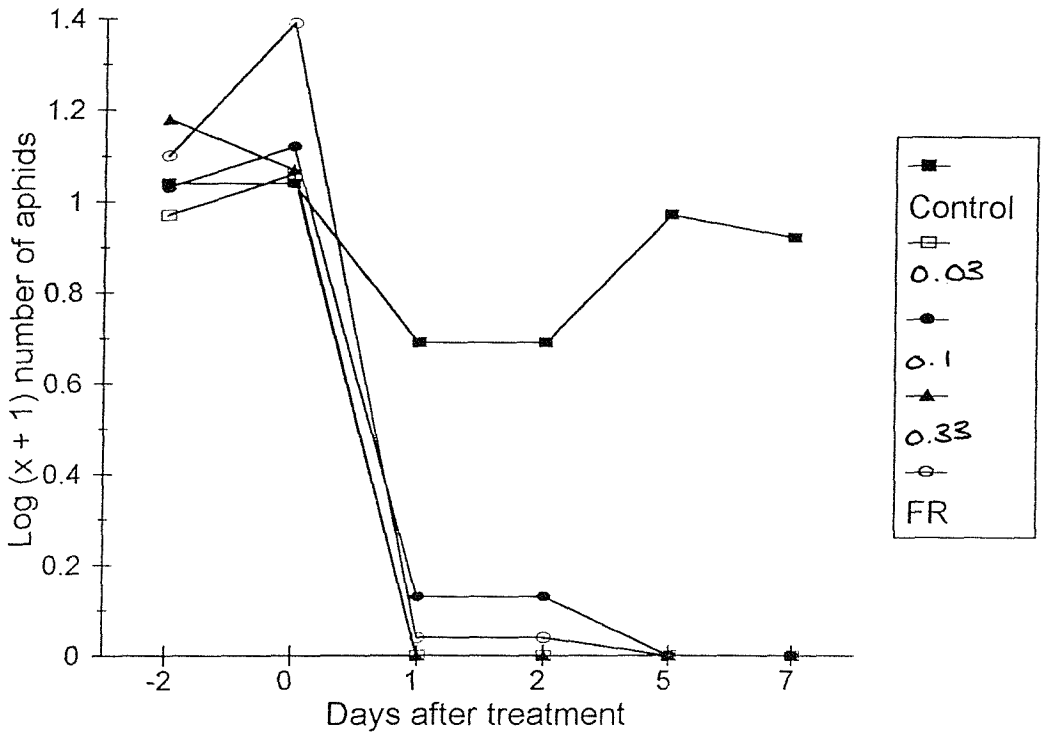
Figure 2.4. Adult:Juvenile ratios for the treated compared to control in the semi-field experiment with deltamethrin.

Table 2.5. Statistical differences calculated by Tukey HSD in aphid numbers on the post-treatment assessment days for the deltamethrin treated plants in the semi-field experiment.

Days after treatment	deltamethrin rate (Fraction of field rate)	Mean (Log X ± SE) aphid numbers per plant	Tukey HSD*		F ratio (d.f)
1	Control	1.81 ± 0.14	a		2.10 (29)
	0.03	1.84 ± 0.07	a		
	0.06	1.59 ± 0.14	a		
	0.13	1.57 ± 0.07	a		
	0.25	1.65 ± 0.15	a		
	Field rate	1.34 ± 0.15	a		
2	Control	1.62 ± 0.14	a		1.55 (29)
	0.03	1.76 ± 0.07	a		
	0.06	1.53 ± 0.04	a		
	0.13	1.34 ± 0.09	a		
	0.25	1.60 ± 0.24	a		
	Field rate	1.35 ± 0.10	a		
3	Control	1.93 ± 0.16	a		2.15 (29)
	0.03	2.01 ± 0.11	a		
	0.06	2.05 ± 0.04	a		
	0.13	1.76 ± 0.07	a		
	0.25	1.98 ± 0.15	a		
	Field rate	1.57 ± 0.15	a		
5	Control	2.11 ± 0.19	a		7.30 (29)
	0.03	2.11 ± 0.06	a		
	0.06	2.13 ± 0.06	a		
	0.13	1.90 ± 0.13	a		
	0.25	1.74 ± 0.19	a	b	
	Field rate	1.23 ± 0.07		b	
7	Control	2.31 ± 0.17	a		4.21 (29)
	0.03	2.24 ± 0.07	a		
	0.06	2.24 ± 0.06	a		
	0.13	2.06 ± 0.13	a	b	
	0.25	1.84 ± 0.23	a	b	
	Field rate	1.44 ± 0.22		b	

* different letters indicate significant differences in aphid numbers between the treatments.

Figure 2.5. Mean number of aphids (Log (x +1) on plants before and after treatment with dimethoate in the semi-field experiment.



inversely related to the amount of active ingredient sprayed. The day at which pre-treatment levels of aphids were exceeded depended on the deltamethrin dose applied. This was five days after spraying for the 0.03 and 0.06 rates and seven days after spraying for the 0.13 and 0.25 rate treated replicates. Numbers in the field rate treated replicates did not exceed pre-treatment levels on any occasion (Fig. 2.2.).

There were no significant differences between aphid numbers in the different treatments during the first three post-treatment assessment days. On the penultimate assessment day aphid numbers in the field rate treatment were significantly different to all the treatments except the 0.25 rate. On the final assessment day, aphid numbers in the field rate treatment were significantly different to all other treatments except the 0.13 and 0.25 rate applications (Table 2.5.).

In general, nymphs were the most numerous age class of aphids and alatae the smallest (Fig. 2.3.). Apart from the field-rate treatment, the percentage of individuals in each age class were similar over the post-treatment days and indicted that no single aphid class was selectively killed within the replicates. In the field-rate treatment, apterous individuals were the largest population group three days after treatment; this may indicate that there was a very low level of reproduction taking place on the first few days after treatment. The adult:juvenile ratio was calculated by dividing the total number of adults (apterous and alate) by the total number of nymphs in each treatment (Fig. 2.4.). The ratios were similar between control and all treatments except the field-rate. In the field-rate treatment the ratio was higher than the control because adults accounted for over 50% of the population (Fig. 2.3.).

The 'predicted mortality' from the laboratory generated dose-response data and OPR from the aphid counts after 24 h was comparable for the 0.13 rate application only. The OPR was lower than the 'predicted mortality' for FR, 0.25, 0.06 and 0.03 rate applications. The 'predicted mortality' correlated better with maximum OPR for all fractions of field rate (Table 2.6.).

After treatment with dimethoate the number of aphids in all treatments declined until 2 days after spraying, numbers only increased in the control replicates (Fig. 2.8.). Five days after spraying there were no aphids on the ears treated with all doses of dimethoate. The poor control achieved at the two lowest rates suggested that they were unsuitable for use in a field situation. Therefore, it was decided (after consulting the literature (i.e. Poehling, 1985, 87 & 89; Wratten & Powell, 1991) and the results of the semi-field experiment) that the insecticide rates to be used in the field experiment should lie between 0.1 and the full field application rate.

Table 2.6. Predicted mortality and OPR of aphids treated with deltamethrin in the semi-field experiment. Calculations are explained in the text.

Insecticide rate (Proportion of field rate)	Predicted % mortality from 24 h dose response data	OPR after 24 h	Maximum OPR
Field rate	75	69	83[c]
0.25	65	48	54[c]
0.13	60	64	69[b]
0.06	50	48	48[a]
0.03	45	38	38[a]

[a] value 1 day after treatment
[b] value 2 days after treatment
[c] value 3 days after treatment

Field Experiment;

The number of aphids on the marked and unmarked ears within each plot were analysed separately and found to follow the same trends. Therefore to reduce repetition, only the results for the marked ears, where aphid numbers were higher and exceeded five per ear before spraying, are presented here.

There were no significant differences in aphid numbers between plots before spraying with deltamethrin (d.f.=4, F-ratio= 1.82, $p>0.05$). In the control plots aphid numbers peaked six days after spraying and then began to decline, and were at their lowest 22 days after treatment. In all the deltamethrin treated plots, aphid numbers declined until three days after spraying and then began to increase until 14 days after spraying (Fig. 2.6.), and were significantly different from the control plots (Table 2.7.). After 14 days aphid numbers in the deltamethrin treated plots began to decline in line with those of the controls, and 18 days after spraying there were only significant differences in aphid numbers in the control and field rate treated plots. On the final assessment day there were no significant differences in aphid numbers in any of the treatments. Throughout the post-spray sampling period there were no significant differences between aphid numbers in any of the deltamethrin treated plots (Table 2.7.).

There were no significant differences in aphid numbers between plots before spraying with pirimicarb (d.f.=4, F-ratio= 3.32, $p>0.05$). After spraying there were differences in the level of control achieved between the different rates of pirimicarb (Fig 2.7.). Aphid numbers in the control plots were significantly different to all treated plots up

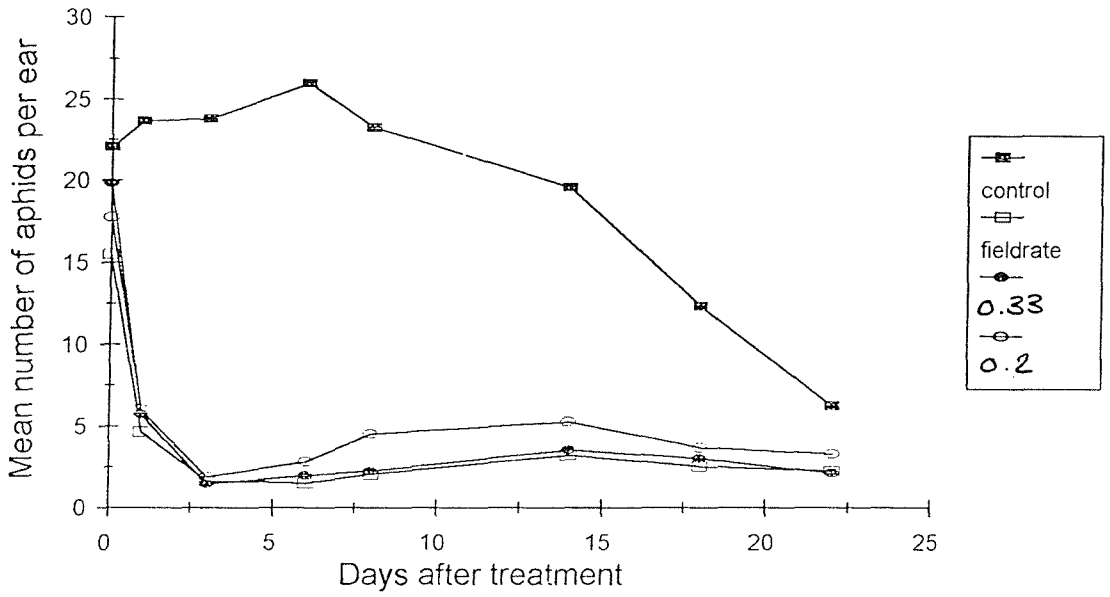


Figure 2.6. Mean number of aphids per ears on plants in field plots treated with deltamethrin.

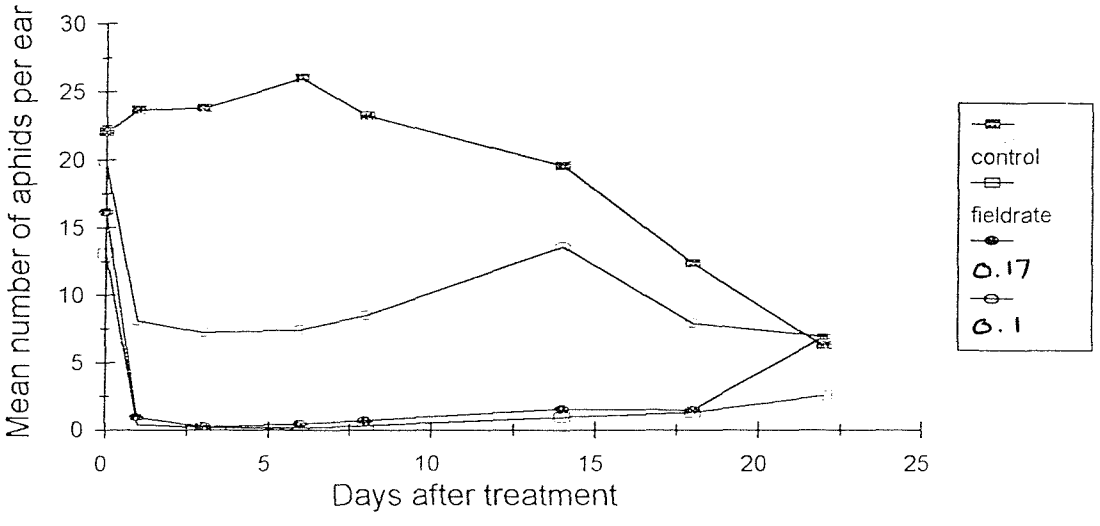


Figure 2.7. Mean number of aphids per ears on plants in field plots treated with pirimicarb.

Table 2.7. Statistical differences calculated by Tukey HSD for aphid numbers on plants after treatment with deltamethrin in the field experiment.

Day after treatment	Deltamethrin rate (Fraction of field rate)	Mean (Log X ± SE) aphid numbers per plant	Tukey HSD*	F ratio (d.f.)
1	Control	2.36 ± 0.03	a	14.54 (19)
	Field rate	1.62 ± 0.11	b	
	0.33	1.72 ± 0.11	b	
	0.2	1.74 ± 0.09	b	
3	Control	2.37 ± 0.05	a	23.97 (19)
	Field rate	1.11 ± 0.16	b	
	0.33	1.05 ± 0.16	b	
	0.2	1.23 ± 0.10	b	
6	Control	2.41 ± 0.04	a	18.54 (19)
	Field rate	1.01 ± 0.20	b	
	0.33	1.23 ± 0.12	b	
	0.2	1.31 ± 0.17	b	
8	Control	2.34 ± 0.07	a	7.46 (19)
	Field rate	1.07 ± 0.25	b	
	0.33	1.31 ± 0.09	b	
	0.2	1.33 ± 0.31	b	
14	Control	2.28 ± 0.06	a	5.74 (19)
	Field rate	1.36 ± 0.18	b	
	0.33	1.52 ± 0.08	b	
	0.2	1.52 ± 0.27	b	
18	Control	2.02 ± 0.12	a	3.25 (19)
	Field rate	1.11 ± 0.25	b	
	0.33	1.40 ± 0.16	a b	
	0.2	1.34 ± 0.28	a b	
22	Control	1.66 ± 0.18	a	1.91 (19)
	Field rate	1.11 ± 0.24	a	
	0.33	1.16 ± 0.19	a	
	0.2	1.56 ± 0.12	a	

* different letters indicate significant differences in aphid numbers between the treatments.

Table 2.8. Statistical differences calculated by Tukey HSD for aphid numbers on plants after treatment with pirimicarb in the field experiment.

Day after treatment	Pirimicarb rate (Fraction of field rate)	Mean (Log X ± SE) aphid numbers per plant	Tukey HSD*	F ratio (d.f.)
1	Control	2.36 ± 0.03	a	39.65 (19)
	Field rate	0.60 ± 0.17	b	
	0.17	0.94 ± 0.13	b	
	0.1	1.84 ± 0.14	c	
3	Control	2.37 ± 0.05	a	69.12 (19)
	Field rate	0.41 ± 0.13	b	
	0.17	0.48 ± 0.17	b	
	0.1	1.81 ± 0.10	c	
6	Control	2.41 ± 0.04	a	50.45 (19)
	Field rate	0.38 ± 0.10	b	
	0.17	0.52 ± 0.23	b	
	0.1	1.85 ± 0.12	c	
8	Control	2.34 ± 0.07	a	31.75 (19)
	Field rate	0.63 ± 0.09	b	
	0.17	0.67 ± 0.22	b	
	0.1	1.82 ± 0.17	a	
14	Control	2.28 ± 0.06	a	28.20 (19)
	Field rate	0.98 ± 0.10	b	
	0.17	1.10 ± 0.19	b	
	0.1	2.08 ± 0.12	a	
18	Control	2.02 ± 0.12	a	15.67 (19)
	Field rate	1.06 ± 0.15	b	
	0.17	1.15 ± 0.09	b	
	0.1	1.84 ± 0.11	a	
22	Control	1.66 ± 0.18	a b	5.18 (19)
	Field rate	1.40 ± 0.07	a b	
	0.17	1.26 ± 0.07	b	
	0.1	1.82 ± 0.08	a	

* different letters indicate significant differences in aphid numbers between the treatments.

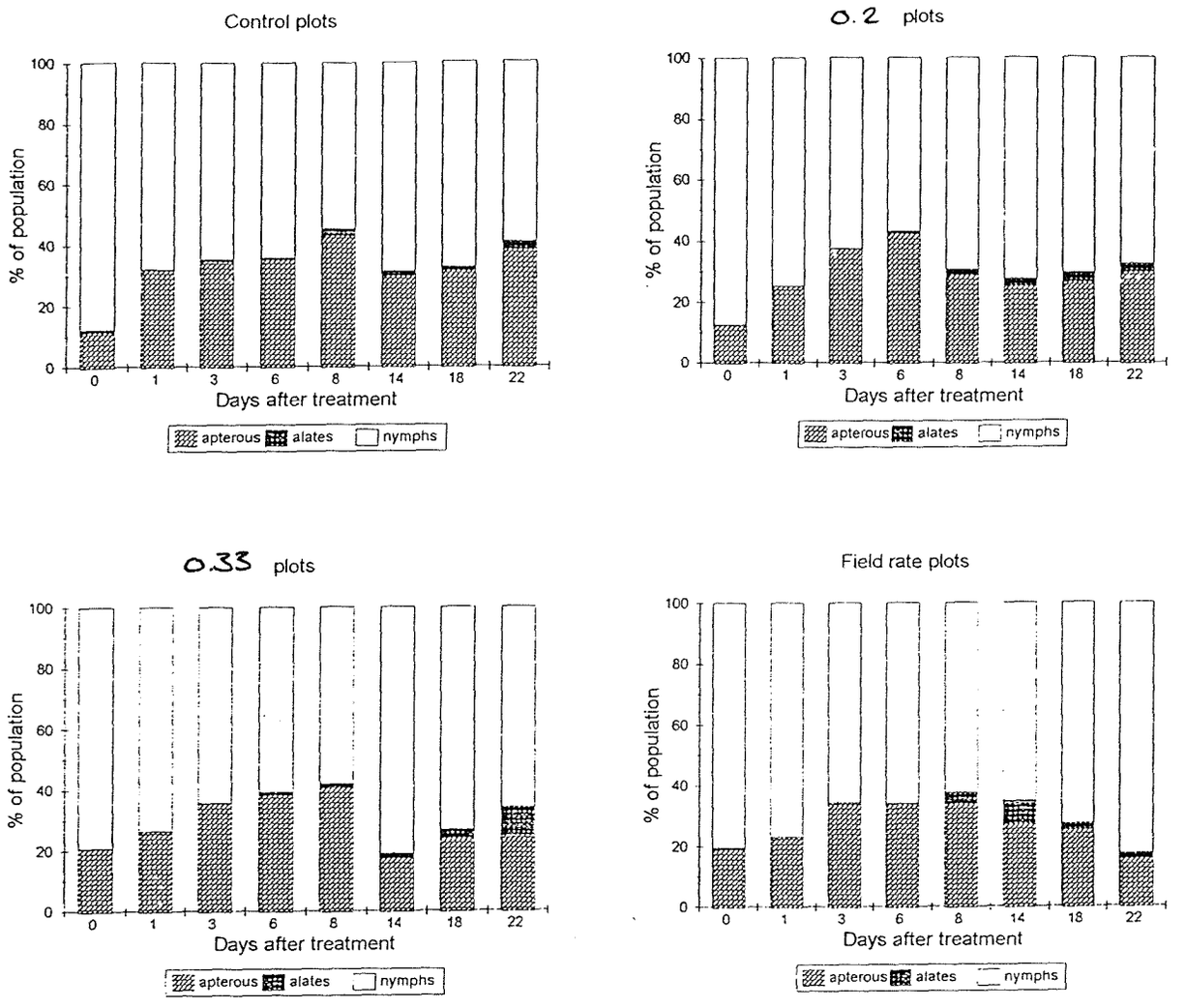


Figure 2.8. Percentage of individuals in each aphid class before and after treatment with deltamethrin in the field experiment.

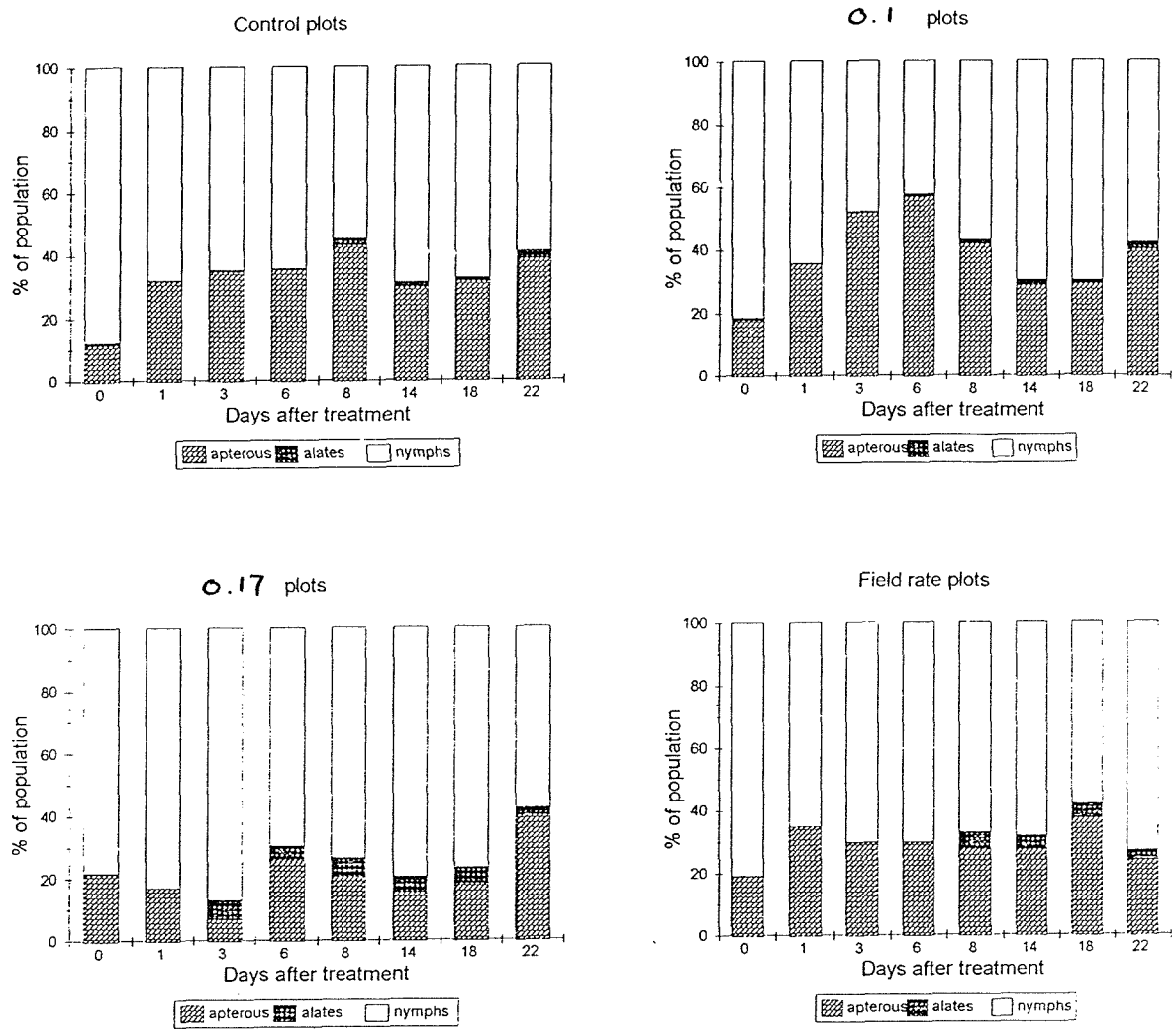


Figure 2.9. Percentage of individuals in each aphid class before and after treatment with pirimicarb in the field experiment.

Table 2.9. Predicted % mortality and OPR in aphid numbers after treatment with deltamethrin in the field experiment. Calculations are explained in the text.

Insecticide rate (fraction of field rate)	Predicted % mortality from 24 h dose response data	OPR after 24 h	Maximum OPR[a]
Field rate	75	70	90
$^1/_3$	65	65	90
$^1/_5$	60	62	87

[a] value 3 days after spraying

Table 2.10. Predicted % mortality and OPR in aphid numbers after treatment with pirimicarb in the field experiment. Calculations are explained in the text.

Insecticide rate (fraction of field rate)	Predicted % mortality from 24 h dose response data	OPR after 24 h	Maximum OPR[a]
Field rate	75	98	99
$^1/_6$	55	93	98
$^1/_{10}$	60	42	50

[a] value 3 days after spraying

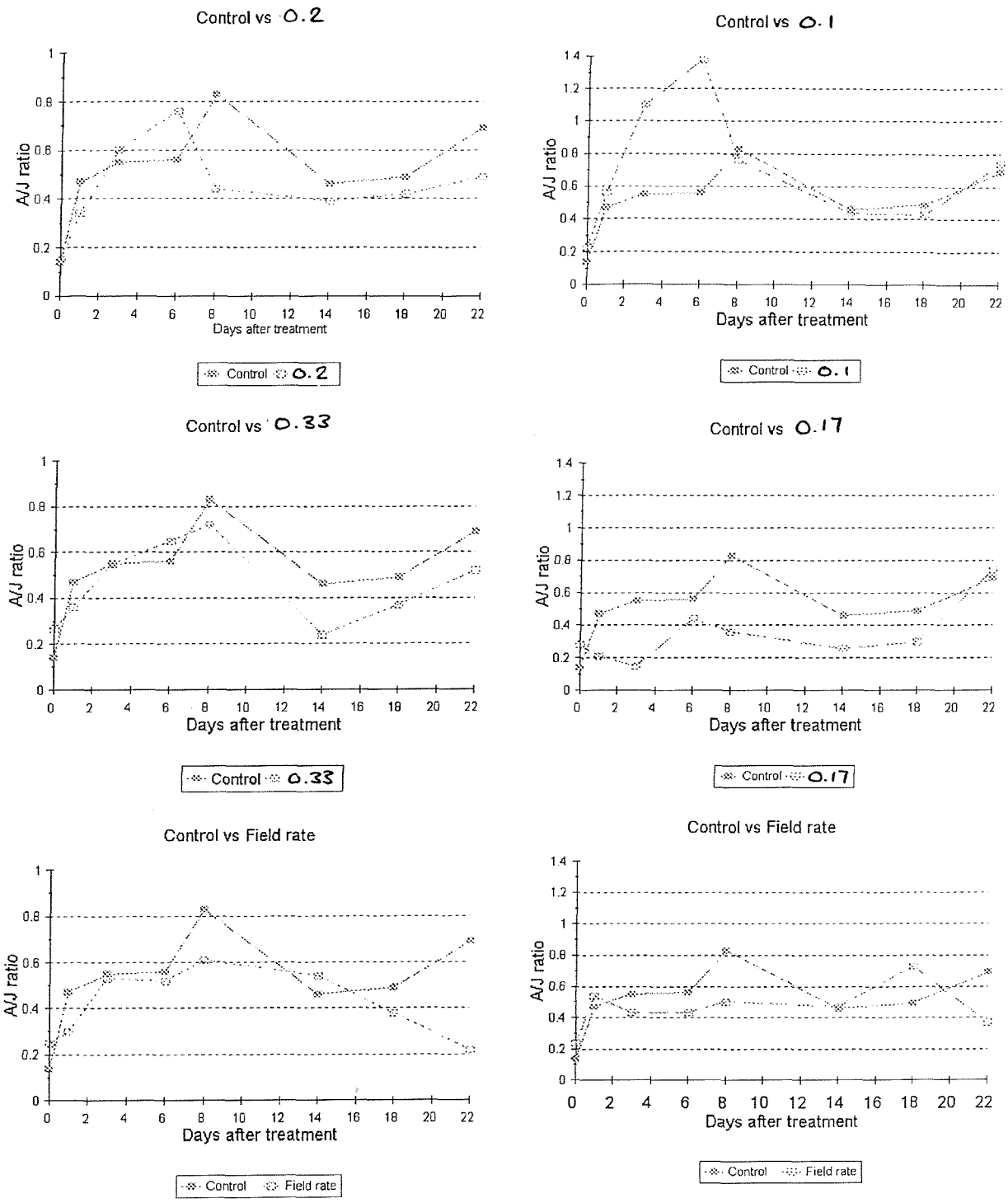


Figure 2.10. Adult:Juvenile ratios for the treated compared to control in deltamethrin (left hand side) an pirimicarb (right hand side) treated plots.

to six days after treatment (Table 2.8.). In the 0.1 rate treatment there was a reduction in aphid numbers until three days after spraying, when they began to increase until 14 days after spraying, they then mirrored the decline showed by the control plots. Between eight and 18 days after treatment the control and 0.1 rate plots were not significantly different from each other (Table 2.8). The number of aphids in the 0.1 rate treated plots were significantly different to those in the other pirimicarb treated plots over all the post-spray assessment days (Table 2.8). Aphid numbers in the 0.17 and field rate treated plots were significantly different from the control plots but not each other until the final assessment day (Table 2.8.). On the final assessment day there were significant differences between the 0.1 and 0.17 rate treated plots only (Table 2.8.). Compared to the deltamethrin treated plots, aphid control in absolute numbers of aphids was better in the pirimicarb treated plots (Fig 2.7.).

The graphs show the aphid populations in all plots were mainly composed of nymphs (Figs 2.8. & 2.9.). Alate individuals were present throughout the sampling period in the control plots and before spraying in the deltamethrin treatment plots (Fig. 2.8.). Alates were absent until six days after spraying in the 0.2 and 0.33 rate treated plots and eight days after spraying in the field rate plots. For aphid populations sprayed with pirimicarb alates were present before spraying only in the 0.1 fraction field rate treated plots (Fig 2.9.) and persisted throughout the post-spray assessment period. Alatae appeared three days after spraying in the 0.17 rate and after eight days in the field rate treated plots. The adult:juvenile ratios, in general, were lower in the deltamethrin treated plots than the controls (Fig. 2.10.). The ratios of the 0.17 and field rate treatments of pirimicarb tended to be lower than the controls, whilst the 0.1 rate was generally higher. It also tended to be higher than the corresponding values for the other pirimicarb treated plots.

The 'predicted mortality' and OPR after 24 h for deltamethrin fitted well for all three rates used (Table 2.9.). Although, maximum OPR was much higher than the 'predicted mortality' for all three rates. The OPR after 24 h was higher than the 'predicted mortality' for the 0.17 and field rate treatments of pirimicarb and lower for the 0.1 rate (Table 2.10). The maximum OPR for pirimicarb was higher than the 'predicted mortality' for the 0.17 and field rate treatments and lower than the 'predicted mortality' for the 0.1 rate. Comparisons between the predicted mortality and OPR could only be done over the first four assessment days because percentage control of the aphids could not be calculated after this.

DISCUSSION

The experiments described in this chapter followed a step-wise progression from the laboratory through to semi-field and finally to field trials for testing the efficacy of reduced rate insecticides against aphid pests found in the UK. The progression was based on a model generated from laboratory dose-response data to select application rates used in the field. In order to quantify the validity of using a model dependent upon the correlation between laboratory bioassay results and subsequent results from field tests, it is necessary to compare the experimental variables suggested by Table 2.1. before an overview of the methodology can be undertaken.

The laboratory bioassay measured the intrinsic toxicity of the insecticides, avoiding any behavioural effects on the insect (Devonshire & Rice, 1989). Although a pest may be susceptible to reduced doses in the laboratory, it does not necessarily follow that this will be mirrored in a semi-field or field situation. Most methods of applying insecticides are extremely inefficient when utilisation is expressed in terms of the proportion of the applied dose taken up by the target organism (Graham-Bryce, 1977). Therefore dosage reductions achievable in the field may be more limited than those suggested by the laboratory results. The semi-field experiment was more complex than the laboratory, and when compared to the field experiment there were still many differences (Table 2.1.). Conditions within the polythene tunnel and glasshouse were ideal for plant and aphid population growth. Colonies of aphids such as *S. avenae* are at their most fecund between flowering (GS 60) and milky-ripe (GS 73) stages of crop growth (Watt, 1979; Holt *et al.*, 1984; Carter *et al.*, 1989). In the absence of biotic and abiotic factors in the field that check population growth, aphid numbers would be expected to, and did, rise very rapidly. Weather and temperature affect the development of aphid populations (Dean, 1974; Dixon *et al.*, 1982; Watson, 1983). The reproductive rates of aphids increase with increasing temperature (Dean, 1974; Acrenna and Dixon, 1989) and temperatures within the range of 20 to 25°C inside the polythene tunnel and glasshouse were ideal for high aphid reproductivity (Dixon, 1989). High reproductive rates coupled together with dry conditions seen in the polythene tunnel and glasshouse, lead to rapid crop growth and a very quick increase in cereal aphid numbers (Cater *et al.*, 1980). There were no aphid-specific or polyphagous predators present at any time during the semi-field experiment. It is well known that aphid specific and ground dwelling polyphagous predators are effective at preventing outbreaks of cereal aphids and their absence was one reason for such high aphid numbers (Sunderland *et al.*, 1980; Chambers *et al.*, 1983; Adams, 1984; Chambers *et al.*, 1986; Winder, 1990 Wratten and Powell, 1991).

In the field experiment the population build up reflected a moderate increase in aphid numbers where critical densities were reached late in the season (since plants were inoculated after flowering had begun). The results show that control of this type of population build up in the field is not significantly different to a field rate application when using insecticide treatments at rates lower than those currently recommended. However, in years where there is an early build up of pests to critical levels, spraying before flowering may leave a residual population of aphids that could build up again and reach damaging levels before the end of flowering (Carter *et al.*, 1989). The 0.1 dose of pirimicarb reduced the initial aphid infestation but aphid numbers began to increase eight days after spraying (Fig 2.9). The rise may be due to immigration of alate aphids into the crop or reproduction by surviving aphids in the crop (ffrench-Constant *et al.*, 1987). Five days after the insecticides had been applied it did rain. This may have affected the toxicity of the compound by washing residual deposits of the plant. A dose as low as 0.1 may have been rendered non-toxic to any alates immigrating into the crop therefore.

Alate individuals colonise cereals in the spring (Dixon & Glen, 1971; Dean, 1973; Dewar, 1977; Watt, 1979) and give birth to more fecund apterae (Wratten, 1977; Carter *et al.*, 1980). When the pest population is increasing numerically it will be composed mainly of apterous adults and their offspring. Alteration of the structure after an insecticide application has consequences for future population growth and can be considered through calculation of the adult:juvenile ratio. A reduction in the A:J ratio after spraying results from the mortality or emigration of adults to other areas of the crop. Consequently, in the absence of immigration and until any nymphs reach maturity and begin to reproduce, there will be a delay before aphid numbers start to increase. An increase in the ratio resulting from the mortality or movement of nymphs, may offset a decrease in population numbers after spraying because adults remaining have the potential to begin reproducing immediately. In the semi-field experiment after treatment with deltamethrin, the A:J ratio of all treatments increased with respect to the control, but the number of aphids did not increase significantly (Fig. 2.4.). This was probably a result of the stochastic numbers of aphids present after spraying. After treatment with the 0.1 rate of pirimicarb the A:J ratio also increased and aphid numbers did increase at a greater rate than the other pirimicarb treatments (Fig. 2.10.).

The use of reduced rates of insecticides to control aphid pest populations has been verified by Storck-Weyhermuller (1987) and Poehling (1987 & 1990). After treatment with the pyrethroid fenvalerate, the results were similar to that of deltamethrin in this study (Poehling, 1990). Pirimicarb was also not as effective at the lowest rate in the study of Poehling (1987), ironically this was also applied at 0.1 of the rate recommended in the UK. One aim of using reduced rates in this present study was to

maintain a residual pest population and avoid starvation effects on predators (Poehling, 1987). No field rate application of an insecticide gave 100% control of the pest, but it is not known if the number of aphids left would have been sufficient to prevent any starvation effects and movement of predators out of the crop. Questions have always been asked about the compatibility of high degrees of efficacy of an insecticide towards the target pest when simultaneously attempting to protect predators whose population dynamics are closely associated with it (Stern *et al.*, 1959; Cavalaro, 1983). Reduction in effects are associated with the use of reduced rate applications; only in areas treated with reduced rates and leaving a sufficiently large pest population can large proportions of aphid specific predators complete their development (Poehling, 1989). Apart from the close relationship between pest and predator, especially relatively immobile larval stages, being impaired by elimination of the pest there may also be subtle sub-lethal effects on predators that are difficult to measure during field trials (Schweizer *et al.*, 1988). Storck-Weyhermuller (1987) showed that after contamination with the full rate of pirimicarb as larvae, the fertility of *Episyrphus balteatus* (Diptera: Syrphidae) and fecundity of *Coccinella septumpunctata* (Coleoptera: Coccinellidae) were both reduced. The effects could be reduced by lowering the application rate.

The validity of using laboratory generated data is dependent on the correlation between laboratory bioassay results and subsequent field trial results. A number of assumptions were made when extrapolating the data, the question is were they relevant for a field situation? The 'predicted mortality' compared to the OPR was better in the field (Table 2.9.) than the semi-field (Table 2.6.) experiment for deltamethrin. This was due to conditions in the glasshouse being ideal for population growth, the exclusion of factors such as predation and vagaries of climate, meant population increases would only have been affected by the targeting and efficacy of the insecticide application. In the field trial the combination of all factors would have helped to suppress aphid populations. The lower than expected aphid control in the semi-field and field experiments at certain doses highlight the caution needed when considering the application rate to use.

The laboratory bioassay measured the intrinsic contact toxicity of the insecticides, any assumptions made were therefore based solely on one route of toxicity. The three compounds vary in their routes of toxicity, all demonstrate contact toxicity but dimethoate has systemic and pirimicarb fumigant properties. The importance of these route was not taken into account when considering the basic model of dose-selection criteria. The predictions made solely from contact toxicity appear to be satisfied in the field however. It appears that the aphid in the middle of a Petri dish is not that anomalous to an aphid on the ear of a wheat plant. Both aphids will be saturated by the high volumes applied and in the field this may nullify any differences in the mode of action between the

insecticides. Increases in the OPR above that predicted may be due to other modes of action supplementing the contact toxicity of the insecticide.

It appears for the aphid *S. avenae* on the ear of a wheat plant predictions generated from laboratory dose-response data can be validated in the field because of the importance of contact toxicity as an exposure route. Predictions are easier to make than for predators and other aphid species. *R. padi* is primarily found on the underside of the flag leaf where exposure by direct contact will be reduced by stratification of the insecticide through the crop and protection afforded by sitting underneath the leaf. The extrapolation of data from the laboratory to the field is more difficult for predators (Jepson, 1987 & 1993). This is because predators may be exposed to applied insecticides through contact, residual or dietary toxicity. Which in turn will be affected by factors such as predator phenology, sorption and distribution of the insecticide and availability of contaminated prey. Consequently assessing effects on predators in the field (i.e. Samsoe-Peterson, 1983, 1985; Hassan, 1985; Hassan *et al.*, 1983; Basedow *et al.*, 1985) and improving predictions of effects (i.e. Brown, 1988; Jepson *et al.*, 1990; Aldridge & Carter, 1992; Barrett, 1992) are important and active areas of research.

The ultimate aim of implementing reduced doses in pest control is to alter the pest:predator ratio in favour of the predator (van Emden, 1987; Stern *et al.*, 1959). This is achievable by reducing the acute toxic effects of the insecticide on the predators and the indirect results of extreme shifts in the predator:prey ratio leading to starvation effects and the movement of predators out of the crop. However, it has also been noted that problems may arise from leaving a residual pest population, especially if the corresponding build up of aphid specific predators is not synchronised with the pest outbreak. Therefore to avoid the necessity of spraying again there is a need to develop long-term population interactions between the pest and its predators. This will not be achieved solely through the judicious use of insecticides but will take into account cultural practices that provide refugia for the natural enemies during the non-crop period. The implementation of reduced rate insecticides will depended on both ecological and economic considerations. Increased concern for the quality of the environment and the maintenance of pest-predator interactions are likely to dictate the future use of insecticides. As the contribution made by insecticides to a farmers variable costs increases, reducing these costs by spraying less of the costly active ingredient may be one option open to the farmer. Potts and Vickerman (1974) showed that cereal fields were not the sterile environment they were once thought to be. Therefore if the crop can tolerate a limited density of pests and has a high potential for pest regulation, the specific and selective use of insecticides could maximise control by natural enemies, reduce variable costs and create a safer environment for both the farmer and the general public.

CHAPTER 3

THE INFLUENCE OF POST-TREATMENT TEMPERATURE ON THE TOPICAL TOXICITY OF INSECTICIDES TO CEREAL APHIDS.

INTRODUCTION

The previous chapter investigated the extrapolation of laboratory data to control summer aphid pests of cereals using reduced rate insecticide applications. The rates applied were selected from a laboratory 'model' that produced a 'predicted mortality' or 'percentage of aphid control' This 'predicted control' was then tested by generating 'observed percentage reductions' from aphid counts after spraying. Beyond the laboratory however, it is well known that a variety of factors influence the toxicity of insecticides to insects (Brown, 1951; Sheperd, 1958; Busvine, 1971; Croft, 1990; Hassell, 1990). One of these factors, especially for the pyrethroids, is temperature (Sparks *et al.,* 1982; Schouest & Miller, 1988; Toth & Sparks, 1990). This chapter removes many of the variables that affect insecticide toxicity in the field, i.e. concentration of dose impinging on the target, light variability and humidity, to investigate the intrinsic toxicity of a topically applied insecticide at discrete post-treatment temperatures. The following chapter then investigates the effect that post-treatment temperature has on the residual toxicity of the insecticide in the laboratory, and extends the investigation by using foliage treated in the field, and therefore exposed to many of the variables controlled in the laboratory, to investigate the effect that post-treatment temperature may have on insecticide efficacy to a target species

In general, toxicity can be affected by temperature in two ways; toxicity may increase with increasing temperature, and so display a positive temperature coefficient. Toxicity can also decrease with increasing temperature, thus giving a negative temperature co-efficient (Sparks *et al.,* 1982; Schouest & Miller, 1988; Toth & Sparks,

Table 3.1. Comparison of studies from the literature investigating the temperature co-efficients of insecticides from various classes to different invertebrate species.

Author(s) (year)	Invertebrate spp.	Temperature range (°C)	Insecticide class	Temperature co-efficient
Hirano (1979)	*Spodoptera litura, Laodelphax striatella, Plutella xylostella*	15-30	pyrethroids, pyrethrins, organochlorines	negative
Everson & Tonks (1981)	*Phytoseiulus persimilis, Tetramychus urticae*	15-30	pyrethroids	positive *
Riskallah (1984)	*Spodoptera littoralis*	20-35	organophosphates pyrethroids	positive negative
Sadd, Elewa & Beheedy (1983)	*Agrotis ipsilon*	15-30	pyrethroids	negative
Rahman & Yadar (1989)	*Callosobruchus maculatus, C. chinensi*	20-35	pyrethroids	negative
Sparks *et al.* (1983)	*Heliothis virescens*	15.6-37.8	pyrethroids	negative
Thaung & Collins (1986)	*Sitophilus oryzae*	22-30	organophosphates pyrethroids	positive negative
Turnbull & Harris (1986)	*Anthomyza gracilus*	15-32	organphosphates pyrethroids	positive negative
Hinks (1985)	*Melanophus sanguinipes*	15.6-37.8	pyrethroids	negative
Burgess & Hinks (1985)	*Phyllotreta cruciferae*	10-32	pyrethroids	positive *

From a comparison of these studies the pyrethroids show a negative temperature co-efficient against insect species in seven of the studies and a positive temperature co-efficient in one, in the other studies* positive temperature co-efficients were seen for mite species rather than insect species. Organophosphate insecticides showed a positive temperature co-efficient in all of the studies.

1990). The activity of organophosphorous compounds is usually positively correlated with temperature (Hsieh *et al.*, 1982; Grafius, 1986), while that of DDT (an organochlorine) and natural pyrethrums is negatively correlated (Gutherie, 1950). The situation is not so clear with carbamate and synthetic pyrethroid insecticides where activity-temperature coefficients ranging from negative to positive have been reported (Harris & Kinoshita, 1977; Sparks *et al.*, 1983; Kumar & Chapman, 1984; Grafius, 1986; Sogorb *et al.*, 1988; Rahman & Yadar, 1989).

Chemical structure appears to influence the temperature coefficient of the compound. Pyrethroids that lack an α-cyano group often have rather large, negative temperature coefficients as opposed to slightly negative or positive coefficients for those with the grouping (Sparks *et al.*, 1982; Sparks *et al.*, 1983; Miller & Salgado, 1985; Wadleigh *et al.*, 1991). From a survey of the literature describing the influence of temperature on synthetic pyrethroid activity, it is concluded that activity is generally negatively correlated with temperature (Table 3.1.). Although temperature appears to be negatively correlated with temperature for the pyrethroids, the insect species tested also affects the temperature-toxicity relationship. The majority of species show a negative temperature relationship but Sparks *et al.* (1982) observed positive temperature coefficients for pyrethroids bioassayed on the tobacco budworm *Heliothis virescens* (F.) (Lepidoptera: Noctuidae).

In order to predict the nature and extent of environmental temperature influence on the general biological activity of insecticides, experiments were carried out on the effect of post-treatment temperature on the toxicity of λ-cyhalothrin (ICI, Karate, 5% EC w/v) and dimethoate (BASF, Perfecthion, 40% EC w/v) against the cereal aphids *Sitobion avenae* and *Rhopalosiphum padi* (Homoptera: Aphididae). The aims of the experiment were i) to elucidate the temperature coefficient shown by topical application of these insecticides against the aphids over a range of post-treatment temperatures, ii) quantify any differences in susceptibility between the aphid species and between the insecticides and iii) examine the consequences of temperature variation for the efficacy of reduced rate insecticides in a field situation.

MATERIALS AND METHODS

S. avenae and *R. padi* used in the experiments were taken from a laboratory culture maintained on winter wheat (var. Knirps) in an insectary (20°C / RH 70-80% / 16:8 L:D). The cultures were reared inside ventilated perspex cages rearing cages (70 x 90 x 60 cm). Wheat was shown on a weekly basis in 12 cm pots (approx. 30 seeds per pot) containing John Innes No. 2 compost. Plants in the culture were changed weekly, leaving one infested plant in the culture, to prevent population build-ups. Aphids from single instars (i.e. 4^{th} instar) were used in the bioassays to minimise differences in susceptibility that could be attributed to changes in anatomy, physiology, size and behaviour of individual aphid instars (Busvine, 1971). Aphids used in the experiment were collected by gently shaking an infested wheat plant over a white photographic tray and removing each aphid individually with a fine paint brush. Post-treatment assessments were made by opening the clip cage and examining the aphids using a binocular microscope. Care was taken not to allow any of the aphids to escape. Assessment of treatment effects on the aphids was based on the categories described in Chapter 2.

On the day of the test a stock solution of the insecticide was made up and serial dilution's made to the required doses. Initial range-finder tests were carried out to select the three dose rates used and controls were treated with the diluent only. The diluent was a 0.01% (v/v) solution of the wetting agent Lutenosol (BASF) in distilled water. The Lutenosol was used to assist droplet adhesion onto the test aphids. The test solution was applied from a 50 ul gas-tight syringe (S.G.E., Australia) fitted with a teflon-coated needle, using a hand-held microapplicator (Burkhard Manufacturing Co. Ltd) (Arnold, 1967). Each aphid was treated with an 11.5 nl droplet under a binocular microscope. The doses were applied to the thorax of an aphid held on the bristles of a fine paint brush in sequential order beginning with the control treatment followed by the lowest dose and so on. 30 aphids per treatment were dosed at room temperature and then placed in clip cages (10 aphids per cage) and attached to wheat seedlings (cv Knirps) at the 2 leaf stage.

A clip cage consisted of a 3 cm clear plastic Petri dish where the lid was inverted to form the base and the base was inverted and used as the lid of the clip cage. Four ventilation holes covered by fine netting were drilled into the lid. Parafilm was placed around the edges of the base and lid to seal the clip cage and also protect the wheat leaf against damage when the clip cage was attached to the leaf. The clip cage was kept together using a hairdressers clip and the whole cage containing the leaf was supported by a wooden barbecue skewer (12 cm in length) pushed into the soil around the leaf.

Within five minutes of treatment, the aphids were placed in controlled environment chambers set at post-treatment temperatures that span the range of temperatures found in the cereal fields of northern Europe (either 10, 15 or 20°C ± 0.5°C / RH 70-80% / 16:8 L:D). Mortality and knockdown assessments were made 48, 72, 96 and 120 h after treatment with the insecticides.

Probit analysis was carried out on the 72 h mortality data to obtain dose-response statistics (Finney, 1971). Abbott's formula was used to correct the data for control mortality (Abbott, 1925). Only dead aphids were included in the calculations, but after 72 h few individuals remained knocked down. Statistical analysis was carried using the SPSS statistical package (SPSS Corporation, version 6.0))

RESULTS

The 24, 48, 72, 96 and 120 hour LD_{50} values given by probit analysis were plotted against time for both aphid species at the different temperatures. There was a similar trend of decline in LD_{50} values over time at all temperatures for both species when treated with λ-cyhalothrin (Figs. 3.1. & 3.2.). A similar but more variable trend was seen for dimethoate treated individuals (Figs. 3.3. & 3.4.). The 24 h LD_{50} response could not be calculated for dimethoate treated *S. avenae* as there was not sufficient aphid mortality. In all cases the LD_{50} values approached a stable endpoint, although mortality may have accumulated over a longer period. The 72 h assessment time was chosen for comparisons of susceptibilities.

The summary statistics from probit analysis of 72 h mortality data are given in Tables 3.2. and 3.3. for λ-cyhalothrin and dimethoate respectively. The LD_{50} values increased with increasing temperature for λ-cyhalothrin-treated aphids in both species. This indicated that λ-cyhalothrin had a negative temperature coefficient against these aphid species. The range of LD_{50} values was 0.036, 0.049 and 0.084 ng a.i. / insect for *R. padi* and 0.006, 0.02 and 0.04 ng a.i. / insect for *S. avenae* at 10, 15 and 20°C respectively.

The LD_{50} values decreased with increasing temperature for dimethoate treated individuals in both aphid species. This indicated that dimethoate had a positive temperature coefficient against these aphid species. The range of LD_{50} values was 1.51, 1.26 and 0.39 ng a.i. / insect *R. padi* and 0.61, 0.47 and 0.33 ng a.i. / insect for *S. avenae* at 10, 15 and 20°C respectively.

To see if there were differences in the toxicity of the two compounds at the different post-treatment temperatures, ratios (Relative Median Potency, RMP) were calculated that gave an estimate of the relative toxicity of the compounds at the LD_{50} level for each aphid species at the two temperatures that were compared (Tables 3.4. & 3.5.). If the confidence interval spans the value of 1 then there was reason to suspect that the insecticide was equally toxic at the temperatures compared. The ratios show that λ-cyhalothrin was equally toxic at all the temperatures compared for *R. padi* (Table 3.4.). For *S. avenae*, λ-cyhalothrin was significantly more toxic at 10°C when compared 15 and 20°C (Table 3.4.). λ-cyhalothrin was equally as toxic at 15 and 20°C. RMP values for *S. avenae* and *R. padi* treated with dimethoate showed it was more toxic at 20 than 10°C for both aphid species at the LD_{50} level (Table 3.5.). There were no differences in toxicity over the other temperature ranges at the LD_{50} level.

Susceptibility differences between the aphids were compared by iterating a sequence of lethal dose ratios calculated from dose-response statistics for each aphid at a particular temperature and calculating a mean ratio. The sequence of doses selected represented responses between LD_{10} and LD_{90}. The mean values obtained were termed

Table 3.2. 72 h probit statistics of response to topical application of λ-cyhalothrin of *S. avenae* and *R. padi* at the different temperatures.

Aphid species	Temperature (°C)	Probit equation	LD_{50} (95% CI) (detransformed) ng a.i. / insect	χ^2 (d.f.) Significance[a]
R. padi	10	y = 1.11 + 0.77 x	0.036 (0.01-0.11)	ns (5)
	15	y = 1.01 + 0.77 x	0.049 (0.02-0.17)	ns (5)
	20	y = 0.83 + 0.77 x	0.084 (0.03-0.36)	ns (5)
S. avenae	10	y = 0.88 + 0.46 x	0.006 (0.003-0.05)	ns (5)
	15	y = 0.47 + 0.46 x	0.02 (0.003-0.17)	ns (5)
	20	y = 0.11 + 0.46 x	0.04 (0.02-0.11)	ns (5)

[a] Significance level: ns, not significant; *, $p < 0.05$

(*R. padi* parallelism test Chi square = 2.30; df=2; p>0.05)
(*S. avenae* parallelism test Chi square = 2.98; df=2; p>0.05)

Table 3.3. 72 h probit statistics of response to topical application of dimethoate of *S. avenae* and *R. padi* at the different temperatures.

Aphid species	Temperature (°C)	Probit equation	LD_{50} (95% CI) (detransformed) ng a.i. / insect	χ^2 (d.f.) Significance[a]
R. padi	10	y = -0.27 + 2.51 x	1.51 (0.10-2.57)	ns (5)
	15	y = 0.19 + 2.51 x	1.26 (0.75-2.43)	ns (5)
	20	y = 0.99 + 2.51 x	0.39 (0.28-0.56)	ns (5)
S. avenae	10	y = 0.88 + 0.46 x	0.61 (0.41-0.95)	ns (5)
	15	y = 0.47 + 0.46 x	0.47 (0.23-1.02)	ns (5)
	20	y = 0.11 + 0.46 x	0.33 (0.22-0.49)	ns (5)

[a] Significance level: ns, not significant; *, $p < 0.05$

(*R. padi* parallelism test Chi square = 0.984; df=2; p>0.05)
(*S. avenae* parallelism test Chi square = 0.592; df=2; p>0.05)

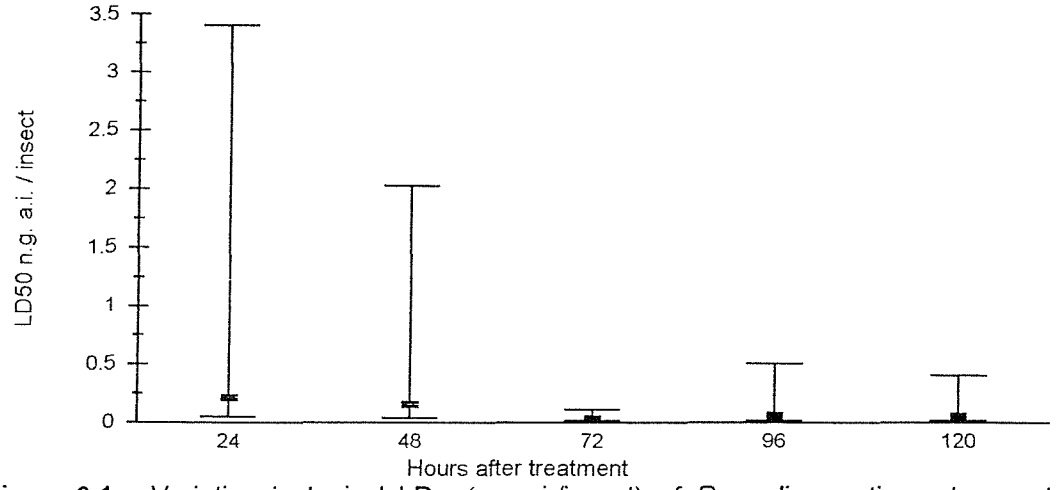


Figure 3.1a. Variation in topical LD_{50} (ng a.i./insect) of *R. padi* over time at a post-treatment temperature of 10°C after treatment with λ-cyhalothrin. Error bars indicate 95% fiducial limits.

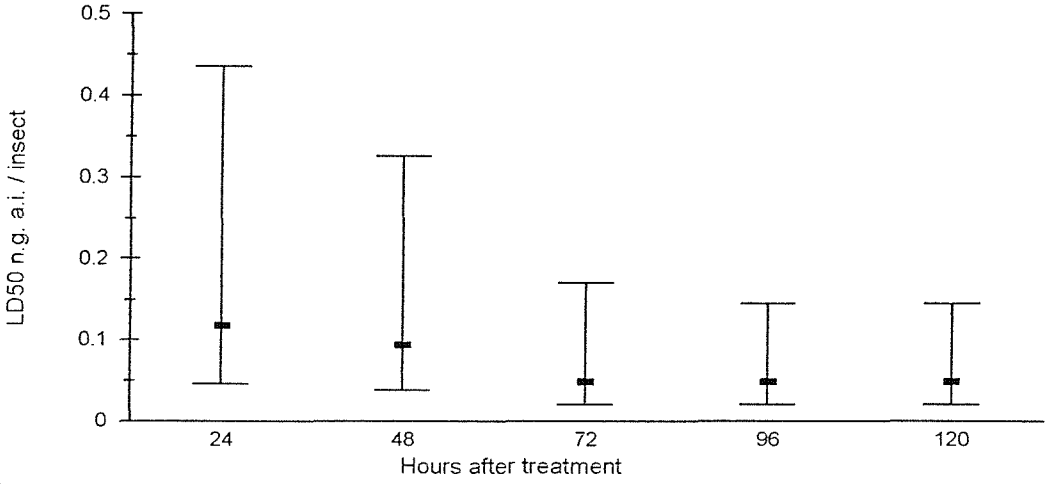


Figure 3.1b. Variation in topical LD_{50} (ng a.i./insect) of *R. padi* over time at a post-treatment temperature of 15°C after treatment with λ-cyhalothrin. Error bars indicate 95% fiducial limits.

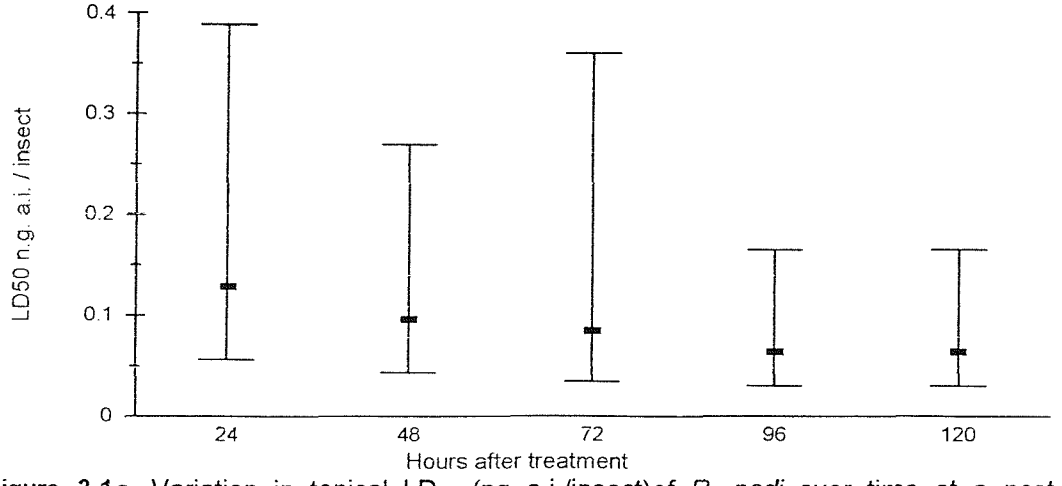


Figure 3.1c. Variation in topical LD_{50} (ng a.i./insect)of *R. padi* over time at a post-treatment temperature of 20°C after treatment with λ-cyhalothrin. Errors bars indicate 95% fiducial limits.

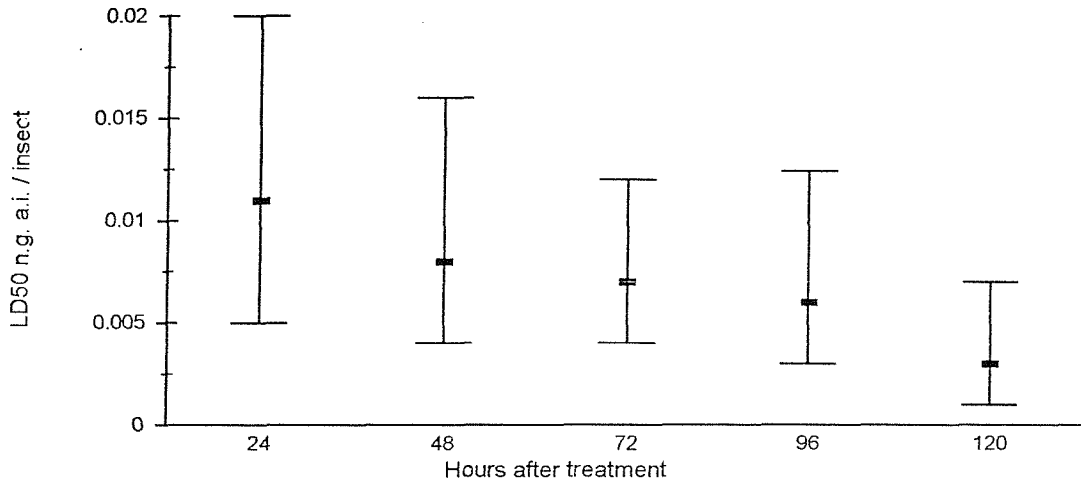


Figure 3.2a. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* over time at a post-treatment temperature of 10°C after treatment with λ-cyhalothrin. Error bars indicate 95% fiducial limits.

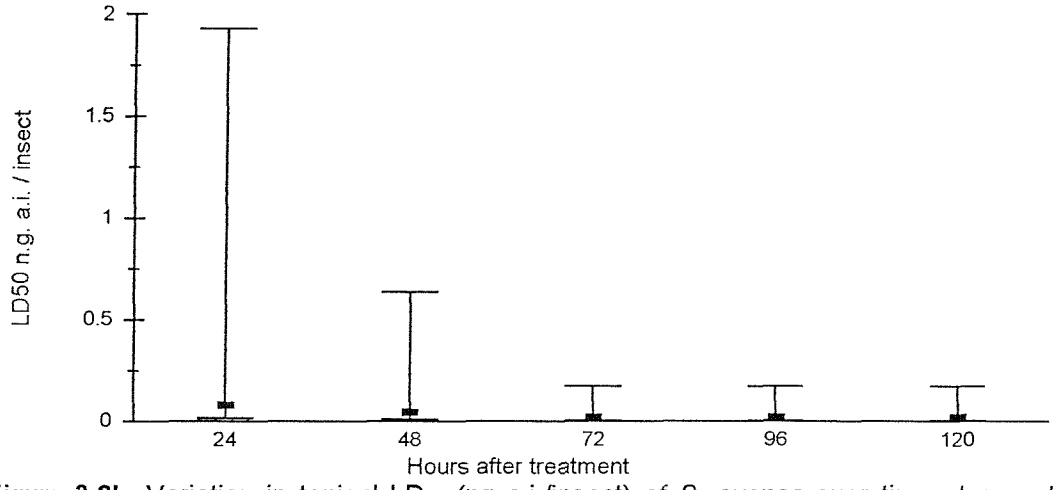


Figure 3.2b. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* over time at a post-treatment temperature of 15°C after treatment with λ-cyhalothrin. Error bars indicate 95% fiducial limits.

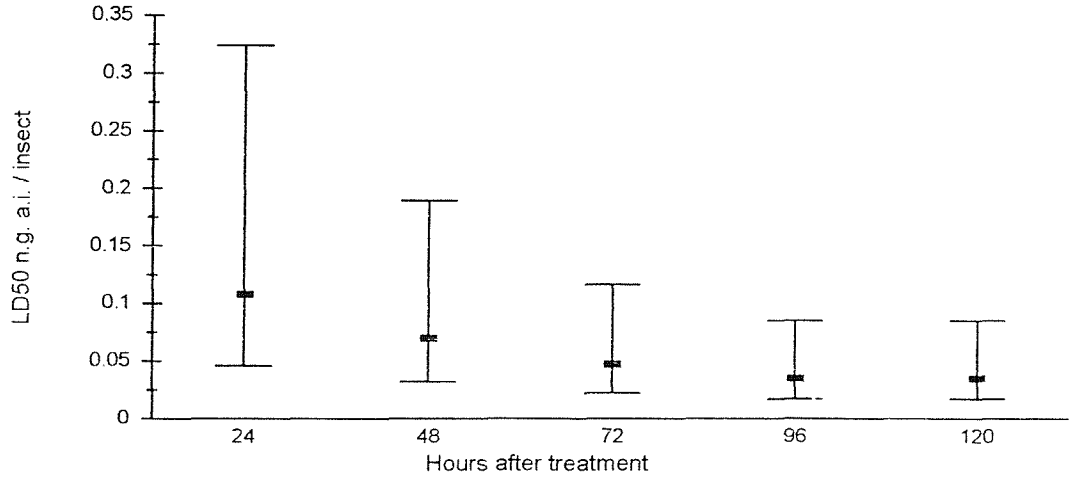


Figure 3.2c. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* over time at a post-treatment temperature of 20°C after treatment with λ-cyhalothrin. Error bars indicate 95% fiducial limits.

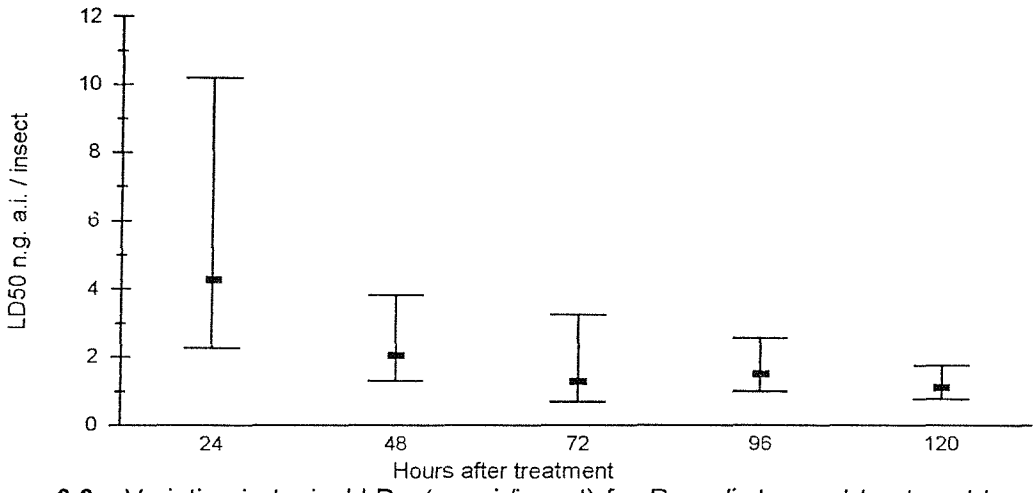


Figure 3.3a. Variation in topical LD_{50} (ng a.i./insect) for *R. padi* at a post-treatment temperature of 10°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

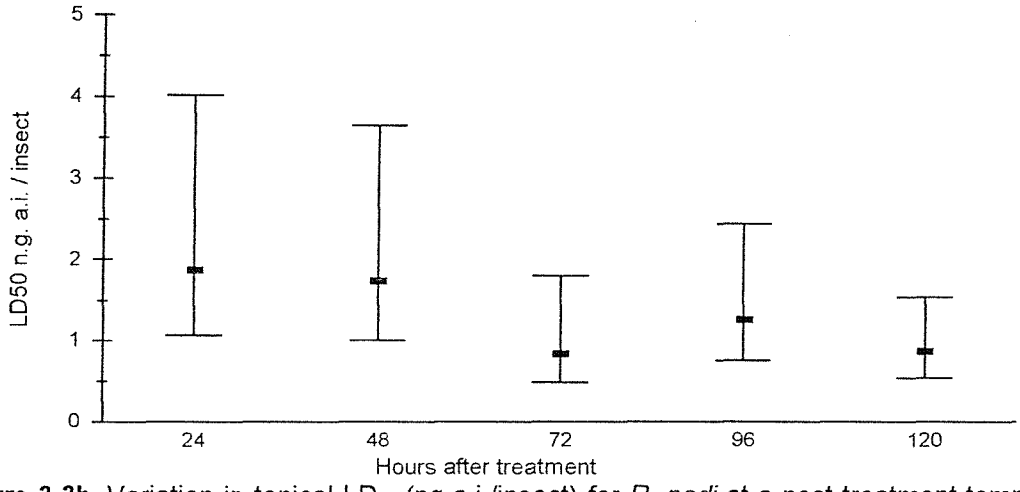


Figure 3.3b. Variation in topical LD_{50} (ng a.i./insect) for *R. padi* at a post-treatment temperature of 15°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

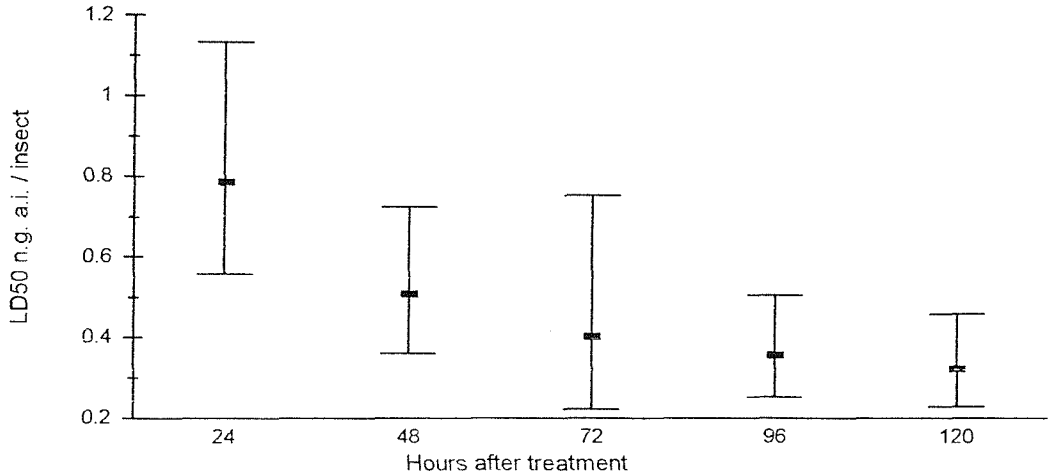


Figure 3.3c. Variation in topical LD_{50} (ng a.i./insect) for *R. padi* at a post-treatment temperature of 20°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

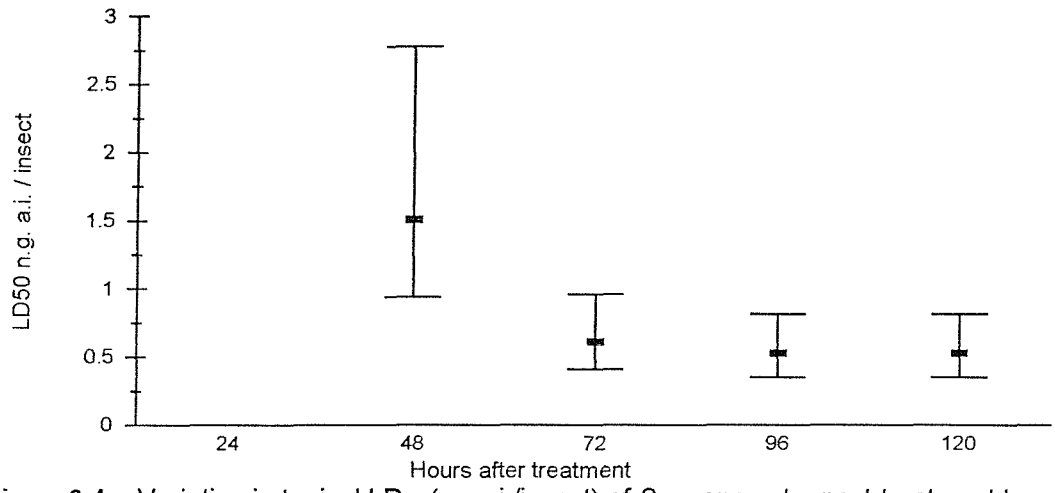


Figure 3.4a. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* at a post-treatment temperature of 10°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

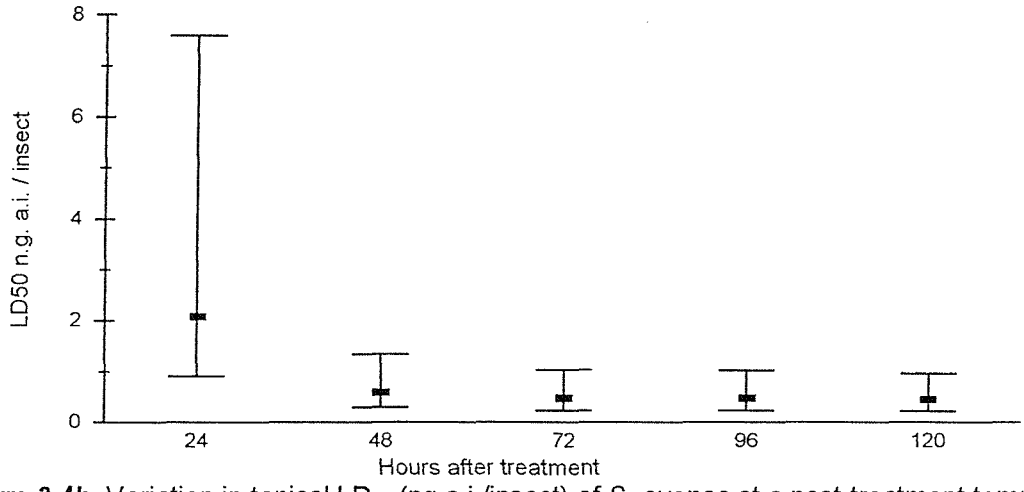


Figure 3.4b. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* at a post-treatment temperature of 15°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

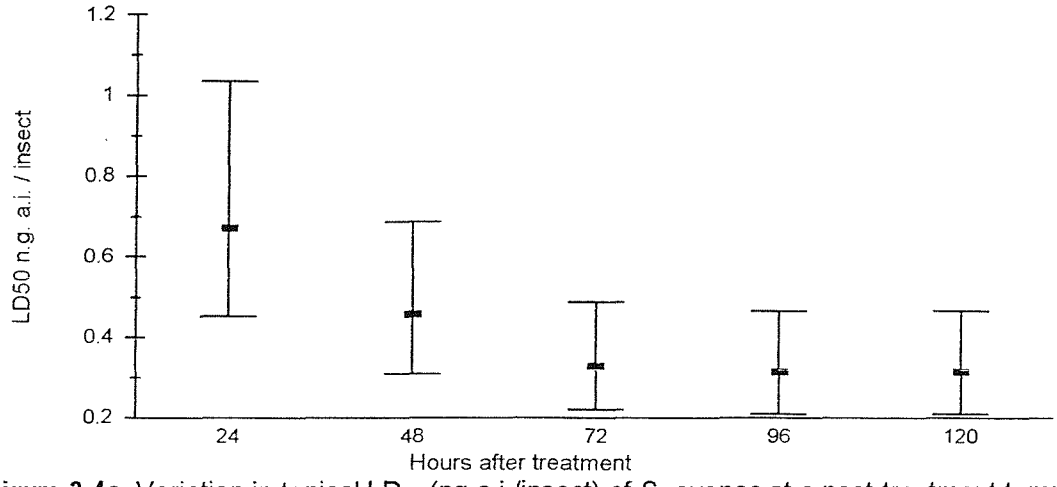


Figure 3.4c. Variation in topical LD_{50} (ng a.i./insect) of *S. avenae* at a post-treatment temperature of 20°C after treatment with dimethoate. Error bars indicate 95% fiducial limits.

"susceptibility indices". These ratios gave an estimate of the relative differences in toxicity of the compounds at each temperature for the two aphid species. If the ratio was greater than 1 then the insecticide was more toxic to *S. avenae* than *R. padi* at the temperature compared. For both λ-cyhalothrin and dimethoate *S. avenae* was more susceptible than *R. padi* (Table 3.6.), with the index decreasing as the temperature increased.

Table 3.4. Relative median potency (RMP) comparing the relative toxicity of λ-cyhalothrin to each individual aphid species over the different temperature ranges.

Aphid species	Temperature ratio range (°C)	RMP (95% CI) (72 h)
R. padi	10:15	0.74 (0.20-2.48)
	10:20	0.43 (0.10-1.42)
	15:20	0.58 (0.14-1.97)
S. avenae	10:15	0.13 (0.01-0.90)
	10:20	0.02 (0.00-0.19)
	15:20	0.17 (0.13-1.16)

RMP calculations obtained from the LD_{50} values at each temperature after 72 h.

DISCUSSION

When applied topically λ-cyhalothrin showed a negative temperature coefficient from comparison of 72 h LD_{50} to both *S. avenae* and *R. padi*. Statistical comparison by RMP showed that the negative temperature coefficient was only significant for *S. avenae* however (Table 3.4.). Negative temperature coefficients based on comparison of LD_{50} values at different temperatures have also been shown in other studies (Harris & Kinoshita, 1977; Harris *et al.*, 1978; Turnbull & Harris, 1986; Toth & Sparks, 1990). Comparison of LD_{50} values for the topical toxicity of dimethoate showed that it was positively correlated with temperature, although it was only shown to be significant for *S. avenae* however (Table 3.5.). This positive coefficient for the organophosphorous compounds has also been shown in other studies with insects (Meisch *et al.*, 1972; Tyler & Binns, 1982; Turnbull & Harris, 1986; Macleod, 1988; Anon, 1990).

Table 3.5. Relative median potency (RMP) comparing the relative toxicity of dimethoate to each individual aphid species over the different temperature ranges.

Aphid species	Temperature ratio range (°C)	RMP (95% CI) (72 h)
R. padi	10:15	1.54 (0.66-4.96)
	10:20	3.20 (1.27-19.47)
	15:20	2.08 (0.94-7.98)
S. avenae	10:15	1.34 (0.74-2.62)
	10:20	1.92 (1.05-4.08)
	15:20	1.43 (0.78-2.82)

RMP calculations obtained from the LD_{50} values at each temperature after 72 h.

Table 3.6. Susceptibility indices comparing the relative toxicities of λ-cyhalothrin and dimethoate over the different temperature ranges.

Compound	Temperature (°C)	Susceptibility ratio (*R. padi*:*S. avenae*) (± 95% CI) (72 h)
λ-cyhalothrin	10	4.46 (± 1.70)
	15	3.02 (± 0.80)
	20	1.56 (± 0.59)
dimethoate	10	2.34 (± 0.41)
	15	1.76 (± 0.21)
	20	1.26 (± 0.54)

Susceptibility ratios were obtained by calculating a mean ratio from the LD_{10}, LD_{30}, LD_{50}, LD_{70} and LD_{90} doses for each respective pair of temperatures after 72 h.

Differences in toxicity between species were also observed, since *S. avenae* was more susceptible than *R. padi* to both λ-cyhalothrin and dimethoate (Table 3.6.). The aphids also differed in their RMP for λ-cyhalothrin. Differences in susceptibility may be due to differential rates of penetration through the insect cuticle or rates of metabolism within the insect (Croft, 1990). These differences will be important when predicting the effect a compound will have at different temperatures, when applied against a broad range of pests each differing in their susceptibility to the compound (Sparks *et al.*, 1982;

Sparks *et al.*, 1983; Toth & Sparks, 1990). In the summer the same insecticide application would be expected to control both *S. avenae* and *R. padi* in cereals, therefore differences in susceptibility to an insecticide have to be considered so that a sufficient dose can be applied to achieve control of both aphid species.

The reasons why the two insecticides should show different temperature coefficients are not clear. The physiological events of insecticide poisoning i.e. intense convulsions leading to lethal paralysis are positively associated with increasing temperature (Miller & Salgado, 1985; Hassell, 1990), it can therefore be seen how an insecticide could have a positive temperature coefficient. Diuresis also increases with increasing temperature (Wigglesworth, 1972) and has been shown to increase after treatment with an insecticide Ingram, 1955; Cassida & Maddrell, 1971; Gerolt, 1976), therefore increased water loss may also help to explain a positive temperature relationship. There are, however, some difficulties in relating these physiological observations to the lethal effect of pyrethroids on intact insects. Air humidity, a variable that could not be measured and can increase with increasing temperature, has been known to reduce the toxic effects of cypermethrin on *Demetrius atricapolis* (L.) (Coleoptera: Carabidae) Jepson *et al.*, 1987) and deltamethrin on *Oedothorax apicatus* (Blackwall) (Aranae: Linyphiidae) (Everts *et al.*, 1991b). For the pyrethroids as the temperature decreases there maybe a change in binding to their receptor site in the insect nervous system (Devonshire pers. comm.). The pyrethroids reversibly bind to the sodium channel in the nerve axon and as temperature decreases the amount of time bound to the receptor site increases. This might increase the firing of the nerve axon with similar consequences to those described above and may eventually lead to increased paralysis and death.

In the laboratory ambient temperature can easily be controlled however, temperatures in the cereal fields of northern Europe fluctuate throughout the day and also the year, especially between autumn and summer insecticide applications. If the results of laboratory experiments could be directly extrapolated to a field situation, the negative temperature coefficient of the pyrethroids suggests that their intrinsic toxicity at lower temperatures (Table 3.3) would allow reductions in dose-rates during autumn

applications, when the ambient temperature would be similar to the lowest temperature used in these experiments. Temperatures above 30°C have been implicated in control failures in cotton when using pyrethroids (Hassell, 1990). Control failure was considered to be due to increases in the post-treatment ambient temperature decreasing the toxicity of the insecticide. Although the actual reasons for this were not known, it may have been due to increased metabolism of the insecticide by the target pest or loss of the pyrethroid from the foliage by evaporation or degradation. Significant differences in the toxicity of dimethoate between 10 and 20°C (Table 3.4.) suggest that dose-rate reductions between winter and summer aphicide applications could be considered for this compound, especially if there was no difference in aphid control and beneficial predators were conserved. Bioassay data is, however, difficult to extrapolate to a field situation, as the bioassay does not consider the vagaries of a field situation. Therefore the potential for reducing rates of summer and autumn applied insecticides would need to be investigated by rigorous field trials possibly using the framework described in Chapter 2.

In order to avoid undesirable effects such as pest resistance, pest resurgence and outbreaks of secondary pests, insecticides are best applied within an integrated control framework (Dent, 1991). Within this framework effects on beneficial predators should be considered. Heimbach & Baloch (1994) showed that pyrethroids displayed negative temperature coefficients against *Pterostichus* (=Poecilus) *cupreus* L (Coleoptera: Carabidae). After treatment many of the beetles recovered, with recovery time being dependent on the post-treatment temperature. Effects of temperature similar to those observed in the laboratory have also been observed in the field (Heimbach & Baloch, 1994). Carabid beetles (*P. cupreus*) were kept in enclosures in cereal fields and λ-cyhalothrin applied in autumn at low temperatures. Mortality of the beetles was due to them being unable to burrow to evade the applied insecticide, consequently fewer beetles were found the following summer which would have serious consequences for beneficial populations on a field scale. This suggests that although the efficacy of an insecticide to a pest may be increased at lower temperatures, effects would also be observed on beneficial predators. The use of reduced-rate insecticide applications would minimise insecticide effects on beneficials (Poehling, 1985, 87 & 89).

The relationship between temperature and toxicity of an insecticide may have potential application in pest management programmes. If post-treatment temperature is considered, dimethoate could be used as a summer aphicide, as temperatures of around 20°C would increase the intrinsic toxicity of this compound and allow dosage reductions compared to a full rate application to still achieve the same level of pest control. However as a general recommendation pyrethroids would appear more suitable for use as an aphicide for both summer and autumn applications. They have been observed to be less detrimental to the environment through lower application rates and reduced effects on beneficials (Vickerman *et al.*, 1987a&b). This coupled with equal toxicity of all temperature ranges likely to be observed in the cereal fields of northern Europe would suggest that they are more suitable aphicides, when there could be wide fluctuations in the ambient temperature. The efficacy of reduced-rate applications compared to full rate applications previously shown may remove doubt over any control failures if the post-treatment temperature fluctuated over the temperature range used in these experiments.

CHAPTER 4

THE INFLUENCE OF POST-TREATMENT TEMPERATURE ON THE RESIDUAL TOXICITY OF LABORATORY AND FIELD APPLIED INSECTICIDES TO CEREAL APHIDS.

INTRODUCTION

The previous chapter investigated the effect of post-treatment temperature on the topical toxicity of λ-cyhalothrin and dimethoate, and showed in general, that λ-cyhalothrin toxicity was negatively, and dimethoate positively, correlated with temperature. However, for pests such as aphids other exposure routes and environmental variables might be important in determining the efficacy of these compounds at different temperatures. In order to begin disentangling the complex matrix of factors controlling efficacy in the field, a bioassay regime for assessing the post-treatment toxicity of field plants was developed. This regime controlled temperature and exposure route but employed plants sprayed in the field for its assessment.

Apart from direct contact with an insecticide cereal aphids may be exposed to residual deposits on the leaf surface, fumigant vapours and systemic action within the plant. Studies have shown that temperature-toxicity relationships of insecticides can be affected by the choice of test species and route of exposure to the insecticide (Harris & Kinoshita, 1977; DeVries & Georghiou, 1979; Sparks *et al.*, 1982; Ewen *et al.*, 1984, Scott & Georghiou, 1984; Subramanyam & Cutkromp, 1987). The topical application of permethrin (a synthetic pyrethroid) to *Haematobia irritans* (L.) (Diptera: Muscidae) gave a negative temperature coefficient between pairs of successively higher temperatures at 21, 27 and 32°C. Exposure to deposits on cloth (simulating control in the field) gave a positive temperature coefficient between 21 and 27°C and no significant difference in toxicity between 27 and 32°C (Schmidt & Robertson, 1986).

In this chapter the aims were to firstly explore the effect of post-treatment temperature on the toxicity of residual deposits of λ-cyhalothrin and dimethoate applied in the laboratory and under field conditions. Secondly to investigate the susceptibility of the two aphid species tested to the insecticides through this route and thirdly consider the potential for reducing dose-rates of the insecticides through the interaction between toxicity, exposure route and temperature.

MATERIALS AND METHODS

S. avenae and *R. padi* used in the experiments were taken from a laboratory culture on maintained on winter wheat (cv. Knirps) in an insectary (20 ± 2°C, 55-70% RH, 16:8 L:D). The cultures were reared inside ventilated perspex cages (70 x 90 x 60 cm). Wheat was sown on a weekly basis in 12 cm diameter pots (approx. 30 seeds per pot) containing John Innes No. 2 compost. Plants in the culture were changed weekly, leaving one infested plant in the cage, to prevent excessive population build-up. Aphids from single instars (i.e. 4^{th} instar) were used in the bioassays to minimise differences in susceptibility that could be attributed to changes in anatomy, physiology, size and behaviour of individual aphid instars (Busvine, 1971). Aphids used in the experiment were collected by gently shaking an infested wheat plant over a white photographic tray and removing each aphid individually with a fine paintbrush. Post-treatment assessments were made by opening the clip cage and examining the aphids using a binocular microscope. Care was taken not to allow any of the aphids to escape. Assessment of treatment effects on the aphids was based on the categories described in Chapter 2. The insecticides used in these experiments were λ-cyhalothrin (ICI, Karate, 5% EC w/v) and dimethoate (BASF, Perfecthion, 40% EC w/v). A 0.001% (v/v) solution of the wetting agent Lutenosol (BASF) in distilled water was the diluent.

i) Residual toxicity of laboratory applied doses;

On the day of the test, a stock solution of the insecticide was made up and serial

dilutions made to the required doses. Initial range finder-tests were carried out to select the doses for use. For each compound there were 3 dose-rates with 2 replicates per treatment. 1.5 ml of a solution of λ-cyhalothrin or dimethoate was pipetted into a glass Petri dish (63.6 cm^2) to cover the bottom of the dish as evenly as possible and left to dry for approximately 2 h. PTFE (Poly(tetrafluroethylene)(Fluon, ICI)) was then painted around the inside of the dish to prevent aphids escaping and 15 aphids introduced into each of the dishes using a fine paintbrush. After each aphid had been placed in the Petri dish the brush was rinsed in distilled water to avoid cross contamination. The Petri dishes, without their lids, were placed inside a fume cupboard at 20 ± 1°C to remove any vapour effects and the aphids left exposed to the insecticide residues for 1 h. They were then transferred to clip-cages on wheat seedlings (var. Knirps) and placed in environmentally controlled chambers of different post-treatment temperatures (10, 15 or 20°C ± 0.5°C / 70-80% R.H. / 16:8 L:D). Assessments were made 24, 48, 72 and 96 h after exposure to the compounds.

ii) Residual toxicity of field deposits of λ-cyhalothrin in the laboratory

The laboratory experiments used a glass Petri dish as the substrate for the aphids. Glass is an inert and unrealistic substrate and it was considered necessary to modify the test in order to predict what effect post-treatment temperature might have on aphids in the field. The toxicity of an insecticide can be strongly affected by changes in biotic and abiotic factors. The bioavailability of the insecticide on a plant surface will be affected by factors such as distribution on the leaf after spraying and thickness and architecture of the wax layer of a leaf surface. A decision was made to spray plants in the field and then bring them back to the laboratory in order to expose the aphids to post-treatment temperatures that could not be achieved in the field.

A field of winter wheat (cv. Galahad, 450 tillers m^2) was sprayed at GS 69 (Zadoks *et al.*, 1974) with λ-cyhalothrin (10 g a.i./ha), using a tractor-mounted hydraulic spray fitted with an 18 m spray boom and 24 110° flat fan (F110/1.71/3) nozzles operating at 3 bar. The application rate of 200 l/ha was achieved at a tractor speed of 10 km per hour. Treated leaves were randomly collected from a plot measuring 2 x 10 m previously marked out on

the day of spraying. Untreated leaves were randomly collected from a 2 x 10 m unsprayed area of the field at the same time as treated leaved were collected.

Continuous exposure to field deposits of λ-cyhalothrin;

Three treated and three untreated leaves were collected for each post-treatment regime 24 h after spraying. Ten aphids were clip-caged onto each individual leaf. A total of 30 aphids were used per treatment. After the aphids had been caged onto the leaves, they were placed in environmentally-controlled chambers at either 10, 15 or 20 ± 0.5°C. Twenty four hours later the aphids were assessed and any live aphids transferred, using a fine paintbrush, to freshly-collected treated or untreated leaves and returned to their respective post-treatment temperatures. Fresh leaves were collected every 24 h because they had wilted to much after this period and were unsuitable for further clip-caging of aphids at 20°C. Aphids were therefore caged at to residues that were 24, 48, 72 and 96 h old during the experiment. Assessments and transfer to fresh leaves occurred every 24 h until the aphids had been exposed to deposits continuously for 96 h.

This experiment investigated continuous exposure to the residual deposits, in order to explain the results further, a 'snapshot' of residual toxicity at different time periods within the 96 h period was necessary

Persistence of effects of field residues at different post-treatment temperatures;

Groups of three treated and untreated leaves were collected 24, 48, 72, and 96 h after spraying and ten aphids clip-caged onto each leaf. After the aphids had been clip caged onto the leaves, they were placed in environmentally controlled chambers at either 10, 15 or 20°C ± 0.5°C. A total of 30 aphids per treatment were used for each post-treatment temperature. After exposure to the residues for 24 h the aphids were assessed and alive aphids clip-caged onto untreated wheat leaves and placed in an environmentally-controlled chamber at 20 ± 0.5°C. Any subsequent recovery was assessed 24 h later.

Statistical analysis

Probit analysis was carried out on the field data and 72 h dose-response data for residual toxicity in the laboratory (Finney, 1971). Abbott's formula was used to correct for control mortality (Abbott, 1925). Only dead aphids were included in the 72 h dose-response calculations, but after 72 h few individuals remained knockdown. All other statistical analyses were carried out using the SPSS statistical package (Version 6.0., SPSS corporation).

RESULTS

i) Residual toxicity of laboratory applied doses

The 24, 48, 72 and 96 h LD_{50} values given by probit analysis were plotted against time for both aphid species at the different post-treatment temperatures. In general there was a similar trend of decline in the LD_{50} values over time for all temperatures and both species treated with λ-cyhalothrin (Figures 4.1. & 4.2). A more variable trend was observed for both aphid species treated with dimethoate (Figures 4.3. & 4.4.).

After 1 h of exposure to laboratory deposits of λ-cyhalothrin all aphids exhibited symptoms of knockdown, subsequent recovery was dependent on post-treatment temperature and the dose applied. Recovery began within 24 h after being removed from the insecticide treated surface for both aphid species. The summary statistics from analysis of 72 h dose-response data are given in Tables 4.1 and 4.2 for λ-cyhalothrin and dimethoate respectively. The LD_{50} values increased with increasing temperature for λ-cyhalothrin in both aphid species (Table 4.1.). The LD_{50} values were 0.006, 0.008 and 0.009 μg a.i. / cm^2 for *R. padi* and 0.002, 0.004 and 0.008 μg a.i. / cm^2 for *S. avenae* at 10, 15 and 20°C respectively. There were significant differences between the LD_{50} values at 10 and 20°C for *S. avenae* treated with λ-cyhalothrin (based on non-overlapping confidence

Figure 4.1a. Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

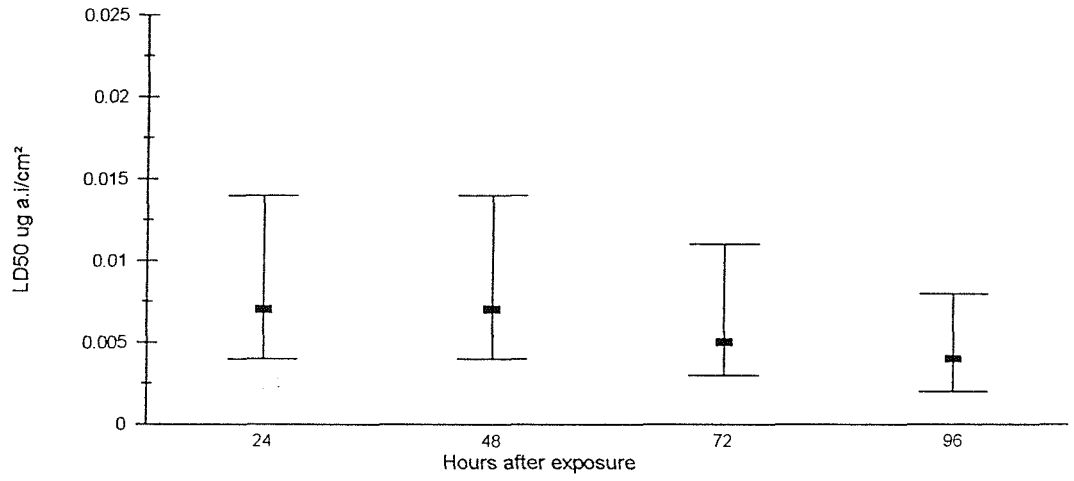


Figure 4.1b Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 15°C. Error bars indicate 95% fiducial limits.

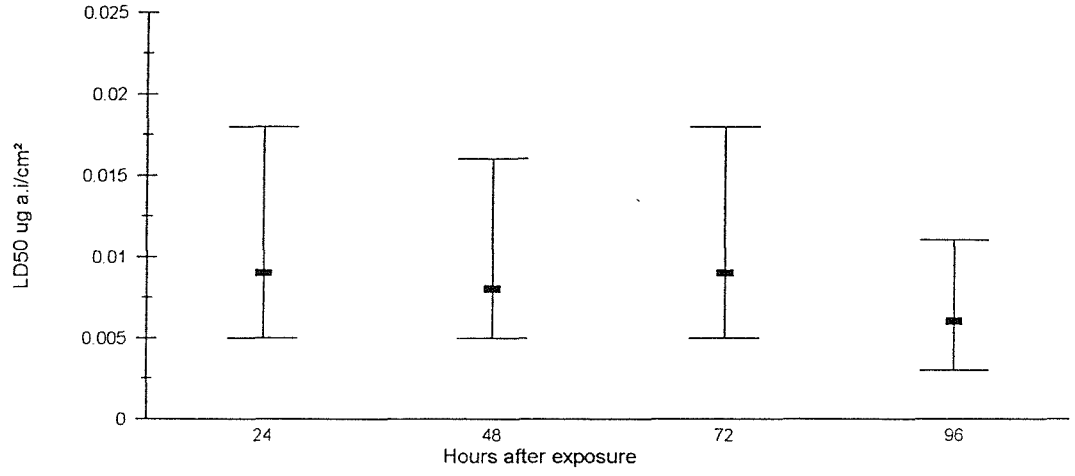


Figure 4.1c. Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 20°C. Error bars indicate 95% fiducial limits.

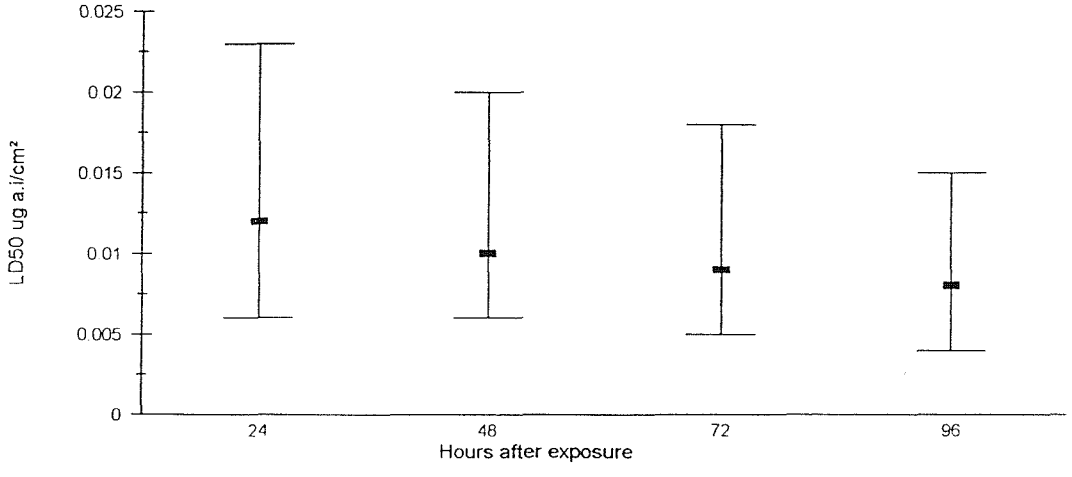


Figure 4.2a. Variation in residual LD_{50} (µg a.i./cm^2) values for *S. avenae* over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

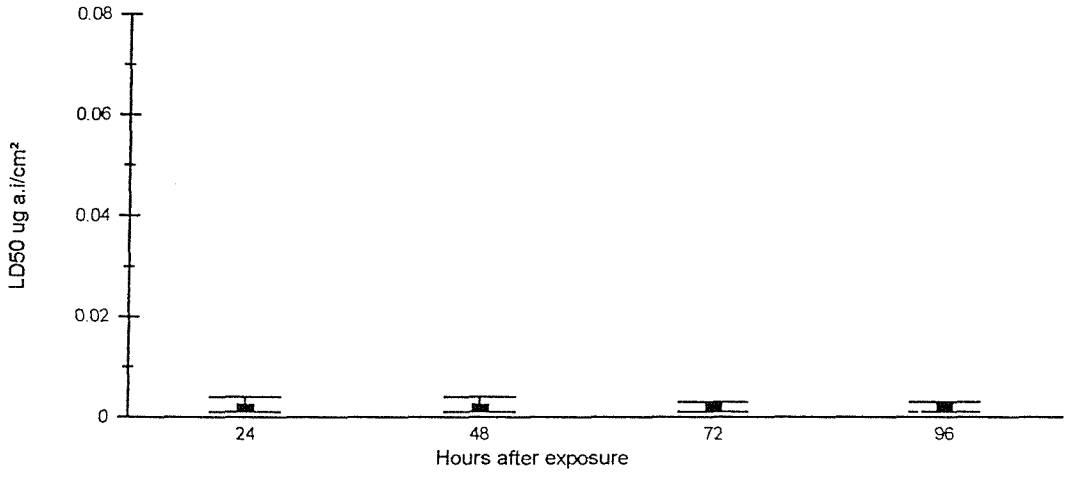


Figure 4.2b Variation in residual LD_{50} (µg a.i./cm^2) values for *S. avenae* over time at a post-treatment temperature of 15 °C. Error bars indicate 95% fiducial limits.

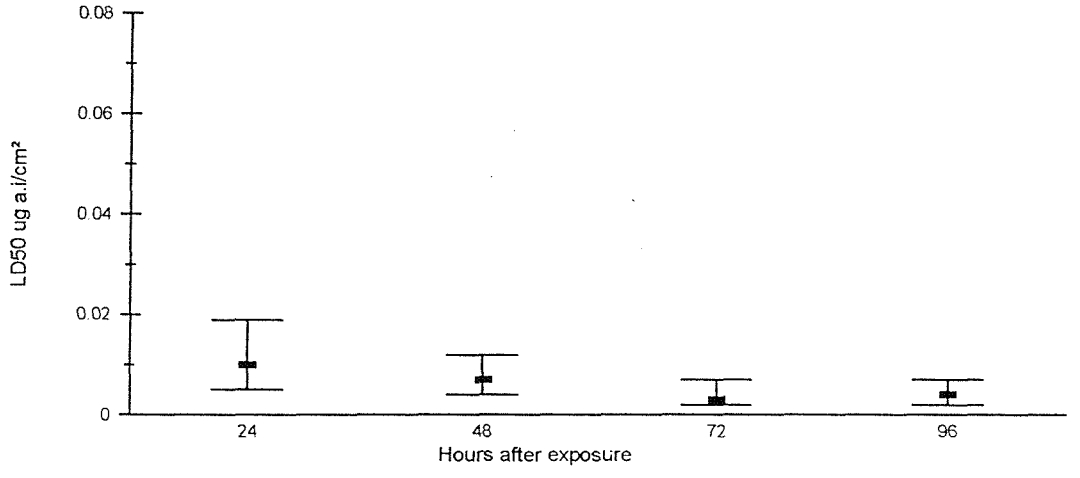


Figure 4.2c. Variation in residual LD_{50} (µg a.i./cm^2) values for S. avenae over time at a post-treatment temperature of 20°C. Error bars indicate 95% fiducial limits.

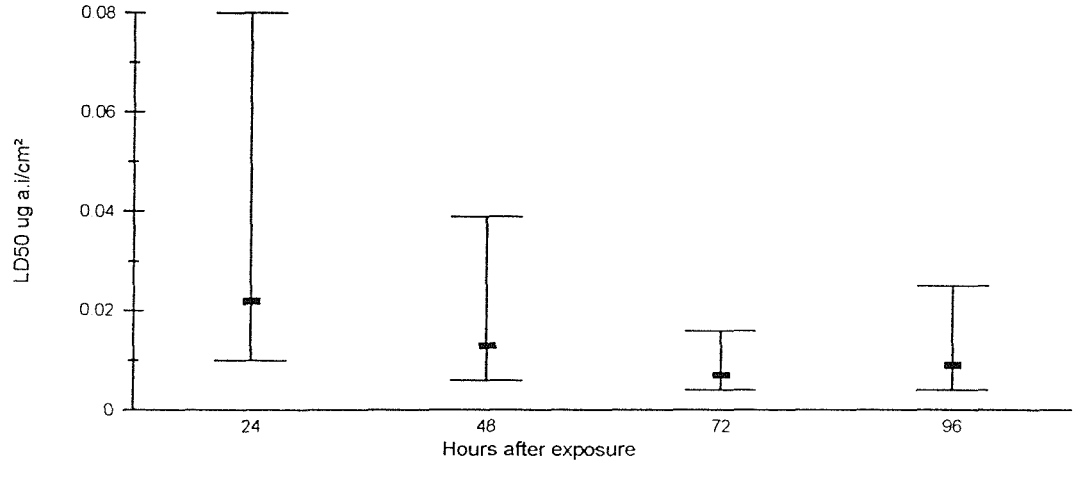


Figure 4.3a. Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

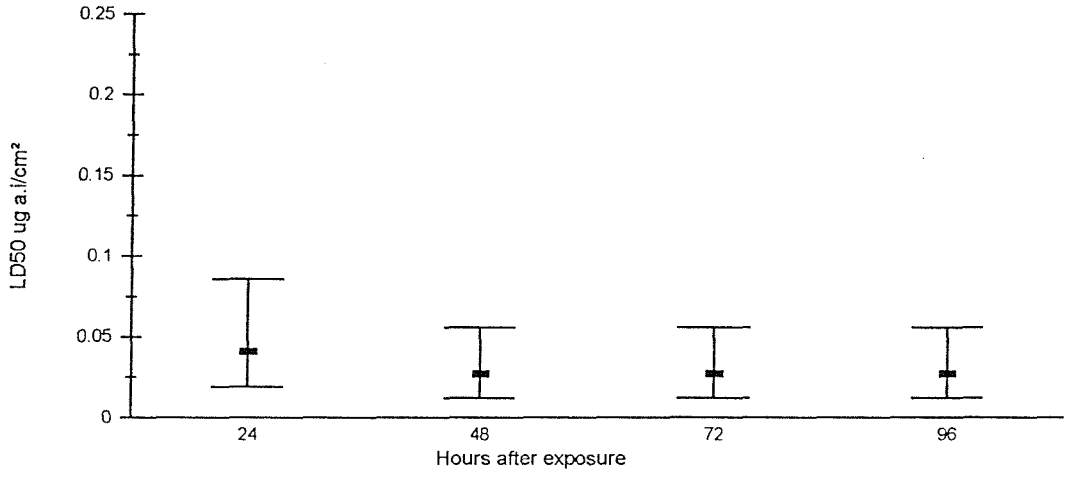


Figure 4.3b Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 15°C. Error bars indicate 95% fiducial limits.

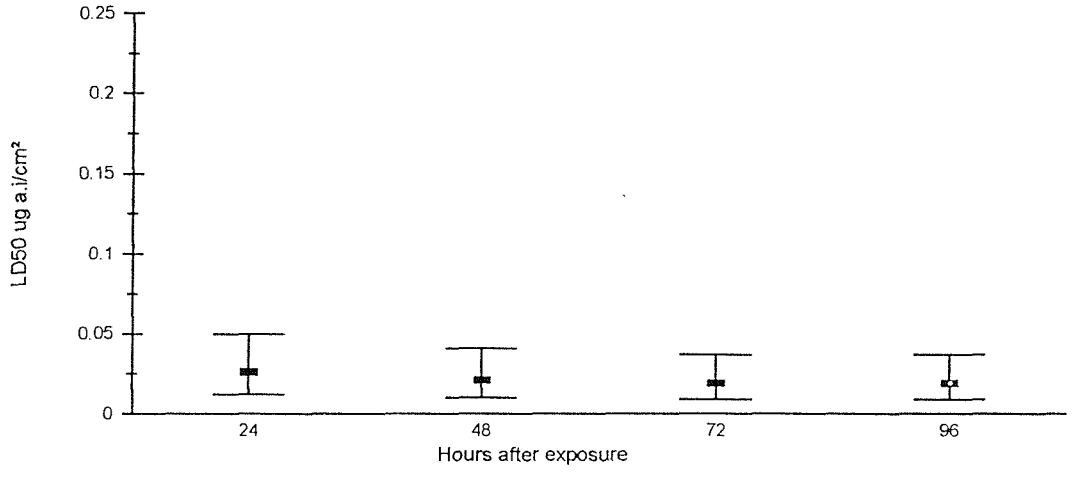


Figure 4.3c. Variation in residual LD_{50} (µg a.i./cm^2) values for *R. padi* over time at a post-treatment temperature of 20°C. Error bars indicate 95% fiducial limits.

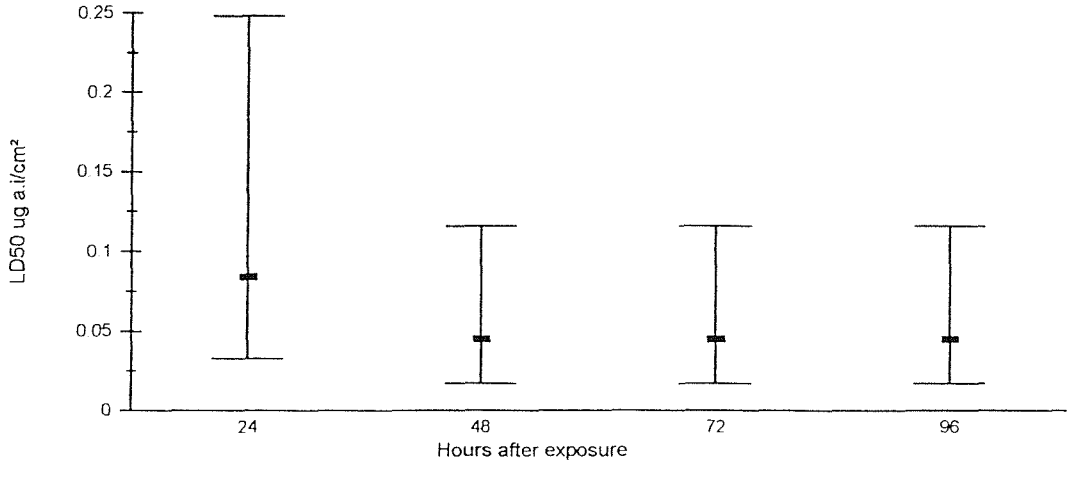


Figure 4.4a. Variation in residual LD_{50} (µg a.i./cm^2) values for S. avenae over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

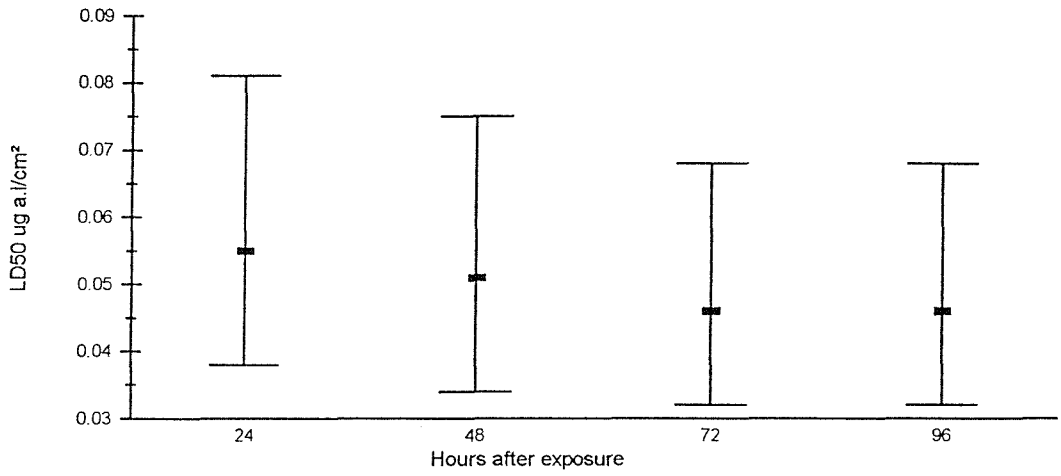


Figure 4.4b Variation in residual LD_{50} (µg a.i./cm^2) values for *S. avenae* over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

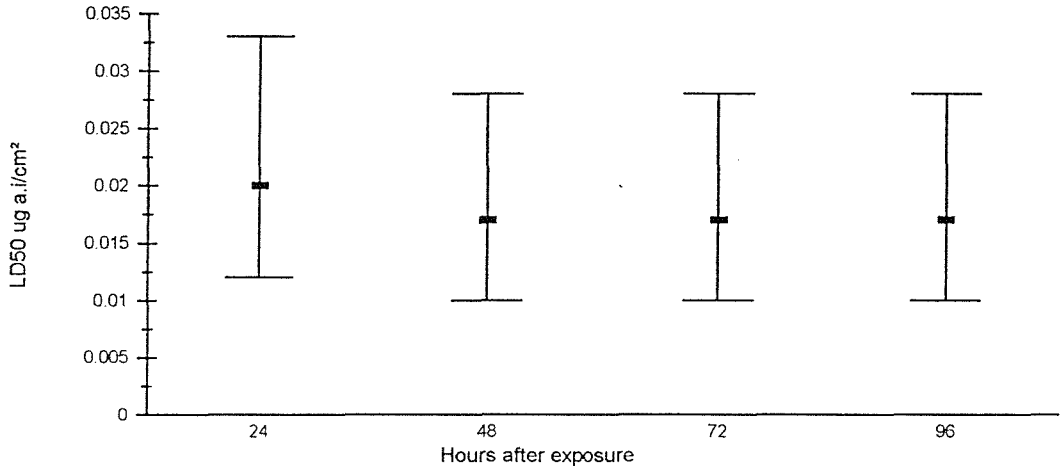


Figure 4.4c. Variation in residual LD_{50} (µg a.i./cm^2) values for *S. avenae* over time at a post-treatment temperature of 10°C. Error bars indicate 95% fiducial limits.

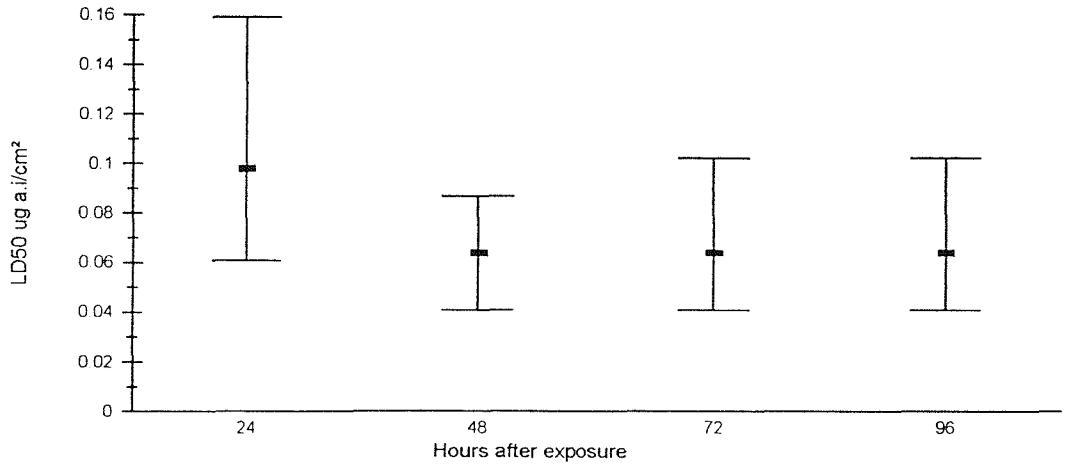


Table 4.1. 72 h probit statistics of response to residual deposits of l-cyhalothrin of *S. avenae* and *R. padi* at the different temperatures.

Aphid species	Temperature (°C)	Probit equation	LD_{50} (95% CI) (detransformed) mg a.i. / cm^2	c^2 (d.f.) Significance[a]
R. padi	10	y = 3.34 + 1.49 x	0.006 (0.003-0.011)	ns (2)
	15	y = 3.16 + 1.49 x	0.008 (0.004-0.015)	ns (2)
	20	y = 3.04 + 1.49 x	0.009 (0.005-0.018)	ns (2)
S. avenae	10	y = 3.64 + 1.32 x	0.002 (0.001-0.003)	ns (2)
	15	y = 3.21 + 1.32 x	0.004 (0.002-0.007)	ns (2)
	20	y = 2.79 + 1.32 x	0.008 (0.004-0.016)	ns (2)

[a] Significance level: ns, not significant; *, $p < 0.05$

(*R.padi* parallelism test Chi square = 0.072; df=2; p>0.05)
(*S. avenae* parallelism test Chi square = 2.537; df=2; p>0.05)

Table 4.2. 72 h probit statistics of response to residual deposits of dimethoate of *S. avenae* and *R. padi* at the different temperatures.

Aphid species	Temperature (°C)	Probit equation	LD_{50} (95% CI) (detransformed) mg a.i. / cm^2	c^2 (d.f.) Significance[a]
R. padi	10	y = 2.81 + 1.01 x	0.027 (0.012-0.056)	ns (2)
	15	y = 3.01 + 1.01 x	0.019 (0.009-0.037)	ns (2)
	20	y = 2.57 + 1.01 x	0.045 (0.016-0.115)	ns (2)
S. avenae	10	y = 5.51 + 2.17 x	0.046 (0.032-0.067)	ns (2)
	15	y = 6.39 + 2.17 x	0.017 (0.009-0.028)	ns (2)
	20	y = 5.22 + 2.17 x	0.064 (0.041-0.102)	ns (2)

[a] Significance level: ns, not significant; *, $p < 0.05$

(*R. padi* parallelism test Chi square = 0.829; df=2; $p>0.05$)
(*S. avenae* parallelism test Chi square = 2.674; df=2; $p>0.05$)

Table 4.3. Relative median potency (RMP) comparing the relative toxicity of l-cyhalothrin to each aphid species over the different temperature ranges.

Aphid species	Temperature ratio range (C)	RMP (95% CI) (72 h)
R. padi	10:15	0.76 (0.32-1.70)
	10:20	0.63 (0.25-1.40)
	15:20	0.82 (0.34-1.87)
S. avenae	10:15	0.47 (0.16-1.12)
	10:20	0.23 (0.06-0.58)
	15:20	0.49 (0.17-1.14)

RMP calculations obtained from the LD_{50} values at each temperature after 72 h.

Table 4.4. Relative median potency (RMP) comparing the relative toxicity of dimethoate to each aphid species over the different temperature ranges.

Aphid species	Temperature ratio range (C)	RMP (95% CI) (72 h)
R. padi	10:15	1.60 (0.52-6.03)
	10:20	0.57 (0.15-1.71)
	15:20	0.36 (0.08-1.08)
S. avenae	10:15	2.52 (1.32-5.79)
	10:20	0.73 (0.38-1.33)
	15:20	0.29 (0.11-0.58)

RMP calculations obtained from the LD_{50} values at each temperature after 72 h.

Table 4.5. Susceptibilty indices comparing relative toxicities of l-cyhalothrin and dimethoate over the different temperature ranges.

Compound	Temperature (°C)	Susceptability ratio (*R. padi*:*S. avenae*) (± 95% CI) (72 h)
l-cyhalothrin	10	2.71 (± 0.14)
	15	1.07 (± 1.03)
	20	1.14 (± 2.05)
dimethoate	10	0.94 (± 1.30)
	15	0.65 (± 0.14)
	20	1.04 (± 1.28)

Susceptability ratios were obtained by calculating a mean ratio from the LD_{10}, LD_{30}, LD_{50}, LD_{70} and LD_{90} doses for each respective pair of temperatures after 72 h.

intervals) (Table 4.1.).

No aphids showed symptoms of knockdown one hour after exposure to dimethoate, but the number of aphids showing symptoms was observed to increased with time. For both aphid species the toxicity relationship changed between the different temperatures, the LD_{50} value for both aphid species decreased between 10 and 15°C, and increased between 15 and 20°C (Table 4.2). The LD_{50} values were 0.027, 0.019 and 0.045 μg a.i. / cm^2 for *R. padi* and 0.046, 0.017 and 0.064 μg a.i. / cm^2 for *S. avenae* at 10, 15 and 20°C respectively (Table 4.2.).

In order to investigate the toxicity of the two compounds to aphids exposed to laboratory applied residues, ratios that gave an estimate of the relative residual toxicity of the compounds at the LD_{50} level for each aphid species at the two temperatures being compared were calculated. If the confidence interval contained a value of 1 then there was reason to suspect that the insecticide was equally as toxic at both temperatures compared. The results show that λ-cyhalothrin was equally as toxic at the all temperatures compared for *R. padi* (Table 4.3.). For *S. avenae*, λ-cyhalothrin was significantly more toxic at 10 than 20°C (Table 4.3.). For *R. padi*, dimethoate was equally as toxic over all the temperatures (Table 4.4.), but dimethoate was more toxic at 15 than

20°C for *S. avenae* (Table 4.4).

It was previously shown that *R. padi* was generally less susceptible than *S. avenae* to both λ-cyhalothrin and dimethoate by topical application. In order to quantify any differences between the aphids exposed to residual deposits of the two insecticides a sequence of lethal dose ratios calculated from the dose response statistics for each aphid at a particular temperature was iterated. The sequence of doses selected represented responses between LD_{10} and LD_{90} to allow for differences between probit slopes. The mean values obtained were termed "Susceptibility indices". These ratios gave an estimate of the relative differences in the residual toxicity of the compounds at each temperature for the two aphid species. *S. avenae* was more susceptible than *R. padi* when exposed to residues of λ-cyhalothrin at 10°C. All other indices were close to one indicating that there was no difference in susceptibility at these temperatures (Table 4.5.). *R. padi* was more susceptible than *S. avenae* when exposed to residual deposits of dimethoate at 15°C. All other indices were close to 1 indicating no difference in susceptibility (Table 4.5.).

ii) Residual toxicity of field deposits of λ-cyhalothrin

Continuous exposure to field deposits of λ-cyhalothrin;

LT_{50} values could only be calculated for *R. padi* at 15°C and *S. avenae* at 10 and 15°C, because of significant heterogeneity of the data at the other temperatures (Table 4.6.). At 15°C the LT_{50} for *S .avenae* was significantly lower than that of *R. padi* indicating that this aphid was more susceptible to residues of λ-cyhalothrin. From the dose-response curves mortality of *R. padi* (Fig 4.5.) and *S. avenae* (Fig 4.6.) was related to post-treatment temperature. For both aphid species the residues were more toxic as the post-treatment temperature decreased.

Persistence of effects of field residues at different post-treatment temperatures;

The toxicity of the residual deposits declined with time for both *R. padi* (Fig 4.7.)

Table 4.6. LT_{50} values for *R. padi* and *S. avenae* continually exposed to field residues of l-cyhalothrin at different post-treatment temperatures.

Aphid species	Temperature °C	LT_{50} hours (95% fiducial limits)	Slope (± SE)	Heterogenity
R. padi	10	n.c.	0.76 (± 0.42)	0.11
	15	89 (64 - 184)	1.41 (± 0.44)	0.37
	20	n.c.	0.35 (± 0.48)	0.10
S. avenae	10	28 (9 - 42)	1.44 (± 0.44)	0.07
	15	40 (19 - 57)	1.43 (± 0.43)	0.24
	20	n.c.	0.79 (± 0.42)	0.13

n.c. LT_{50} value could not be calculated

Table 4.7. LT_{50} values for *R. padi* and *S. avenae* continually exposed to field residues of l-cyhalothrin at different post-treatment temperatures.

Aphid species	Temperature °C	LT_{50} hours (95% fiducial limits)	Slope (± SE)	Heterogenity
R. padi	10	21 (11 -28)	2.87 (± 0.69)	0.41
	15	n.c*	2.12 (± 0.89)	0.65
	20	n.c.*	-	-
S. avenae	10	40 (30 - 49)	2.79 (± 0.57)	0.74
	15	32 (23 - 39)	3.10 (± 0.60)	0.63
	20	23 (13 - 30)	2.94 (± 0.68)	0.92

n.c. LT_{50} value could not be calculated
* mortality did not reach 50%

and *S. avenae* (Fig 4.8.), and there was higher aphid mortality as the temperature decreased. *S. avenae* was again more susceptible to the residues of λ-cyhalothrin than *R. padi*. The time when residues of λ-cyhalothrin still killed 50% of the aphids placed onto the treated leaves could only be calculated at 10°C for *R. padi*, as 50% kill was not achieved at the other post-treatment temperatures (Table 4.6.). For *S. avenae* this time decreased as the post-treatment temperature increased (Table 4.6.). At 10°C the duration of these effects were longer for *S. avenae* than *R. padi*. Aphids scored as knockdown and then clip-caged onto untreated wheat leaves all recovered after 24 h.

DISCUSSION

Post-treatment temperature appears to affect the residual toxicity of laboratory and field applied doses of λ-cyhalothrin. As the post-treatment temperature decreased there was an increase in the residual toxicity of laboratory applied doses (Table 4.2.), a decrease in LT_{50} values when continuously exposed to field applied residues (Table 4.6.) and an increase in the toxicity of residues of different ages (Table 4.7.).

In the laboratory experiment the aphids were exposed to λ-cyhalothrin at the same temperature minimising behavioural or physicochemical differences that may have affected insecticide uptake. Standardising the exposure conditions may have increased the possibility of all aphids taking up an equal dose of insecticide through residual contact. Post-treatment recovery may therefore be due to temperature dependent metabolism of the insecticide rather than differences in insecticide uptake or penetration into the insect (Croft, 1990; Hill *et al.*, 1993; Heimbach & Baloch, 1994). Aphids that were removed from the field treated leaves all recovered from showing symptoms of knockdown within 24 h, this indicates that continued exposure to an insecticide may be important in aphid pest control in the field (Hill *et al.*, 1993). Moving the aphids to a higher temperature than that at which the aphids were originally kept at may also have been important in their recovery. It was shown by Heimbach & Baloch (1994) that the recovery of *Poecilus cupreus* (Coleoptera: Carabidae) treated with λ-cyhalothrin remained knocked down for longer as the ambient temperature decreased even when

not exposed to residual deposits of the insecticide.

The effect of post-treatment temperature on the toxicity of residual deposits of dimethoate in the laboratory was more complex than λ-cyhalothrin. Comparison of LD_{50} values for *S. avenae* and *R. padi* (Table 4.2.) showed that dimethoate had a positive temperature co-efficient between 10 and 15°C and a negative temperature co-efficient between 15 and 20°C. Comparison of topical LD_{50} values for dimethoate and the two aphid species showed a positive temperature coefficient in both cases (Table 3.3). This change in the relationship as the temperature increases has been shown in other studies (e.g. Hinks, 1985; Grafius, 1986). The change however appears to be limited to the pyrethroids (Hinks *et al.*, 1987), and has been demonstrated for the residual uptake of pyrethroid insecticides (Schmidt & Robertson, 1986; Rahman & Yadar, 1989). Biological or physicochemical factors reducing uptake or penetration of the insecticide into the aphid may have been responsible for the change in the temperature coefficient. At 20°C there may have been increased volatilisation of the insecticide from the cuticle of the insect before it had time to penetrate into the insect or a change in behaviour might have increased removal of insecticide deposits from the aphid, both would have reduced the toxicity of the insecticide (Croft, 1990).

Studies have indicated that temperature-toxicity coefficients vary between species for the same insecticide (Iordanou & Watters, 1969; Sogorb *et al.*, 1983; Johnson, 1990;). It would also appear that changing the route of exposure can alter the temperature-toxicity co-efficient observed between two species for the same insecticide. After exposure to residual deposits of λ-cyhalothrin in the laboratory the aphids were equally as susceptible as each other (Table 4.5). When treated topically with the insecticide *S. avenae* was always more susceptible than *R. padi* (Tables 3.4. & 3.5). Investigating differences in the susceptibility to pesticides of both beneficial and pest organisms through different routes of exposure at different post-treatment temperatures would therefore be of value in determining the risk posed to each species after an insecticide application.

The importance of residual toxicity to aphids has not been studied in great detail (Croft, 1990; Hill *et al.*, 1993). Direct contact with the insecticide appears to be the most important route of toxicity (Chapter 2), but aphids are continuously falling from and returning to the crop canopy (Winder, 1990). Aphids produce alarm pheromone in response to an insecticide application (Montgomery & Nault, 1971) causing possible increases in the rate of drop-off. Therefore the role of residual toxicity might be an important route of exposure as aphids return to the crop canopy over treated plants. Trends in the decay at different post-treatment temperatures provide a basic guide to persistence of effects of insecticides applied in the field. They suggest that for pyrethroids ambient temperature could prolong the toxicity of an applied insecticide. Although the aphids may not be killed by the residues uptake of a sufficient dose may cause knockdown and increase the chance of being predated upon by beneficial insects. The trends in declining toxicity are unlikely to be solely due to the loss of the insecticide by metabolism, breakdown leaching or plant uptake (Unal & Jepson, 1991). They also indicate a change in the bioavailability at lower temperatures of the insecticide applied (Everts, 1990).

The toxicity of field residues of insecticides decline with time (Westcott *et al.*, 1987; Jepson, 1988; Jepson & Unal, 1991), in consequence, when exposed to residues of field applied λ-cyhalothrin, ambient temperature influenced the time to which a 50% response of the exposed aphid population could be elicited. When aphids were continuously exposed to residual deposits mortality accumulated over the days following exposure with an end point of approximately 72 h (Figs 4.5 & 4.6.). 72 h post-spray was also the point when the toxicity of the field residues decreased significantly (Figs. 4.7 & 4.8.), and this would appear to account for an end point of 72 h when aphids were continuously exposed. From the trend of mortality declining with time, it could be postulated that the final cumulative aphid mortality value gives an estimation of the maximum potential mortality that would result for immigrant aphid populations exposed to residues continuously following their arrival at different times after spraying. At lower post-application ambient temperatures in the field immigrant and residing aphid populations are more likely to suffer higher mortality.

The increase in efficacy of insecticides at different temperatures could support the use of reduced rates of insecticides to reduce variable costs and adverse effects on beneficial organisms. A farmer, if spraying an organophosphorous compound, could consider applying it early in the morning as the temperature will rise during the day. In contrast, a pyrethroid could be applied late in the day to take advantage of temperatures falling in the evening. Considering the temperature differences between autumn and summer application of pyrethroids, and that 65% of cereals may be sprayed before the end of December (Davies *et al.*, 1993), the possibility of reducing doses in the autumn should be considered.

CHAPTER 5

THE EFFECT OF SUB-LETHAL DOSES OF AN INSECTICIDE ON MEASUREMENTS OF APHID POPULATION GROWTH AND PERFORMANCE

INTRODUCTION

In a field situation, pests survive an insecticide application by either avoiding contact with the insecticide or surviving sub-lethal doses that impinge upon them. When using reduced dose-rates the likelihood of receiving a sub-lethal dose may increase because of the lower amounts of active ingredient applied and further dilution resulting from stratification in the crop (Cilgi & Jepson, 1992). The reduced efficacy also leaves a residual pest population (Chapter 2), so there is a danger of resistance developing or sub-lethal doses stimulating nymph production (Gordon & McEwen, 1984; Jackson & Wilkinson, 1985). Stimulating fecundity will ultimately affect aphid population growth, possibly resulting in an increase in pest numbers after treatment. If there are insufficient predators and parasitoids within the crop, this could result in pest control failure. This effect is known as resurgence (Chelliah *et al.*, 1980), and reduction in predation and stimulation of reproduction by sub-lethal doses of an insecticide being the important factors for its occurrence (Roan & Hopkins, 1961; Luckey, 1968; Dettrich *et al.*, 1974; Kerns & Gaylor, 1992). The phenomenon has been well documented for the brown plant hopper, *Nilaparvata lugens* (Stal) (Homoptera: Delphacidae) (Chelliah *et al.*, 1980)

The danger of pyrethroids stimulating nymph production (Gordon & McEwen, 1984; Jackson & Wilkinson, 1985), could cause problems for reduced rate insecticide use and since population growth is linked with temperature, the reduced efficacy of pyrethroids at higher temperatures might further enhance population growth A study was therefore undertaken seeking evidence about one of the underlying mechanisms of resurgence, effects of sub-lethal doses of insecticide on measures of aphid population growth and performance. Several measures of aphid population performance are used to screen crop plants for resistance to aphids. These include fecundity and development time (Holt, 1981), mean relative growth rate (MRGR) (van Emden, 1969) and estimates

of intrinsic rate of increase achieved by rearing aphids under controlled conditions until reproduction has ceased (Dean, 1974; Wyatt & Brown, 1977). These measures are suitable for assessing the effects of sub-lethal insecticide doses on aphid population growth after treatment with an insecticide. Population growth and insecticide toxicity are both influenced by temperature, this study therefore investigated potential population growth after treatment with a sub-lethal dose over a range of post-treatment temperatures.

MATERIALS AND METHODS

Aphids used in the experiment were taken from a laboratory culture maintained on wheat (var. Knirps) at 20°C/16:8 L:D. The culture was maintained as described in chapters 3 and 4. First instar nymphs of *Rhopalosiphum padi* (Homoptera: Aphididae) were removed carefully from the culture using a fine paint brush. The aphids were weighed on a microbalance (0.0001g) and then treated at room temperature with an 11.5 nl droplet of λ-cyhalothrin, applied by a hand-held micro-applicator (Burkhard Manufacturing Co Ltd) fitted with a gas tight 50 ul syringe (SGE, Australia) under a binocular microscope. Lutenosol (BASF) was used as the pesticide diluent and as the treatment for control individuals. It has previously been demonstrated that pyrethroids are more toxic at low temperatures to this aphid species (Chapter 3). The dose of insecticide applied to the aphids was dependent on post-treatment temperature (see below); the dose being defined as sub-lethal if a maximum of 20% of the treated aphids died. A total of 30 treated and 30 control aphids were used at each temperature. After dosing, the aphids were clipped-caged individually onto 10 day old wheat seedlings (var Knirps) and reared at post-treatment temperatures of 25, 18 or 12°C in environmentally controlled chambers (16:8 L:D, RH 70-80%).

The clip cages were checked daily and the aphids' development time (from 1st instar nymph to adult) and the eventual adult weight determined. Mean relative growth rate (MRGR), a measure of an individual aphids average growth rate per day, was then calculated using the formula described by Leather & Dixon (1984):

$$MRGR\ (mg, mg^{-1}, day^{-1}) =$$

$$\frac{\ln W_2(mg) - \ln W_1(mg)}{t_2 - t_1}$$

Where W_1 and W_2 are the weights at birth and maturity respectively, and $(t_2 - t_1)$ is the time taken to develop from birth to maturity (i.e. the development time).

The number of nymphs produced over a time period equal to the aphids development time was recorded. This was used to calculate r_m values (Fisher, 1920; Wyatt & White, 1977; Leather & Dixon, 1984), a measure of the intrinsic rate of natural increase for each aphid:

$$r_m = 0.74\ (\ln F_D / D)$$

Where F_D is the number of nymphs produced over a period of time equal to that of the pre-reproductive period (D) and 0.74 is a constant.

After the adult weight had been measured, the aphid was clip-caged onto a fresh seedling of the same age as the original. Any nymphs that were produced before the adult was weighed were removed and excluded from subsequent calculations.

The LD_{20} values at 25, 18 and 10°C were 0.036, 0.018 and 0.009 ng a.i./insect respectively. After checking for equality of variances, independent t-tests on $\log_{10}$ transformed adult weight, development time, MRGR, numbers of nymphs produced and r_m data were calculated. Model 1 regression analysis (SPSS, v 6.0.) was used to compare development time at each temperature independent of the dose applied.

RESULTS

The data for development time, MRGR and r_m measurements were plotted against temperature and Model 1 linear regressions fitted to the scatter diagrams.

Table 5.1. Mean values of variables (± 95% C.I.) for control and treated aphids at the three post-treatment temperatures.

Temperature	Variable (units)	Control aphids (± 95% C.I.)	Treated aphids (± 95% C.I.)
25°C	Adult weight (mg)	0.35 (± 0.041)	0.33 (± 0.002)
	Development time (days)	5.04 (± 0.186)	5.18 (± 0.027)
	MRGR mg, mg^{-1}, day^{-1}	0.61 (± 0.041)	0.57 (± 0.004)
	Number of nymphs	18.80 (± 2.353)	16.68 (± 2.589)
	r_m (Intrinsic rate of natural increase)	0.43 (± 0.021)	0.40 (± 0.004)
18°C	Adult weight (mg)	0.39 (± 0.041)	0.36 (± 0.041)
	Development time (days)	8.61 (± 0.310)	8.30 (± 0.477)
	MRGR mg, mg^{-1}, day^{-1}	0.38 (± 0.021)	0.37 (± 0.021)
	Number of nymphs	18.52 (± 2.766)	17.04 (± 2.841)
	r_m (Intrinsic rate of natural increase)	0.26 (± 0.021)	0.25 (± 0.021)
12°C	Adult weight (mg)	0.52 (± 0.041)	0.35 (± 0.063)
	Development time (days)	13.16 (± 0.433)	15.10 (± 0.709)
	MRGR mg, mg^{-1}, day^{-1}	0.24 (± 0.021)	0.18 (± 0.021)
	Number of nymphs	18.04 (± 2.518)	12.14 (± 2.841)
	r_m (Intrinsic rate of natural increase)	0.16 (± 0.021)	0.12 (± 0.021)

Table 5.2. Results of Model 1 regression analysis comparing variables with temperature for control (d.f. = 73, $p < 0.05$) and treated (d.f. = 67, $p < 0.05$) aphids.

Aphid treatment	Variable	Equation	t	r^2
Control	Development time	y = 20.15 - 0.62 x	-29.16	0.92
	MRGR	y = -0.12 + 0.03 x	17.78	0.81
	r_m	y = -0.1 + 0.02 x	18.46	0.82
Treated	Development time	y = 23.26 - 0.75 x	-20.44	0.86
	MRGR	y = -0.18 + 0.03 x	21.53	0.88
	r_m	y = -0.14 + 0.02 x	18.12	0.83

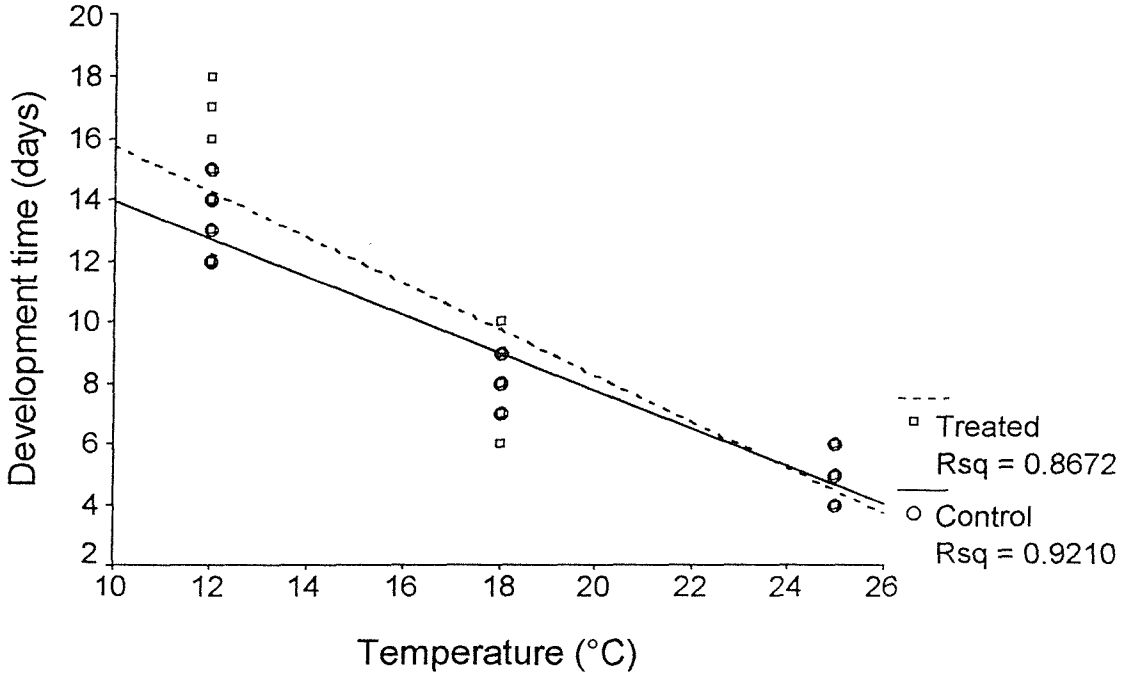


Figure 5.1. Model 1 regression analysis of the effect of temperature on the development time of the control and insecticide treated aphids (see Table 5.2. for statistics).

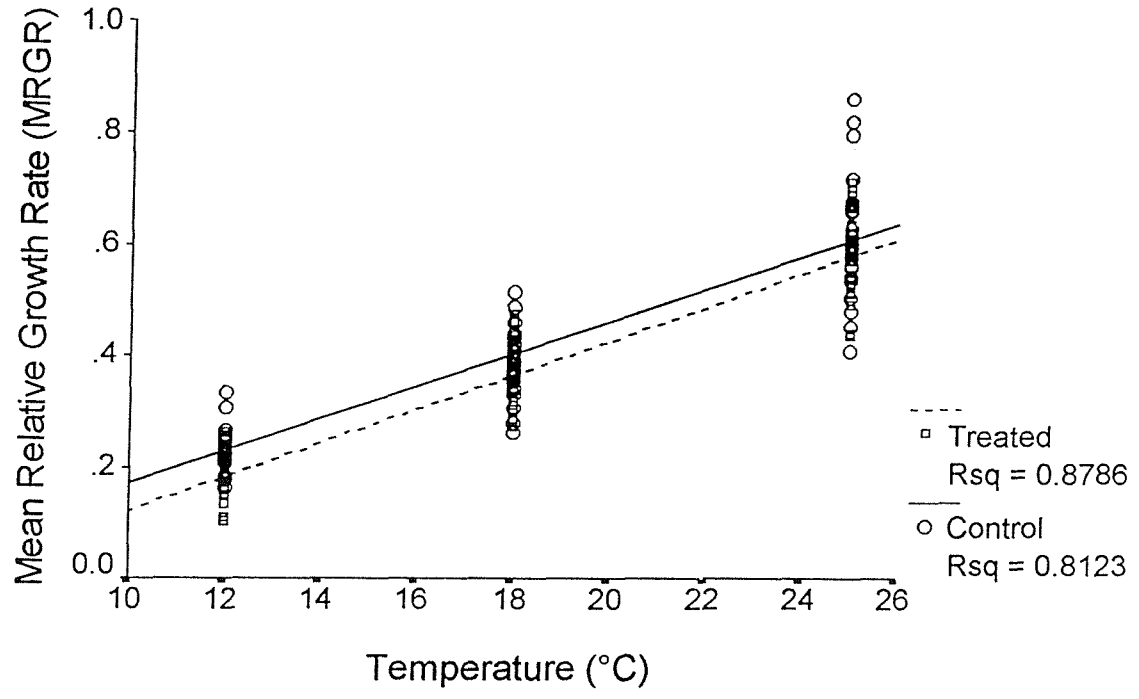


Figure 5.2. Model 1 regression analysis of the effect of temperature on the mean relative growth rate of the control and insecticide treated aphids (see Table 5.2. for statistics).

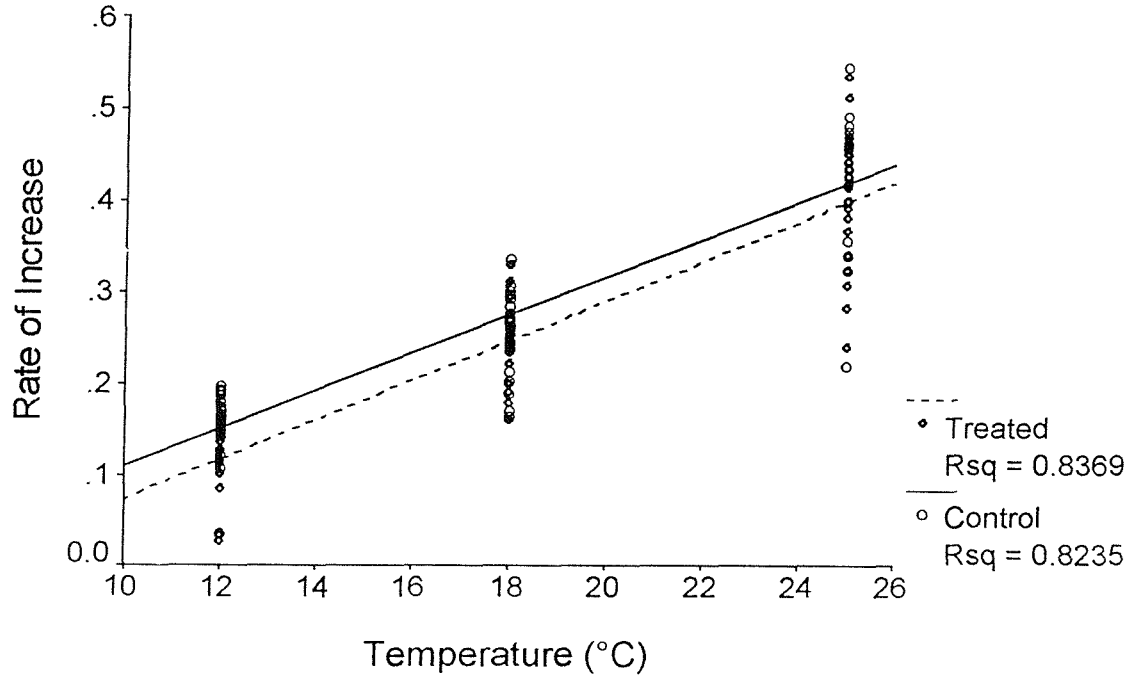


Figure 5.3. Model 1 regression analysis of the effect of temperature on the intrinsic rate of increase of the control and insecticide treated aphids (see Table 5.2. for statistics).

Although there was a large scatter of the points around the fitted line, there were significant linear relationships between all three measurements of aphid performance and temperature for both the control and treated aphids (Table 5.2.).

As the temperature increased the number of days to complete development decreased (Fig. 5.1). The slopes of the regression lines for development time between control and treated aphids were significantly different ($t = 3.088$, d.f. = 135 $p < 0.05$), although this was due to the low t value caused by the better fit of the control than treated data. The MRGR and r_m measurements both increased with increasing temperature (Figs 5.2. & 5.3.). There were no significant differences between the slopes of the regression lines for MRGR and temperature ($t = -0.6481$, d.f. = 135, $p > 0.05$) and r_m and temperature ($t = -0.603$, d.f. = 135, $p > 0.05$) for the control and treated aphids.

At 18 and 25°C there were no significant differences between control and treated aphids for the different variables measured (Table 5.1.). The mean values of the variables measured were however always higher for control than treated aphids. At 12°C there were significant differences between the control and treated aphids for all the variables measured. Control aphids were heavier when adults ($t = 5.21$, d.f. 44, $p < 0.05$), had a shorter development time ($t = -5.03$, d.f. 44, $p < 0.05$), had higher MRGR ($t = 5.91$, d.f. 44, $p < 0.05$), produced more nymphs when adults ($t = 2.75$, d.f. 44, $p < 0.05$) and had higher r_m values ($t = 4.17$, d.f. 44, $p < 0.05$).

DISCUSSION

Temperature has been shown to affect the measurements of aphid population performance investigated in this study. The effect of the insecticide on these measurements appears temperature dependent. The results show that the rate of development from nymph to adult decreases with increasing temperature. Other studies have shown that temperature has a marked effect on the rate of increase of cereal aphids (Dean, 1974; DeLoach, 1974; Wyatt & Brown, 1977). Dean (1974) showed over a similar temperature range that development time decreased with increasing temperature for the aphids *Sitobion avenae* (Say), *Metapolophium dirhodum* (Wlk) and

Rhopalosiphum padi (L.). In the case of *R. padi* a faster development at 25 °C gave a maximum rate of increase at that temperature, this can also be seen in these results (Table 5.1.). Only at 12°C were significant differences observed between the control and treated aphids (Table 5.1.), and this may be a result of the influence that low temperature has on the toxicity of λ-cyhalothrin.

As the temperature increased the final adult weight decreased and development time increased. Differences between treated and untreated aphids were only again significant at 12°C (Table 5.1.) with the treated aphids taking longer to develop. Further differences between development time and adult weight may have been observed had the aphids been checked more often, since the aphids will have matured at different times during the day. Although growth is more rapid at higher temperatures, larger aphids are not produced because development rate appears to accelerate disproportionately through the development period, causing growth to be truncated earlier (Chambers, 1979; Acrenna & Dixon, 1989). Even though the treated aphids had a longer development time at 12°C, their adult weight was much less than the untreated controls. This suggests a sub-lethal effect of the insecticide and the lower adult weight has further implications for other measures of aphid performance.

Reproductive rate is positively correlated with both aphid size and temperature (Wratten, 1977; Watt, 1979). Aphids usually achieve their highest reproductive rates early in life (Dixon, 1989) and the lack of a correlation in this study may have been because total fecundity was being measured. Gordon & McEwen (1984) found that female *M. persicae* reared on cabbage leaves dipped in azinphosmethyl produced more offspring in the first 3 days of their reproductive life than those reared on untreated leaves.

The effect of treatment with a sub-lethal dose of an insecticide on nymph production is not clear, but this study would suggest that any effect is temperature dependent, Only at 12°C are significant differences in nymph production between treated and untreated aphids seen. At 18 and 25°C lower mean numbers of nymphs were produced by the treated aphids but the differences were not significant (Table 5.1.). ffrench-Constant *et al.* (1988) also showed that other pyrethroids did not affect nymph

production over similar temperatures. Where increases in fecundity have been observed, it has been proposed to be a result of the direct action of the insecticide on the reproduction of the organism (Sun, 1945; Bartlett, 1968; Parry & Ford, 1972; Jackson & Wilkinson, 1985; Lowery & Sears, 1986 a & b). The susceptibility of the aphids to an insecticide can also affect fecundity, susceptible aphids can have reduced fecundity whilst in resistant aphids fecundity may be increased (Ffrench-Constant *et al.*, 1987).

The mean relative growth rate increased with increasing temperature and there was a corresponding increase, with temperature, of the r_m value as well. It was only at 12°C that significant differences were seen between the control and treated aphids for the MRGR and r_m values and this was due to a reduction in adult weight for the treated aphids at 12°C. The pest status of many aphids is partly a result of their remarkable rate of increase (Dixon, 1989). However the r_m value of a single aphid is likely to be of limited value for determining population increase in the field because aphid populations are not of a stable age distribution (Carter *et al.*, 1980). However rate of increase has been useful for comparing the potential contributions that different morphs of a species (Dixon & Wratten, 1971) or different species make to population growth when reared under the same conditions (Leather & Dixon, 1984).

There are other factors that may affect the measures of aphid performance investigated in this study. Birth weight also affects development time, if aphids are small at birth they take considerably longer to reach maturity than when large, however before treatment aphids of similar size were selected to minimise such variation. There were also no significant differences in the nymph weights of the control and treated aphids used in the experiments. Changes in rearing conditions may affect development time (Dixon & Wratten, 1971), the aphids in this experiment had been reared continuously in the laboratory and after treatment were allowed to develop under similar conditions apart from ambient temperature. However changes from high to low temperatures have been known to reduce aphid performance (T. Thieme pers. comm.). Even so the differences at 12°C would still appear to be attributed to the effect of the insecticide. Studies in which food quality has been measured indicate that aphids feeding on high quality food develop more quickly and achieve a larger size than when feeding on poor quality food

(Mittler, 1958). Plants in this study were expected to be of uniform quality, reducing variation from this source.

The only extrinsic factor that varied between the aphids was whether or not they were treated with the insecticide. The results therefore suggest that λ-cyhalothrin has no stimulatory effect on aphids. Kerns & Gaylor (1992) also showed that sub-lethal doses of an insecticide do not affect r_m, development time or nymph production. However, further research needs to be carried out on the effects observed at 12°C; these may result from the negative temperature coefficient displayed by this pyrethroid. Therefore, because the insecticide is intrinsically more susceptible at lower temperatures, a treated aphid may have to direct resources that would normally be involved in growth into detoxifying the insecticide, reducing performance at this temperature. This could possibly be investigated by comparing the size of the fat body of treated aphids. This plays a major part in the metabolism of pesticides and also as stores of energy for growth and reproduction (Wigglesworth, 1972).

CHAPTER 6

THE INFLUENCE OF SUBSTRATE TYPE ON THE RESIDUAL TOXICITY OF REDUCED RATE INSECTICIDES TO A RANGE OF CARABIDAE

INTRODUCTION

Beneficial ground dwelling arthropods, such as carabid beetles, are important predators of aphids (see Chapter 1). In stable environments carabid populations may fluctuate little from year to year, they could potentially therefore have significant and constant mortality effects on aphid pests (Luff, 1982). Undesirable side-effects such as pest resurgence and outbreaks of secondary pests may occur when large quantities of insecticides which are toxic to predators are applied to a crop over prolonged periods. This has often been attributed in part to a loss of beneficial organisms and recent large scale studies have confirmed that populations of certain beneficial organisms may be driven to extinction by current insecticide application practices (Grieg-Smith *et al.*, 1992).

Beneficial insects within the crop ecosystem are known to be exposed to insecticides by three main routes of uptake; direct fallout from spraying, residual contact with contaminated surfaces and oral uptake from contaminated prey (Croft, 1990; Jepson, 1993). Of these three routes residual uptake will be important for ground dwelling beneficials (Mullie & Everts, 1991; Jagers op Akkerhuis & Hamers, 1992). The effect of an insecticide application on beneficial predators will be a function of species susceptibility and the dose received (Jepson *et al.*, 1987; Wiles & Jepson, 1992b). In conventional arable practice approximately, 1% of the pesticide applied to the crop may actually reach the target pest (Graham-Bryce, 1977). This suggests that beneficial predators, in either the crop or non-crop habitats, may suffer a high risk of coming into contact with lethal doses of the applied pesticide. As previously mentioned (Chapter 1) the aim of using reduced insecticide rates is to maintain adequate pest control while preserving beneficial organisms. This tactic may prevent undesirable side-effects from arising.

Criteria have been developed to test the effects of insecticides on beneficial organisms in order to minimise unwanted side-effects (Hassan, 1985, 1989 & 1992; Hassan *et al.*, 1987 & 1991; Samsoe-Peterson, 1985). This process may be useful when ranking the toxicity of pesticides to beneficial invertebrates, but because the tests often only measure responses on a single, unrealistic substrate i.e. glass, and to a single pesticide dose i.e. the recommended field rate of the pesticide, they need to be modified if we are to predict the risks posed to beneficial invertebrates after pesticide application in the real world. When considering the risks that beneficial organisms face after an insecticide application many operational, chemical, physical and ecological factors have to be considered (Hartley & Graham-Bryce, 1980; Ford & Salt, 1987; Felsot & Law, 1989; Arnold & Briggs, 1990; Gerstl, 1991). The level of exposure of both target pest and non-target beneficial species in the field will be determined by the behaviour and distribution of both the chemical and invertebrate species. The toxicity of an insecticide can be strongly affected by changes in biotic and abiotic conditions (Critchley, 1972; Croft, 1990; Everts *et al.*, 1991a; Heimbach & Baloch, 1994); an important factor is substrate type (Wiles, 1992; Heimbach *et al.*, 1992; Wiles & Jepson, 1994). bioavailability of a pesticide on soil and plant surfaces is generally negatively correlated with it's octanol/water partition coefficient (Briggs, 1973; Ford & Salt, 1987; Nicholls, 1991). It will also be affected by factors such as the thickness and architecture of the wax layer of a leaf surface (Adams *et al.*, 1987) and the organic matter (Harris, 1967; Briggs, 1981) and clay content (Arnold & Briggs, 1990) of soils. To be able to understand and predict the consequences of applied insecticides on ground-dwelling beneficial organisms, the influence of soil type should be known because any effects will be dependent on the bioavailability of the insecticide. In turn this will be affected by a number of physio-chemical factors including adsorption, degradation, binding, leaching and volatilisation (Graham-Bryce, 1977; Nicholls, 1991; Brown, 1989).

The series of laboratory bioassays described in this chapter investigated the residual toxicity of the three insecticides used in chapter 2 against non-target beneficial invertebrates. The chapter is divided between two experiments; the first experiment involved exposing a number of species of carabid beetle to a wide-range of insecticides. Exposure on glass allowed the insecticides to be evaluated for their intrinsic toxicity towards the selected beneficial organism. The second bioassay permitted determination

of the relative risks posed by deltamethrin and dimethoate residues to one of these ground beetles at rates previously applied to control aphid pest populations and, on a realistic substrate. Comparison with the toxicity of the compounds on glass and soil permitted substrate mediated-toxicity (Wiles, 1992; Wiles & Jepson, 1994) for these two compounds to be made. Estimates of the likely effect of reduced-dose rates on epigeal beneficials could then be deduced. This might be of particular value when extrapolating data generated on artificial substrates to field conditions.

MATERIALS AND METHODS

Exposure to residual deposits on glass

The carabid species chosen for the bioassays were *Agonum dorsale* (Pont.), *Demetrias atricapillus* (L.), *Bembidion lampros* Herbst and *B. obtusum* Serville. These were collected in cereal field margins and hedge banks in October 1992 at the Leckford Estate, Stockbridge, Hampshire by hand-held air aspirator and surface searching. After collection the beetles were stored in plastic boxes (10 x 15 x 27cm) containing a layer of moist soil and pellets of cat food ("Delicat", Quaker, Lartz) in a cold room at 4° C. Seventy-two hours prior to insecticide exposure the beetles were counted into groups of one hundred and stored in polystyrene boxes containing fresh cat food and damp filter paper. The boxes were then kept in a controlled environment room (18-20°C, RH 60-70 %, 16:8 L:D) until required.

The insecticides used in the bioassays were deltamethrin (Decis, 2.5% E.C. w/v), dimethoate (Croptex Dimethoate, 40% E.C. w/v) and pirimicarb (Aphox, 50% W.G. w/w). A Potter laboratory spray tower was used to apply the compounds onto 7.5 cm-square glass plates and onto glass Petri dishes (1cm x 5.5cm diameter) depending upon the test species being used. The tower was calibrated to deliver at a volume rate of 200 l/ha, equivalent to that of conventional field rate applications. Field rate and fractions of field rate were applied for each test compound (4 reps per treatment, see Table 6.1.). Controls were treated with distilled water and insecticide treatments applied in sequential order starting with the lowest dose first.

Table 6.1. Concentration of insecticides used in the two experiments.

Experiment	Insecticide	% of field rate used (all insecticides)	Field rate application (g a.i./ha)
Residual toxicity on glass	deltamethrin dimethoate pirimicarb	0.56, 0.312, 0.62, 0.125, 2.5, 5, 10, 20, 50, 100	6.25 340 140
Residual toxicity on glass and soil	deltamethrin dimethoate	10, 20, 33, 100	6.25 340

The sprayed glass plates and Petri dishes were left for approximately 1 h to dry before the test species were exposed to the insecticide residues. For the *A. dorsale* bioassay, unsprayed plastic cylinders (3cm x 5.5cm diameter) were clipped onto the sprayed 7.5 cm square glass plates. The interior of the plastic chambers had previously been coated with "Fluon" to prevent the beetles from escaping. Groups of five *A. dorsale* were then placed inside each chamber using a fine paint brush giving a total of 20 beetles.

For the other carabid species, the test apparatus was modified to form a sealed chamber to prevent the beetles escaping. A test unit comprised a 7.5 cm square glass plate and a glass Petri dish which were separated by a 1-mm-thick gasket of cork to provide an escape-proof seal. The lid and base of the chamber were held in place with a metal clip after the insects were introduced. A ventilating humidified air flow was provided to each test unit via a pair of syringe needles inserted through the cork gasket. One was connected to an aquarium pump (Elite 800, 1500 cc output/min) via tubing and the other on the opposite side, served as an outlet for the airflow. This also served to extract the pesticide vapour from the test unit to ensure that any mortality which occurred was due only to the residual effects of the chemicals. Five *B. lampros* and five *B. obtusum* were placed together in each test chamber using an aspirator giving a total of 20 beetles per treatment.

For the *D. atricapillus* experiment the beetles were kept in a cold room at 4° C for 3 h prior to exposure and placed in the test chambers in this room to reduce their activity; as *D. atricapillus* could climb out of the Petri dishes at laboratory temperatures before the glass plate was in place. Again five individuals were placed in each of the test chambers giving a total of 20 beetles.

The test chambers were then randomly placed on benches in a controlled environment room (20 ± 2°C, 16:8 L:D). Humidity inside the test chambers was *c.* 80% ± 5% (measured with a Lovibond Comparator using cobalt thiocyanate indicator paper). Assessments of the effects of the treatments were made at 24, 48 and 72 h after the insects, were initial exposed to the insecticide deposits. Individual beetles were classified according to the assessment criteria used in Chapter 2.

Exposure to residual deposits on soil and glass

Adult *A. dorsale* were collected from cereal field margins and hedge banks between September and October 1993 at the Leckford Estate, Stockbridge, Hampshire by hand-held air aspirator and surface searching. After collection the beetles were stored in plastic boxes (10 x 15 x 27cm) containing a layer of moist potting compost (JI 2) and were fed on ground cat food ('Delicat', Quaker, Lartz) in a cold room at 4°C. Seventy-two hours prior to insecticide exposure the beetles were moved to fresh boxes containing fresh cat food and damp filter papers. These boxes were then kept in a controlled environment room (19 ± 1°C. 60-70% RH, 16:8 L:D).

The insecticides used in the bioassays were deltamethrin (Decis, 2.5% E.C. w/v) and dimethoate (Croptex Dimethoate, 40% E.C. w/v). A Potter laboratory spray tower was used to apply the compounds onto 7.5 cm^2 glass plates or 9 cm diameter plastic pots, containing agricultural soil whose walls had previously been coated with 'Fluon'. The soil was collected from the Leckford Estate and subsequently analysed for water, mineral and organic content (see Table 6.3.). A 2cm layer of soil was lightly compacted into the bottom of each plastic pot and a plastic insert placed around the side of the pot prior to spraying. This ensured that the beetles would only come into contact with residual deposits of the insecticides on the soil. The tower was calibrated to deliver a

Table 6.2. Analysis of soil used in experiment investigating the effect of residual toxicity of deltamethrin on the predation of aphids by *P. cupreus*.

Soil Profile	Analysis %
Clay	3.2
Silt	10.9
Sand	59.4
Gravel	13.3
Organic matter	12.1
pH (H_2O)	6.7
Moisture	20.1

volume rate of 200 l/ha, equivalent to that of conventional field rate applications. Controls were treated with distilled water and the insecticide treatments applied in sequential order starting with the lowest dose first (4 reps per treatment, see Table 6.1.).

The sprayed glass plates and pots of soil were left for approximately 1 hour to dry after spraying before 5 beetles were exposed to the insecticide deposits of each replicate. In the case of the glass plates, a ventilated chamber (Wiles & Jepson, 1992a) was placed over them and secured with elastic bands to prevent escape of the beetles. This ventilated chamber was not needed for the pots because the beetles were unable to escape.

The glass plates and pots were then placed in random order on benches in a controlled environment room (20 ± 2°C, 55-70% RH, 16:8 L:D). Assessments of the effects of the treatments were made at 1, 2, 4, 24, 48, 72, 96 and 120 h after the insects were initially exposed to the insecticide deposits. Individual beetles were classified according to the criteria used in the previous experiments.

Statistical analysis

For both experiments Abbot's formula was used to correct for control mortality (Abbott, 1925). In the first experiment probit analysis was carried out on the 72 h dose-response data to obtain dose-response statistics (Finney, 1971). In the second experiment probit analysis was carried out on the time-response data to obtain time-response statistics for each concentration of insecticide used. Only dead insects were included in the calculations, although after 72 h few individuals remained knocked down.

RESULTS

Exposure to residual deposits on glass

The dose range in the first experiment was not selected in order to provide a span of particular mortalities, as a consequence probit analysis could not be carried out on the concentration:mortality data. Comparisons of relative toxicities were therefore based on the individual dose-response data values. This problem may be overcome by initial 'range-finding' tests that give mortality ranges of 5-95%, recommended for precision estimation (Robertson & Priesler, 1993). However comparison of the results in this way was not the intention of the experiment.

For *D. atricapillus* (Fig. 6.4.) and the two *Bembidion* species (Figs. 6.1. & 6.2), 100% mortality was observed with dimethoate at only 0.62% of the field rate. Even for the most tolerant of the carabids tested, *A. dorsale*, 100% mortality was seen at only 2.5% of the field rate (Fig. 6.3). In contrast, *A. dorsale* was more susceptible to deltamethrin as 100% mortality was caused at only 1.25% of the field rate (Fig. 6.3.). *B. obtusum* (Fig. 6.2.) was relatively more tolerant of deltamethrin since 100% mortality was observed at 5% of the field rate. *B. lampros* and *D. atricapillus* were more tolerant of deltamethrin than dimethoate, mortality was 85 & 95% respectively at the field rate concentration (Figs. 6.1. & 6.4. respectively). Pirimicarb was the least toxic of the three

Figure 6.1. 72 h dose-response curves for *Bembidion lampros* exposed to residual deposits of deltamethrin, dimethoate and pirimicarb on glass in the laboratory.

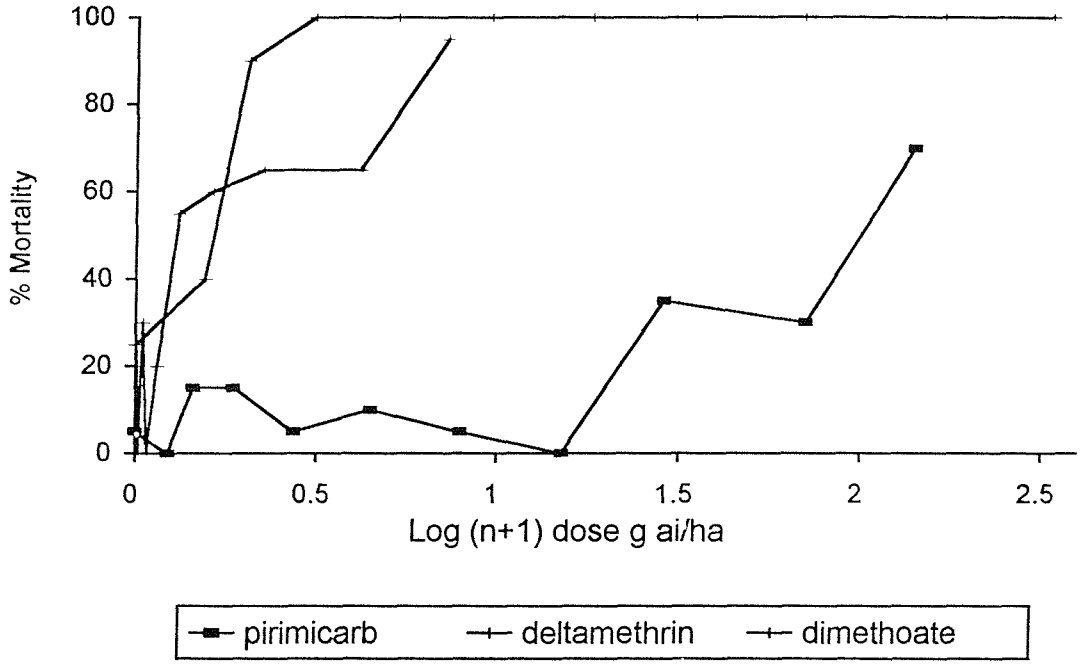


Figure 6.2. 72 h dose-response curves of *Bembidion obtusum* exposed to residual deposits of deltamethrin, dimethoate and pirimicarb on glass in the laboratory.

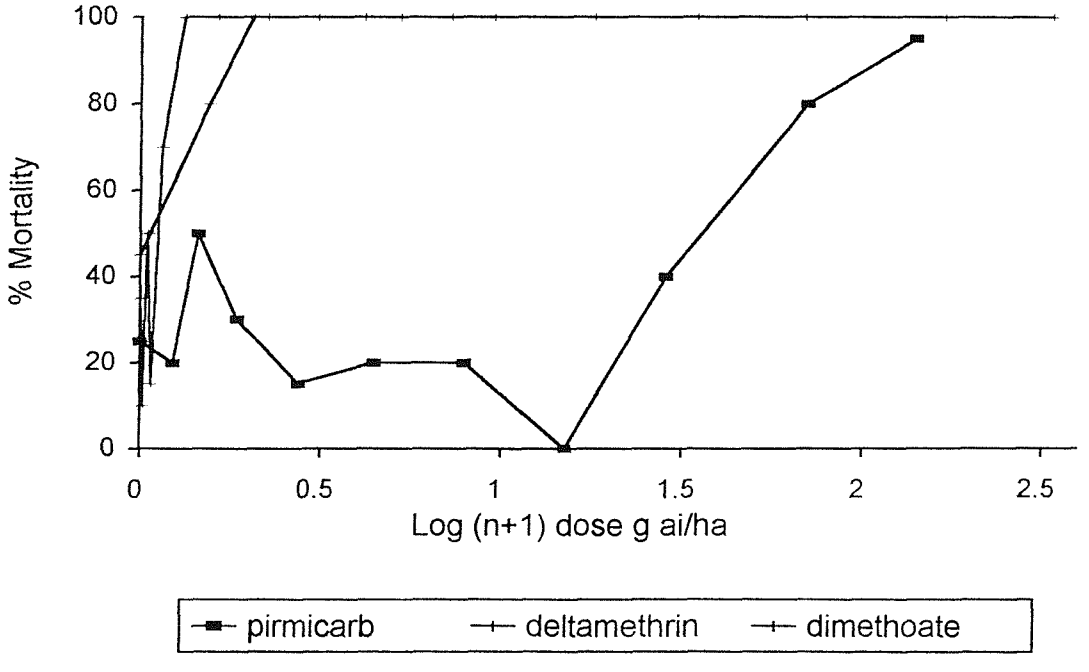


Figure 6.3. 72 h dose-response curves for *Agonum dorsale* exposed to residual deposits of deltamethrin, dimethoate and pirimicarb on glass in the laboratory.

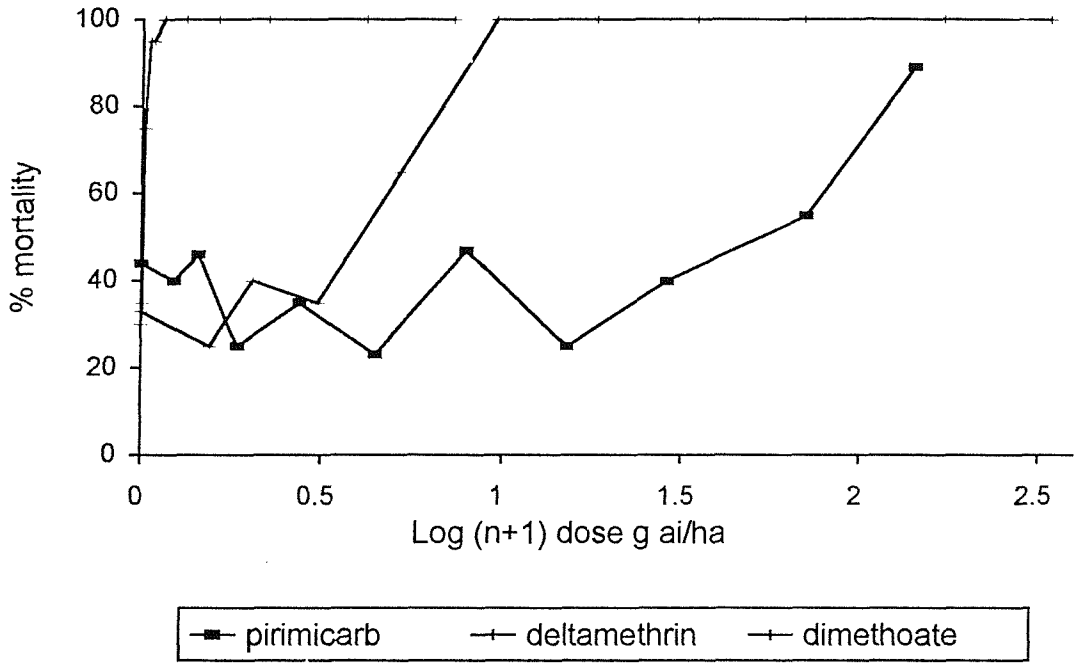


Figure 6.4. 72 h dose-response curves for *Demetrius atricapillus* exposed to residual deposits of deltamethrin, dimethoate and pirimicarb on glass in the laboratory.

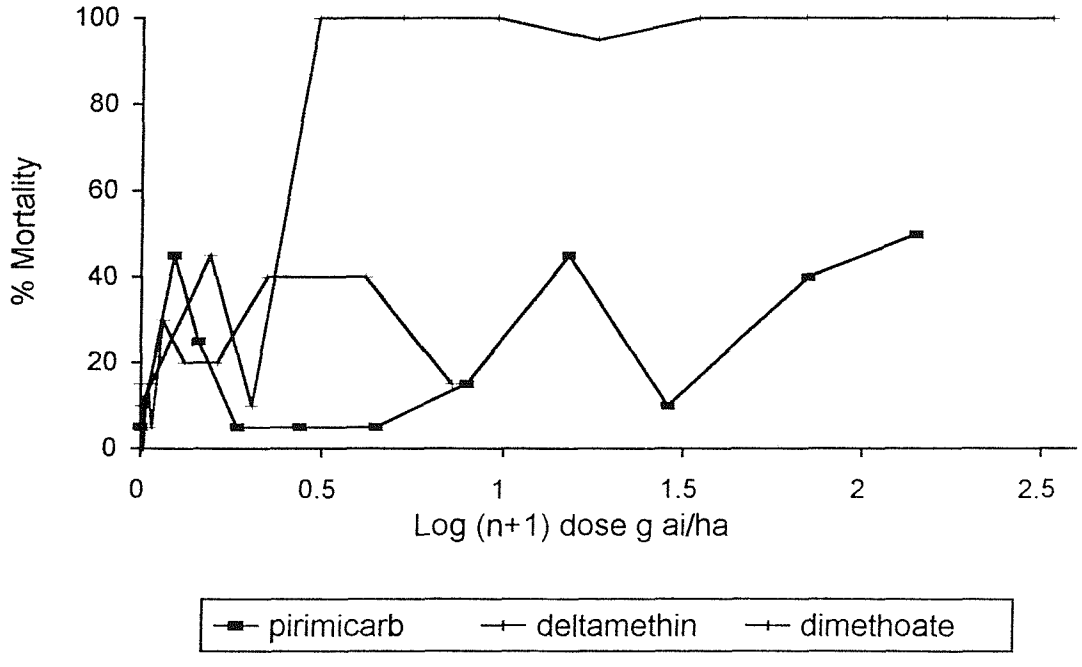


Table 6.3. Probit statistics for field rate application of the insecticides in the bioassays.

Insecticide	Probit slope	LT50 hrs (95% limits)	Heterogeneity ns = P> 0.05
deltamethrin	4.97	11.80 (3.48 - 67.89)	0.51 ns
dimethoate	1.24	71.95 (34.88 - 302.17)	2.17 ns

insecticides tested. Only at the field rate application was there any significant mortality of the beetles and unlike the other compounds mortality of the beetles never reached 100%.

From the dose-response curves for all the insecticides the toxicity of dimethoate declined steeply at lower concentrations, unlike deltamethrin and pirimicarb which both gave more graduated responses. The implications of these different dose response curves are discussed later (Chapter 8).

Exposure to residual deposits on soil and glass

Differences in responses shown by the beetles to the different insecticides were seen more clearly on soil than glass at the field rate application. For the pyrethroid the beetles were quickly knocked down but then began to recover after 4 h, for the organophosphorous product there was a gradual increase in toxic symptoms and no recovery afterwards. The insecticides were equally as toxic on glass and dimethoate was more toxic to *A. dorsale* than deltamethrin on soil at their respective field rate concentrations.

There was a difference in the toxicity of deltamethrin on soil and glass (Figs 6.5. & 6.6.). All beetles displayed symptoms of knockdown after 4 h exposure to deltamethrin on glass with no subsequent recovery. On soil, effects were only seen at field rate

Figure 6.5. Percentage of *A. dorsale* affected over time after exposure to residual deposits of deltamethrin on glass in the laboratory.

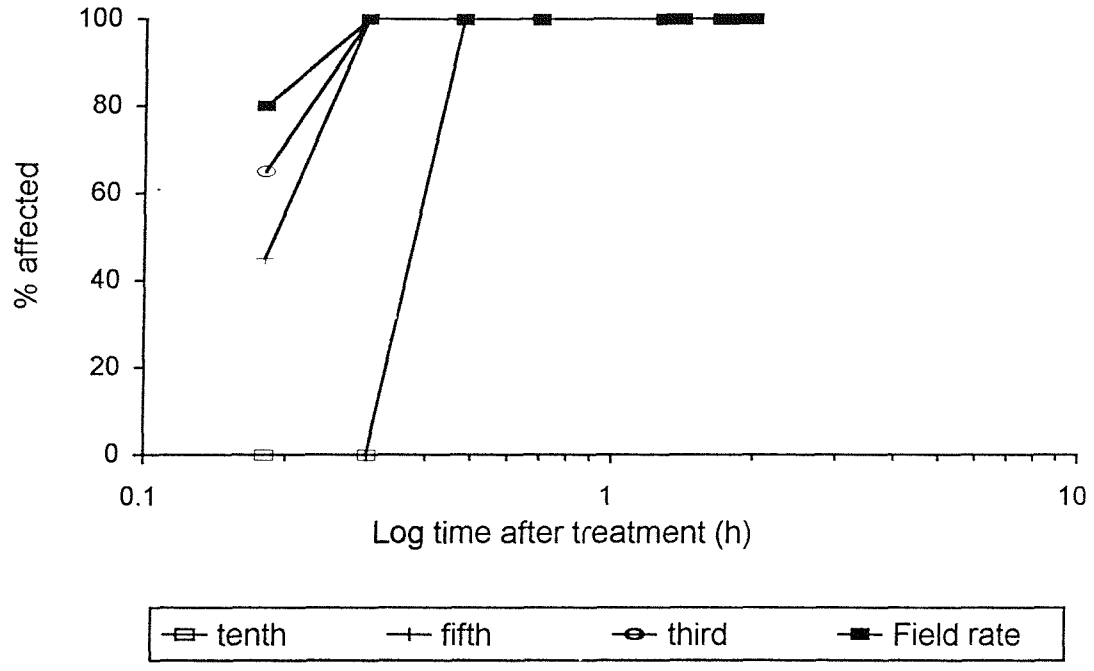


Figure 6.6. Percentage of *A. dorsale* affected over time after exposure to residual deposits of deltamethrin on soil.

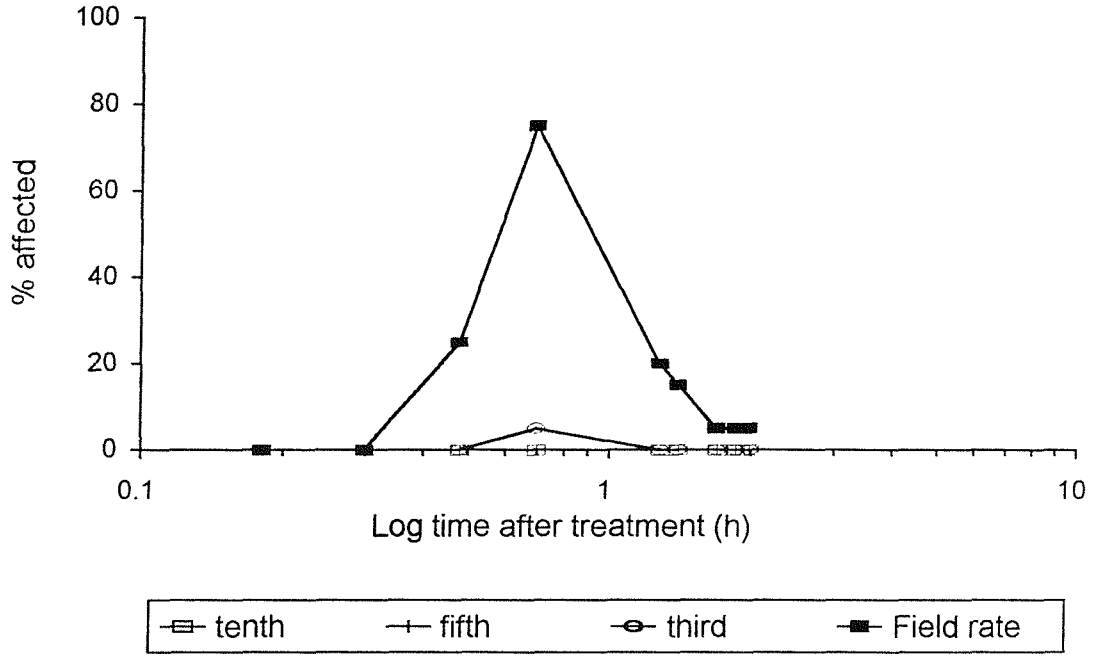


Figure 6.7. Percentage of *A. dorsale* affected over time after exposure to residual deposits of dimethoate on glass in the laboratory.

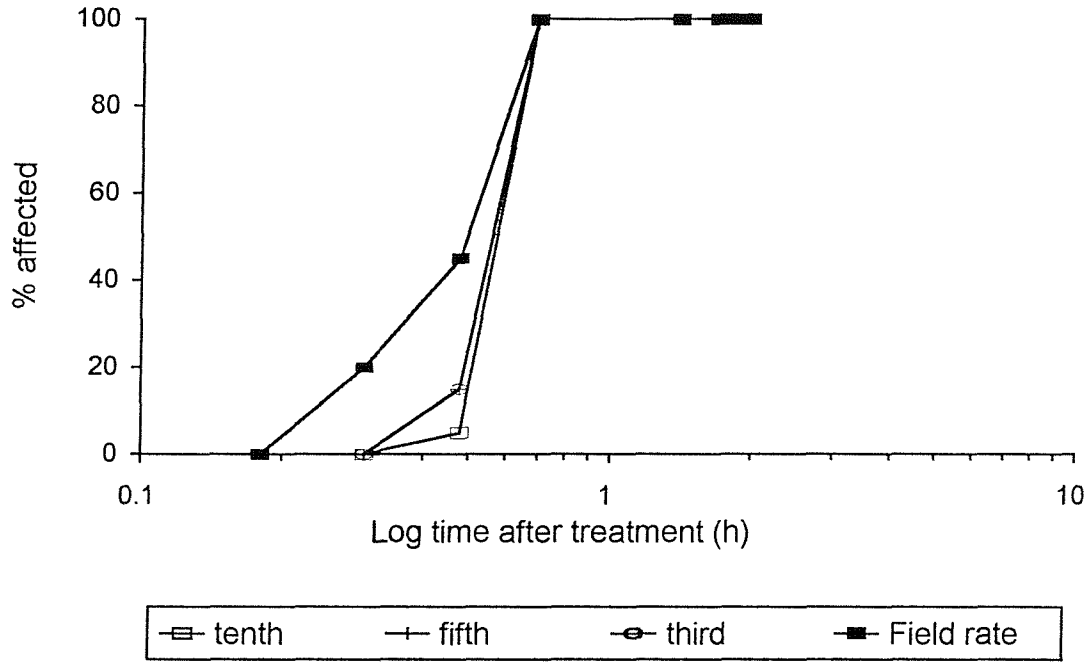
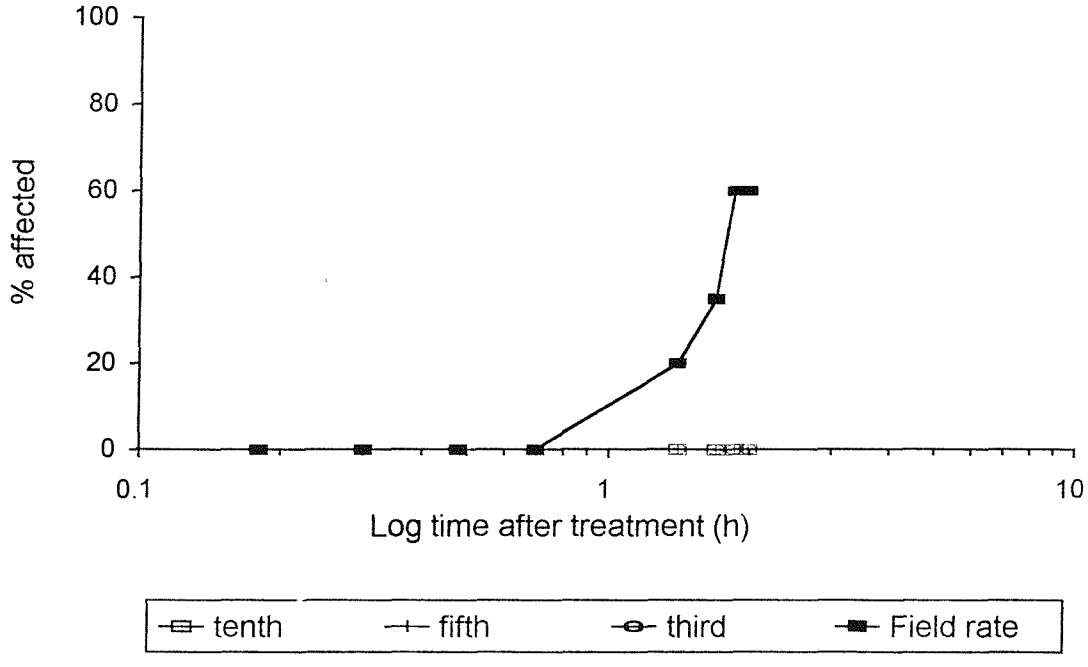


Figure 6.8. Percentage of A. dorsale affected over time after exposure to residual deposits of dimethoate on soil in the laboratory.



application, less than 30% of the beetles were knocked down after 4 h, although 75% were knocked down after 24 h there was only 5% mortality of the beetles after 24 h.

The toxicity of dimethoate on soil and glass also differed (Figs 6.7. & 6.8.). After 24 h exposure on glass all the beetles displayed symptoms of knockdown and none subsequently recovered. After 24 h exposure on soil no beetles displayed knockdown symptoms and only at the field rate was there any subsequent mortality.

Probit analysis of the time-response data for each of the concentrations of deltamethrin and dimethoate on both soil and glass was only possible for the field rate concentrations on soil (Table 6.3.). This was because the response of the beetles was too low in the soil bioassays and significant heterogeneity in the data of the glass bioassays. The time to knockdown of 50% of the beetles was shorter for deltamethrin than dimethoate.

DISCUSSION

According to the results of the first bioassay only pirimicarb of the insecticides tested would be relatively harmless to the test species, although there is evidence to suggest that this can be particularly toxic to beneficials under similar laboratory conditions (Brown, Lawton & Shires, 1983). The high carabid mortalities at very low doses of deltamethrin and dimethoate suggest that beneficials both within and outside of the crop area would suffer high mortality after spraying of these compounds if the results could be extrapolated directly to a field situation.

In the second experiment there was a clear difference in toxic effects between glass and soil deposits of both deltamethrin and dimethoate and also between the two compounds. The time response data demonstrates the response of the beetles to the two different insecticides in the soil bioassay. For deltamethrin in the soil bioassay, there was a knockdown effect observed followed by recovery. In the case of dimethoate there was a gradual increase in toxic symptoms with no recovery of affected beetles. These symptoms could not be elucidated in the glass bioassays because the beetles exposed

to both of the compounds were affected over a shorter period compared with those on soil. The difference in toxic effects between the glass and soil bioassays means that results from laboratory residual bioassays used for extrapolation studies should clearly be treated with caution and careful consideration of the choice of substrate made.

The differences in residual toxicity between the substrates is likely to result from changes in the bioavailability of the insecticides between the different surfaces. The availability of the insecticides will be a function of substrate properties and the physical chemistry of the insecticides applied (Salt & Ford, 1987; Unal & Jepson, 1991; Mansour *et al.*, 1992). Glass is a relative inert surface, therefore many of the physio-chemical factors that affect the breakdown of insecticides in a field situation are not present. Croft (1990) suggests that direct contact tests for predators may be useful for measuring 'intrinsic' differences of insecticide susceptibility, but residue tests would probably better estimate pesticide impact in the field. However mortality predictions for a field situation maybe overestimated because the test organism could be potentially exposed to many more times the amount of active ingredient then would generally be present. This was confirmed in the second experiment where the same doses were compared on different substrates. Mortality was greatly reduced on more realistic substrates. Indeed sorption processes and degradation by micro-organisms are known to restrict the toxicity of most pyrethroids on soil (Elliott *et al.*, 1978)

On soil the movement and degradation of the applied insecticide will depend upon three general factors; properties of the chemical, properties of the soil and the weather (Nicholls, 1991). In this experiment the most important factor will have been the degree of sorption on to the different surfaces. In general if the octanol-water partition coefficient is used as an indicator of lipophilicity, sorption to the soil of non-ionised organic compounds tends to increase with the lipophilicity of the chemical (Connell, 1994). Most organophosphorous compounds tend to sorb fairly strongly to soil surfaces and although dimethoate could be considered an exception to this rule, its high mobility within the soil profile and short half-life in the soil reduces its bioavailability on this surface (Racke, 1992). Increasing sorption is more correlated with soil organic matter content than any other soil property (Nicholls, 1991). The soil did have a high organic content (Table 6.2.) and the bioavailability of the compounds were quickly reduced

compared to glass. The important source of degradation is microbial biomass, to which fungi and bacteria are mainly attributed as being responsible for microbial degradation. It may be expected that increasing microbial biomass may make a compound less available. However, Jagers op Akkerhuis and Hamer (1992) investigating the bioavailability of ^{14}C deltamethrin to linyphid spiders, found that soil covered with fungi and moss increased deltamethrin bioavailabilty by a factor of approximately 100 compared to bare soil.

Rates of breakdown of an applied chemical are also affected by temperature. The rates of degradation in soil at different temperatures in soil cannot be predicted from the chemical structure or simple physiochemical properties of the compound (Briggs, 1981 & 1989). However in general degradation rates increase with increasing temperature and decrease rapidly as the soil dries (Nicholls, 1991). The temperature in the controlled temperature room would have been higher than average temperatures found in the field, therefore it would be expected that rates of loss of the applied compounds would have been higher and mortality of the test species could be under-estimated. This is particularly important for the pyrethroid insecticides, since it has already been shown that they are more toxic at lower temperatures (Chapter 5).

In the case of dimethoate, which unlike the pyrethroids has a high vapour pressure, volatilisation may be an important route of chemical loss and also mode of action. The significance of volatility depends on a number of factors; vapour pressure, solubility, adsorptive behaviour and persistence of the compound, and such environmental characteristics as temperature, moisture and air movements (Racke, 1992). Certainly for dimethoate, which could be considered to have an intermediate volatility of the organophosphorous compoundss, environmental factors greatly modulate the kinetics of volatilisation. Volatilisation rates from surfaces such as glass plates or leaves are often much greater than those from soil surfaces (Spencer *et al.*, 1969). It was noted in the laboratory tests the soil dried out over the course of the experiment. This may have affected the volatilisation of dimethoate, since as soil dries, a point is approached where only a monolayer of water remains on the soil surface. If the soil dries further, sorption of the chemical increases rapidly, resulting in less bioavailability with

time (Spencer *et al.*, 1969). However it is not known what role this played in the toxicity of the insecticide.

Soil texture and structure influence the dispersion of an applied insecticide and hence its bioavailability. Studies have shown that the type of soil influences the effects of pesticides on test organisms (Monke & Mayo, 1990; Heimbach *et al.*, 1992; Mansour *et al.*, 1992). Therefore extrapolation studies should not only consider the substrate but also the structure of the chosen substrate. Other authors (Wiles, 1992; Wiles & Jepson, 1994) have suggested that correction factors should be taken into account when considering the risk posed on different substrates.

Studies on the deposition rates of chemicals through a cereal crop have shown that there is a general stratification of the applied chemical through the crop canopy (Wiles, 1992; Cilgi & Jepson, 1992). Therefore it is likely that beneficial organisms present on bare ground will be at more risk from an insecticide application than those under a growing crop canopy. Cilgi and Jepson (1992) found on average 16% of the applied field rate on soil when they measured the deposition rate through the crop canopy. All of the carabid beetles tested in these experiments may be present in cereal fields during the summer, however the relative risk posed by the insecticides depends on which bioassay result is extrapolated. From the first experiment only pirimicarb would appear a safe insecticide to apply in terms of attempting to preserve beneficials. The high carabid mortalities seen for deltamethrin and dimethoate even at very low doses would suggest that 16% of the field rate depositing on the ground could result in high carabid mortality. Conversely the results from the second experiment would suggest that little risk is posed to more robust carabids like *A. dorsale* even at doses higher than a deposition rate of 16% of the applied field rate. *A. dorsale* would also be afforded further protection because it is nocturnal and would not be exposed to direct fallout when spraying takes place. Indeed the results of Wiles (1992) show that the LD_{50} values for predators exposed to residual deposits of deltamethrin are many more times that of the current recommended field-rate. Mortality could also be under-estimated because only one route of exposure has been considered in these experiments. For example *D. atricapillus* has been observed climbing the cereal plant (Vickerman and Sunderland, 1975) and may therefore not only be exposed to higher insecticide deposition levels

found on the foliage but also to direct fallout from spraying. Observable levels of mortality for this beetle on glass plates maybe repeated on the foliage of cereal crops because fresh residues on each substrate may be equally toxic (Wiles, 1992; Wiles & Jepson, 1994)

The results of these two bioassays correlate well with the results of other laboratory and field tests. Brown *et al.* (1983) used compounds from the same chemical classes (pyrethroid, organophosphorous and carbamate) as those used in the first experiment. They demonstrated that methyl parathion was overall the most toxic compound, cypermethrin was less toxic and pirimicarb the least toxic when a guild of natural enemies were exposed to residual deposits of the insecticides on a glass substrate. Unal and Jepson (1991) showed that deltamethrin was less toxic than dimethoate on the soil surface to *B. lampros*, in this chapter this was also true for *A. dorsale*. Despite being tested in artificial conditions the toxicity rankings of the three insecticides to the beetles are similar to those of field studies (Basedow *et al.*, 1985; Vickerman *et al.*, 1987 a & b; Cole *et al.*, 1986; Heimbach, 1991).

It would appear that exposure to residual deposits of an insecticide on the soil surface appears to pose little risk to ground dwelling beneficials. The risk can be further reduced by decreasing the dose applied. Not only then is potential contamination of the environment reduced and beneficials preserved, the aphid pest will also be adequately controlled. It is not known however what sub-lethal effects there may be on the preserved beneficials and one aspect of this is investigated in the next chapter.

CHAPTER 7

SUB-LETHAL EFFECTS OF DIFFERENT INSECTICIDE EXPOSURE ROUTES ON THE PREY CONSUMPTION RATES OF CARABID BEETLES.

INTRODUCTION

There are three main routes through which aphid predators may be exposed to insecticide applications; direct, residual and dietary (Croft & Brown, 1975; Jepson, 1989; Croft, 1990). It is unclear however, whether sub-lethal doses received by these routes of exposure affect the predatory potential of aphid natural enemies. This is particularly important in reduced dose-rate application strategies, since the main aim is to enhance biological control of the pest by the preservation of aphid predators (van Emden, 1987; Poehling, 1989). Subtle sub-lethal effects, may affect the biological control potential of the preserved predators by interfering in some way with their predatory behaviour. This might then lead to a reduction in biological control and possible undesirable side-effects such as pest resurgence.

Carabid beetles are known to be important ground-active predators in the cereal ecosystem (Potts & Vickerman, 1974; Edwards *et al.*, 1979; Chiverton, 1986, Wratten & Powell, 1991). Direct and residual contact with an insecticide may have long-term effects on predatory potential, not only because mortality will reduce numbers of predators in the field, but also because sub-lethal effects such as knockdown could lead to increased rates of mortality from predation or render the beetles unable to feed, thereby further reducing the predatory potential of any preserved beneficials. Dietary exposure to insecticides may be important in the short-term because a large number of insecticide-contaminated aphids may fall to the ground after spraying and could be consumed by predators (Dixon & McKinlay, 1992). Studies have shown that polyphagous predators will consume insecticide contaminated prey (Everts et al., 1991a) and that when under intoxication, consumption rates are reduced (Mullie & Everts, 1991).

This study was undertaken to examine pesticide side-effects on the predation of aphids using both full and reduced-rate applications. The beetles *Poecilus cupreus* (L.) (Coleoptera: Carabidae) and *Agonum dorsale* (Pontoppidan) (Coleoptera: Carabidae) were chosen as the test species. *P. cupreus* was chosen because it was readily available from a laboratory-bred culture. Individual beetles used were therefore of a uniform age, of known sex and disease free and would not have the inherent biological variability found in beetles taken from the field. *A. dorsale* was used when *P. cupreus* was not available in sufficient numbers. This beetle is present in the cereal ecosystem when insecticides are applied against cereal aphid pests and is known to be a significant predator of aphids (Griffiths, 1983). It has also been shown to suffer reduced population levels following pesticide applications (Greig-Smith *et al.*, 1992). Deltamethrin was chosen as the test insecticide. This compound is one of the most commonly applied insecticides to control aphids in cereals (Davies *et al.*, 1993) and its use in this and Chapters two and six allows an evaluation of the impact that reduced dose rates may have on ecological mechanisms that will affect subsequent pest control after an insecticide application.

MATERIALS AND METHODS

Summary

a) Topical and residual exposure to deltamethrin;

i) Test invertebrates
ii) Insecticide formulation
iii) Presentation of aphids for consumption
iv) Topical application of deltamethrin
v) Residual exposure to deltamethrin
vi) Assessment of aphid consumption

b) Dietary exposure to deltamethrin

i) Test invertebrates

ii) Treatment of aphids with deltamethrin

iii) Presentation of aphids for consumption

c) Statistical analysis

a) Topical and residual exposure to deltamethrin

i) Test invertebrates

Adult one month old *P. cupreus* were obtained from W. Neudorff GmbH KG, Emmerthal, Germany. The aphid species used as prey in the experiments was the bird-cherry aphid *Rhopalosiphum padi* (L.) (Homoptera: Aphididae). Individuals were cultured on barley (cv. Halcyon) in the insectary (18 - 22°C, 55-70% RH, 16:8 L:D). Prior to the experiments, all the *P. cupreus* were maintained in a controlled environment room (20 ± 2°C; 55-70%R.H.; 16:8 L:D). The beetles were kept in small plastic containers (10 x 15 x 27cm) containing a layer of moist potting compost (John Innes No. 2) and were fed on ground moist cat food ('Delicat', Quaker Latz).

Adult aphids were removed from the cultures and immediately freeze-killed by exposing them to temperatures of between -10 and -15°C for approximately 2 hours inside plastic Petri dishes. Care was taken to avoid freeze-drying the aphids. It was known that predators such as carabid beetles will consume dead prey items provided they have not decayed or desiccated excessively (Theile, 1977). Preliminary observations with the beetles confirmed this.

Twenty-four hours before the experiments were due to begin, all food was removed from the boxes containing the beetles. Fifteen beetles were used in each of the insecticide treatments making a total of 60 beetles in each of the two experiments.

Table 7.1. Doses of deltamethrin used in the topical and residual application experiments with *P. cupreus*.

Fraction of field rate	Topical (ng ai ul^{-1})	Residual (g ai ha^{-1})
FR	30	6.25
0.33	10	2.08
0.10	3	0.625

ii) Insecticide treatment of beetles

Commercially formulated deltamethrin (Hoechst Decis, 2.5 % E.C. w/v) was used as the test chemical, with distilled water as the diluent and the control treatment. Stock emulsions of deltamethrin were prepared before each experiment from which serial dilutions were made to obtain the required test concentrations. The highest concentration chosen was 30 ng a.i. ul^{-1}, this approximates to the recommended field concentration of deltamethrin (31.25 ng a.i. ul^{-1}, i.e. 6.25 g a.i./ha in 200 litres water) for use as a summer cereal aphicide in the UK (Anon, 1993). Reduced doses applied to the test invertebrates were serial dilutions of the recommended field rate emulsion (see Table 7.1.).

iii) Presentation of aphids for consumption by the beetles

Twenty-five aphids were stuck onto a filter paper (3 cm diameter) using a flour and water mixture, in a 5 x 5 grid pattern. During the first 24 hours in the residual exposure experiment the aphids were presented to the beetles suspended above the soil on a novel bait card (see Fig. 7.1) in the same grid pattern as the normal filter paper grids. The bait card consisted of a piece of cardboard (3 x 3 cm) supported at the corners by dress making pins. Onto the cardboard was attached the piece of filter paper containing the grid of aphids. In the arrangement, the pins supported the grid of aphids approximately 1 cm above the surface of the soil allowing the beetles to feed on the aphids but minimising the possibility of accidental contamination of the aphids by the beetle when walking across a normal filter paper. The bait cards were placed on to the soil in the containers at the same time as the beetles. For the duration of the experiment

the beetles were kept in an environmentally controlled room (20 ± 2°C; 55-70%R.H.; 16:8 L:D).

iv) Topical Application of deltamethrin to the beetles

The beetles were gently held by their back legs after being anaesthetised using a CO_2 supply for 10 seconds. Topical applications were made, to the junction between the thorax and the abdomen with a 250-µl Hamilton gas-tight syringe mounted in a Burkhard hand microapplicator (Burkhard Manufacturing Co. Ltd). The syringe was calibrated to deliver drops of 1-µl of each of the deltamethrin dilutions. Control beetles were dosed with a 1-µl droplet of distilled water. Care was taken to avoid droplet run-off from the beetles and the droplets were allowed to dry before the beetles were moved.

v) Residual exposure to deltamethrin of the beetles

A 2cm layer of sieved (2 mm sieve) agricultural soil, analysed according to the methods described by Allen (1989) (cited by Wiles (1992) (Table 7.2.), was gently compacted in the bottom of a plastic pot (9cm diameter x 5cm high). The soil surface was treated using a Potter laboratory spray tower which had previously been calibrated to deliver a spray volume equivalent to 200 l/ha. The dilutions of deltamethrin used were the same as those used in the topical dosing experiment. Distilled water was again used to treat control pots (Table 7.1.). The soil was allowed to dry for approximately 1 hour before the beetles were introduced to it. The beetles were exposed to the residual deposits of deltamethrin for 24 hours they were then removed and placed in clean pots containing a 2cm layer of clean untreated potting compost (John Innes No. 2).

vi) Assessment of aphid consumption

Immediately after treatment and for the following three days the beetles were presented with fresh bait cards every 24 h. The number of aphids eaten during the previous 24 h was recorded and the old bait card removed. The response of the beetles was also recorded, i.e. moving as normal, knocked down (with antennae, mandibles or

Table 7.2. Analysis of soil used in experiment investigating the effect of residual toxicity of deltamethrin on the predation of aphids by *P. cupreus*.

Soil Profile	Analysis %
Clay	3.2
Silt	10.9
Sand	59.4
Gravel	13.3
Organic matter	12.1
pH (H_2O)	6.7
Moisture	20.1

legs moving but unable to right themselves permanently) or dead (showing no response to stimulation) when the bait cards were removed.

b) Dietary exposure to deltamethrin;

i) Test invertebrates

Adult *A. dorsale* were captured between December 1992 and February 1993 in cereal fields and field margins at Leckford, near Stockbridge, Hampshire, by surface searching using a hand-held aspirator. After collection the beetles were kept in a dark cold room (4°C) in small plastic containers (10 x 15 x 27cm) containing a layer of moist potting compost (John Innes No. 2) and were fed on ground moist cat food ('Delicat', Quaker, Latz), until 72 hours before the experiment was due to begin. They were then moved to a controlled environment room (20 ±2°C; 55-70%R.H.; 16:8L:D). Twenty-four hours before the experiment was due to begin all food was removed from the boxes containing the beetles.

ii) Treatment of aphids with deltamethrin

Adult aphids were removed from the cultures and immediately freeze-killed by exposing them to temperatures of between -10 and -15°C for approximately 2 hours inside plastic Petri dishes. The aphids were then placed in a glass Petri dish (9 cm

diameter) and sprayed with a diluted deltamethrin solution (30 ng a.i. ul^{-1}) using a Potter laboratory spray tower that had previously been calibrated to deliver at 200 l/ha. Control aphids were treated with distilled water.

iii) Aphid consumption rates by the beetles

After the aphids had been sprayed under the Potter tower twenty five were attached to a piece of filter paper (3 cm diameter) using a flour and water mixture. Care was taken to minimise the amount of handling of the aphids at this stage. The aphids were placed in plastic pots (9cm diameter x 5cm high) containing a 2 cm layer of moist John Innes No. 2 compost and a single beetle. The Beetles were provided with contaminated aphids for either 24, 48 or 72 hours. A total of 40 beetles were used in the experiment, ten beetles in each treatment. After the period of providing the beetles with contaminated aphids had ceased, they were fed aphids that had been sprayed with distilled water. Controls were provided with distilled water treated aphids throughout. Fresh treated and untreated aphids were provided every 24 h and the number of aphids eaten by each beetle was recorded for the following 24 hour period. Any knockdown, regurgitation or mortality affects on the beetles was also recorded.

c) Statistical Analysis

The log (x+1) transformed aphid consumption data were analysed for differences between treatments over the different days by one-way ANOVA. Tukey's HSD was used to determine the significance of any differences between individual means in significant heterogeneous groups using the SPSS statistical package (SPSS Corporation, v 6.0).

RESULTS

i) Topically dosed beetles

During the first 24 hours after treatment the field-rate treated beetles consumed the highest numbers of aphids per beetle, although there were no significant differences

Table 7.3. Mean number of aphids eaten by *P. cupreus* after topical treatment with deltamethrin.

Time (hours)	Treament (fraction of field rate)	Mean number of aphids eaten per beetle (± SE)	F ratio (d.f.)	Tukey HSD*
24	Control	4.0 (± 2.8)	1.99 (58)	a
	0.10	2.5 (± 2.5)		a
	0.33	5.3 (± 3.7)		a
	1	8.9 (± 4.0)		a
48	Control	3.8 (± 2.6)	1.03 (58)	a
	0.10	4.4 (± 3.6)		a
	0.33	7.6 (± 3.8)		a
	1	7.8 (± 3.8)		a
72	Control	3.7 (± 2.6)	1.38 (58)	a
	0.10	2.7 (± 2.2)		a
	0.33	4.5 (± 2.1)		a
	1	7.0 (± 3.5)		a
96	Control	2.0 (± 0.6)	0.92 (58)	a
	0.10	4.9 (± 3.5)		a
	0.33	3.0 (± 1.9)		a
	1	4.5 (± 2.9)		a
120	Control	2.9 (± 2.2)	0.53 (58)	a
	0.10	5.8 (± 3.9)		a
	0.33	4.9 (± 3.6)		a
	1	5.5 (± 3.9)		a

* different letters indicate significant differences ($p<0.05$) in number of aphids eaten.

Table 7.4. Mean number of aphids eaten by *P. cupreus* when exposed to residual deposits of deltamethrin for 24 h.

Time (hours)	Treament (fraction of field rate)	Mean number of aphids eaten (± SE)	F ratio (d.f.)	Tukey HSD*
24	Control	23.5 (± 0.5)	11.26 (58)	a
	0.10	11.9 (± 5.3)		b
	0.33	7.3 (± 5.5)		b
	1	4.4 (± 3.3)		b
48	Control	20.3 (± 3.5)	2.57 (58)	a
	0.10	23 (± 0.5)		a
	0.33	23.1 (± 0.9)		a
	1	19.2 (± 3.2)		a
72	Control	21.6 (± 3.0)	0.51(58)	a
	0.10	23.3 (± 0.4)		a
	0.33	22.1 (± 3.0)		a
	1	20.7 (± 3.0)		a
96	Control	23.9 (± 0.2)	1.32 (58)	a
	0.10	23.2 (± 1.0)		a
	0.33	22.1 (± 2.2)		a
	1	22 (± 1.6)		a
120	Control	22.1 (± 3.0)	0.23 (58)	a
	0.10	21.5 (± 3.4)		a
	0.33	21.7 (± 2.9)		a
	1	22.1 (± 1.6)		a

* different letters indicate significant differences ($p<0.05$) in number of aphids eaten.

Table 7.5. Mean number of aphids eaten by *A. dorsale* when presented with deltamethrin treated and untreated aphids (see text for details).

Time (hours)	Hours exposed to treated aphids	Mean number of aphids eaten (± SE)	F ratio	Tukey HSD*
24	0	14.7 (± 0.8)	13.63 (38)	a
	24	7.5 (± 1.0)		b
	48	8.5 (± 0.8)		b
	72	8.1 (± 1.0)		b
48	0	10.2 (± 1.5)	12.10 (38)	a
	24	13.4 (± 1.9)		a
	48	4.4 (± 0.8)		b
	72	3.8 (± 0.9)		b
72	0	12.6 (± 1.4)	10.71(38)	a
	24	13.8 (± 1.2)		a
	48	13.4 (± 1.3)		a
	72	5.0 (± 1.2)		b
96	0	12.7 (± 1.8)	0.66 (38)	a
	24	13.1 (± 1.5)		a
	48	15 (± 1.3)		a
	72	15.3 (± 1.9)		a

* different letters indicate significant differences ($p<0.05$) in number of aphids eaten.

between treatments (Table 7.3.). None of the beetles died during the experiment and none were observed to be suffering symptoms of knockdown. From comparison of Tables 7.3. and 7.4. all topically treated beetles, including the controls, consumed fewer aphids than those exposed to residual deposits of the insecticide.

ii) Residually exposed beetles

The mean number of aphids eaten was related to the dose applied (Table 7.4.). All of the beetles exposed to the insecticide field rate exhibited symptoms of knockdown but recovered when placed on clean soil. The beetles exposed to reduced rates of insecticide did not show any symptoms of knockdown. The number of aphids eaten by the control and treated beetles were significantly different from each other during the first 24 h (Table 7.4.), although there were no differences between the treatments (Table 7.4.). After the beetles had been moved to clean soil subsequent aphid consumption did not vary significantly.

iii) Dietary exposure of the beetles

When the beetles were presented with contaminated aphids they consumed significantly fewer aphids than the controls (Table 7.5.). Aphid consumption returned to that of the controls within 24 h of the contaminated aphids being replaced by uncontaminated ones (Table 7.5).

DISCUSSION

The results of these experiments suggest that the three routes of exposure have different effects on laboratory aphid consumption by beneficial organisms. Although they were handled in exactly the same way before treatment, the *P. cupreus* control beetles in the topical experiment consumed fewer aphids than those in the residual experiment. This may have been due to anaesthetising the beetles or a difference in their nutritional state before the start of the experiment. Beetles in both the topical and residual experiment were starved for 24 h before treatment but how long they had gone without

food previously was not known. The length of time without food has been shown to affect aphid consumption i.e. Wiles & Jepson (1993). There was no suggestion that deltamethrin treated aphids had any repellent effects on *A. dorsale.* All the beetles presented with the contaminated aphids consumed at least one aphid.

Effects on prey consumption were observed when *P. cupreus* were exposed to residual deposits of deltamethrin at doses less than those required to kill them (Table 7.4.). The mean number of aphids was related to the dose applied, although there were no significant differences between the deltamethrin treatments. The reduction of aphids consumed in the field rate treated beetles could be attributed to the beetles being knocked down and unable to feed. Those exposed to 0.33 and 0.10 field rates did not show any symptoms of knockdown, so the reason for the reduction in feeding is attributable to sub-lethal poisoning. When the beetles were moved to uncontaminated soil their feeding returned to the rates of the controls (Table 7.4.). Dempster (1968) showed exposure to residual deposits of DDT reduced the feeding rate of *Harpalus rufipes* (Coleoptera: Carabidae) Degeer. Their feeding rate returned to control levels when they were removed from the contaminated soil.

Previous authors have shown that dietary uptake of an insecticide causes mortality in predators (Azab *et al.*, 1971; Brust *et al.*, 1986; Wiles & Jepson, 1993). No *A. dorsale* died after eating contaminated aphids, but the presence of deltamethrin did affect feeding. Once the contaminated aphids were replaced with uncontaminated ones prey consumption returned to that of the controls. This suggests that after eating contaminated prey no long term sub-lethal effects on predation would occur once this prey was removed. Wiles & Jepson (1993) showed that the consumption of untreated aphids in *Nebria brevicollis* (F.), was reduced for a number of days after being fed deltamethrin contaminated aphids. This suggested a short-term reduction in the predatory efficiency of beetles consuming contaminated prey. The regurgitation response observed in the study of Wiles & Jepson (1993) after eating contaminated aphids was not seen in this study. This may have prevented mortality, since a loss of water, possibly compounded by this response, has been implicated in pyrethroid poisoning (Greenwood *et al.*, 1990; Broderick *et al.*, 1991).

When extrapolated to a field situation the sub-lethal effects observed in the residual and dietary experiments would result in a short-term reduction in the predatory efficiency of predators exposed to an insecticide application. These effects can be reduced using lower application rates. However several questions need to be addressed in order to evaluate the importance of these routes in the field. The toxicity of all three routes in combination needs to be evaluated before better predictions can be made of effects in the field.

Previous work (i.e. Chapter 6, Poehling, 1989; Taye & Jepson, 1991; Wiles, 1992) has shown that lower application rates reduce the mortality of predators. Predators are known to be important in controlling pest outbreaks (Burn, 1992; Vickerman, 1992), but can only do so by maintenance of their predatory potential. This is achieved by reducing their mortality and subsequent sub-lethal affects after an insecticide application. Reduced dose applications have been shown to achieve both in the laboratory, but quantification of affects on predation are difficult to identify in the field (Burn, 1992). Field studies over a single season using baited cards have shown reductions in predation after conventional pesticide applications (Speight & Lawton, 1976; Sotherton et al., 1988; Burn, 1987; Mauremotoo, 1991). But longer term studies, where predation from bait cards has increased after conventional pesticide applications, indicate the caution needed when attempting to predict possible levels of predation from knowledge of predator numbers in a cereal field (Burn, 1992).

Sub-lethal effects on predation in the laboratory were only shown to occur in the presence of an insecticide and aphid consumption returned to control levels when the contaminant was removed. Questions therefore have to answered about how long an insecticide remains toxic by residual and dietary exposure. A less persistent or toxic compound to beneficial predators may reduce the risk of residual exposure, whilst dietary effects would only be there so long as contaminated aphids remained suitable for consumption before they desiccated. It is also not known whether predators show any preference between uncontaminated and contaminated prey if both are available.

CHAPTER 8

GENERAL DISCUSSION

One aim of the thesis was to provide practical advice on the use of reduced dose-rate insecticide applications. This chapter therefore discusses the results that have been generated in relation to the effects of reduced dose-rates on aphid pests and aphid antagonists in cereal fields. Possible directions for future research are also outlined.

Control of aphid pests using reduced dose-rate insecticides;

Aphicides are applied to prevent economic losses as a result of aphids damaging the crop. In order for farmers to apply reduced dose-rates of insecticides it must be shown that reduced rates give equally as good control as full rates and savings made from chemical usage are not offset by reductions in yield and price for the crop. Economic returns will depend on a) timing of the spray application in relation to the development of the aphid population, b) the efficacy of the insecticide dose-rate, c) the cost of the insecticide and its application and d) the value of the crop (Mann *et al.*, 1991). In the UK, the Ministry of Agriculture, Fisheries and Food (MAFF) control threshold for *Sitobion avenae* (Homoptera: Aphididae) is based on applying insecticides when 66% of ears are infested during flowering and up to the milky ripe stages (Anon, 1988). Therefore the UK threshold only takes into account a) and does not consider b), c) or d) (George & Gair, 1975; Oakley *et al.*, 1988; Mann *et al.*, 1991).

When such a fixed threshold is used, aphid control will be dependent on population development. The necessity of a late treatment may have no economic benefit over not spraying, however a later treatment reduces the chance of an aphid population recovering to economic damaging levels (Mann & Wratten, 1988, Poehling, 1989). Late developing aphid populations in the field in Chapter 2 were equally as well controlled using reduced dose-rates compared to a conventional field rate application by deltamethrin, with no resurgence of any treated population. Control achieved using reduced dose-rates of pirimicarb highlighted the caution needed when selecting doses to maintain control equivalent to that of a full field rate application.

Inefficiency of control by the insecticide applied is important to consider when selecting a dose to apply. The predictions of percentage reduction in Chapter 2 gave an indication of the efficacy of doses applied in the field. It therefore follows that a high aphid population will leave a higher residual population after application. This may be less important if the aphid population is low since actual differences in aphid numbers between reduced rate and full rate applications would be very small. However, where the rate chosen gives considerably poorer control than a full rate application, an aphid population may build up again making a second spray necessary and nullifying initial savings made from spraying at a reduced rate,

There is no indication that chemical costs are going to increase substantially in the near future (Anon, 1994), more likely, revenues from selling grain will decrease, especially if intervention prices continue to fall (Wratten, 1992). In these circumstances farmers may consider cutting rates to increase profits, however, if adequate control of the pest is not achieved additional treatments would be required. Farmers may then consider a return to prophylactic tank spraying of a fungicide and broad-spectrum insecticide to reduce costs further and keep pests in check rather than clearing a field completely. This type of spray regime could have serious consequences for the development of resistance and would go against the dogma of applying at reduced dose-rates as it may upset interactions between insect pests and predators depending on the chemical applied and its timing.

To allow for variations in spraying economics, control thresholds must be dynamic rather than static (Mann *et al.*, 1991). Threshold models that allow for such variation, i.e. Reinink, 1986; Mann & Wratten, 1988, do not include provision for changes in insecticide efficacy when reduced dose-rates are applied. Using simple models that predict the efficacy of a reduced dose-rate application in the field would allow the actual dose applied, reductions in aphid numbers and effects on yield to be entered into control models to more accurately predict the savings a farmer would make from applying a reduced dose-rate application.

The experiments investigating effects on measurements of individual aphids growth and development are useful for considering population development of aphids

left in the field after spraying. The measurements underline aphid population growth, and could be entered into simulation models i.e. Watt *et al.*, 1984 to predict if aphids would reach economic damaging levels following a reduced dose-rate application.

The sporadic nature of summer cereal aphids reaching damaging levels and the crop rotations of a modern farm make long-term interactions between aphid pests and their antagonists difficult to develop and maintain. Often summer aphicide applications are the only way to control cereal aphids therefore. However the majority of applications to control aphids are applied before December of the year prior to harvest (Davies *et al.*, 1993). Can a farmer make savings on autumn applications of aphicides? Autumn applications are for the control of BYDV vectors, therefore the consequences of leaving a residual population of BYDV infected aphids may have a greater effect on yield at harvest than leaving a corresponding number of aphids after a summer aphicide application. Although it needs to be fully explored in a field situation, the relationships that pyrethroids have with temperature and reduced dose-rate control compared with full field rate application would suggest that autumn rates could be cut with no significant reduction in vector control.

Cereals have a high share of total pesticide costs in most European countries. In the UK pesticide usage on cereals accounts for some 60% of national sales (Brouver *et al.*, 1994). Under the Fifth Environmental Action Programme, a significant reduction of pesticide use per unit of land is required by the year 2000 (Beaumont, 1993). Provision of better information at the individual farm level will contribute to a reduction in pesticide usage. Farming practice aimed at full control of weeds, pests and diseases needs to move towards more rational practices. Reducing pesticide rates to levels that still provide equal control as full rates is just one means of achieving this.

Effects reduced dose-rate insecticides on aphid antagonists;

The aim of using reduced dose-rate insecticides is to exploit theoretical differences in the dose-response relationships of insect herbivores and their natural enemies (van Emden, 1987; Croft, 1990). By comparing the 24 h dose-response curves

generated in Chapter 2 for *Rhopalosiphum padi* (Homoptera: Aphididae) and in Chapter 6 for *Agonum dorsale* (Coleoptera: Carabidae) these differences do appear to exist.

The best example is that of pirimicarb (Fig. 8.3.), where only at the field rate application is any *A. dorsale* mortality observed whilst aphid mortality is 100% at much lower doses. Differences in the dose-response curves of *R. padi* and *A. dorsale* are also observed for dimethoate (Fig. 8.1.) although the area of selectivity between the pest and predator are very much smaller than for pirimicarb. Deltamethrin shows a similar toxicity to both *R. padi* and *A. dorsale* (Fig. 8.2.). Although there appears to be no selectivity between pest and predator for deltamethrin, a lack of effects on natural surfaces later in Chapter 6, indicates the need to select appropriate substrates when testing insecticide effects on predators. The most efficient and effective way of assessing the effect of insecticides on beneficial predators would be to adopt a sequential testing regime, using the route of exposure that predators are most likely to be exposed to an applied insecticide. Such sequential assessments have been suggested by Barrett (1992) and Carter (1992).

In this scenario (Fig. 8.4.), Level 1 would be a laboratory assessment of the insecticides toxicity at various doses. The test substance would be applied to an inert surface i.e. glass, providing conditions of maximum exposure (Barrett, 1992). Depending on the results generated no further testing need be done if the product was shown not to be 'harmful' to the beneficial predator, i.e. mortality was below an acceptable level of mortality. At Level 2, further laboratory studies using a more realistic substrate could be attempted, i.e. similar to the glass/soil comparisons of Chapter 6. Alternately a semi-field study could be carried out of similar methodology to Chapter 2, i.e. using an application technique similar to that used in the field but reducing the environmental control of a laboratory experiment. At Level 3, a field trial could be conducted where the test design would answer the question of what effect reduced dose rate insecticides would have on beneficial predators in a field situation.

From the laboratory studies reduced dose-rate applications reduce the potential for adverse side-effects on beneficial predators. Direct mortality may be reduced from the application of reduced dose-rate insecticides, more subtle sub-lethal effects have to

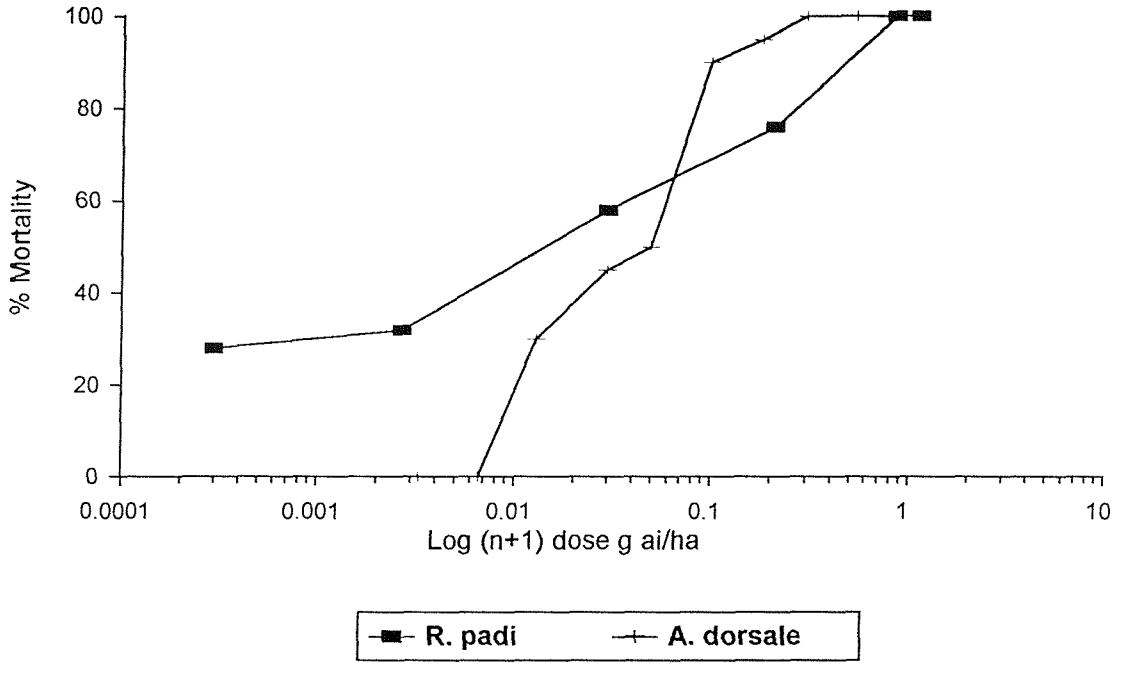


Figure 8.1. Dose-response curves for *R. padi* and *A. dorsale* treated with dimethoate in Chapters 2 and 6 respectively.

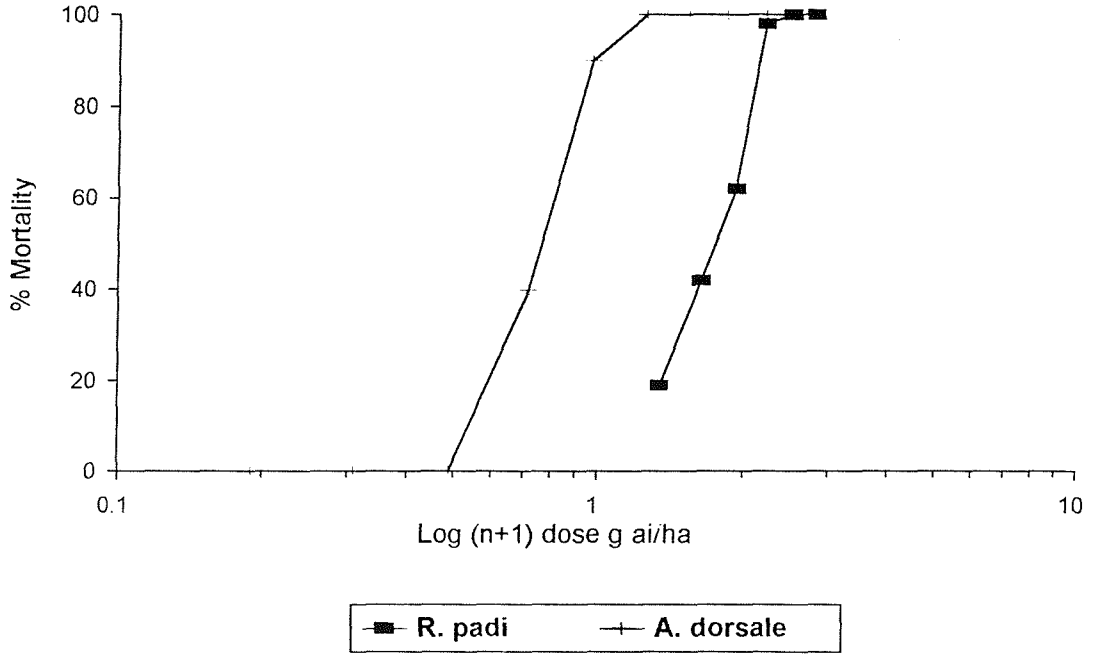


Figure 8.2. Dose-response curves for *R. padi* and *A. dorsale* treated with deltamethrin in Chapters 2 and 6 respectively.

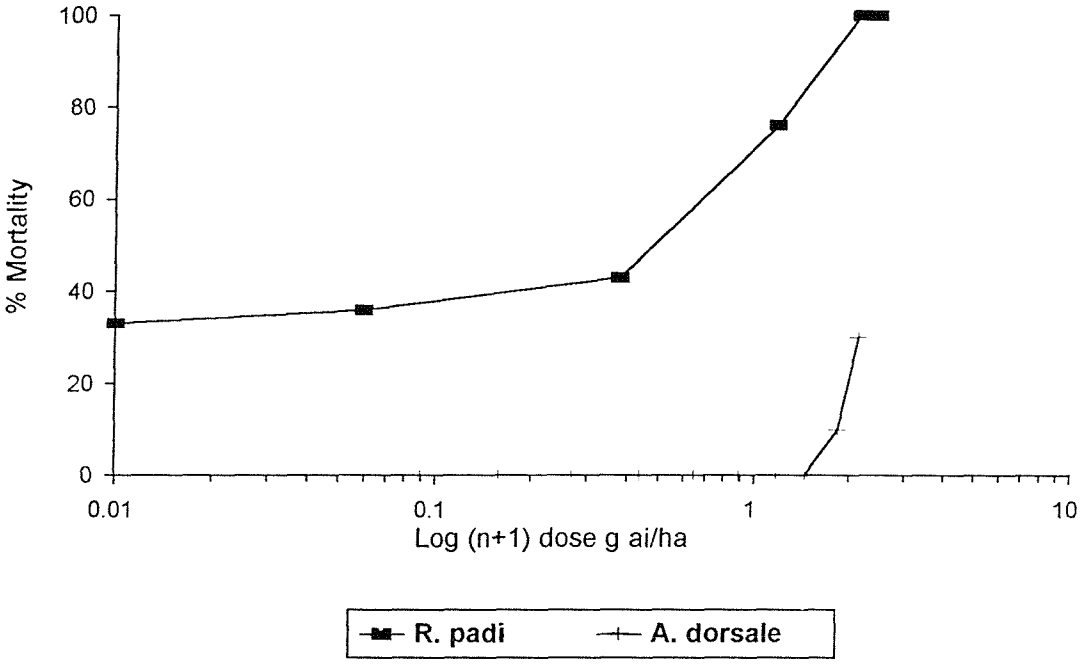


Figure 8.3. Dose response curves for *R. padi* and *A. dorsale* treated with pirimicarb in Chapters 2 and 6 respectively.

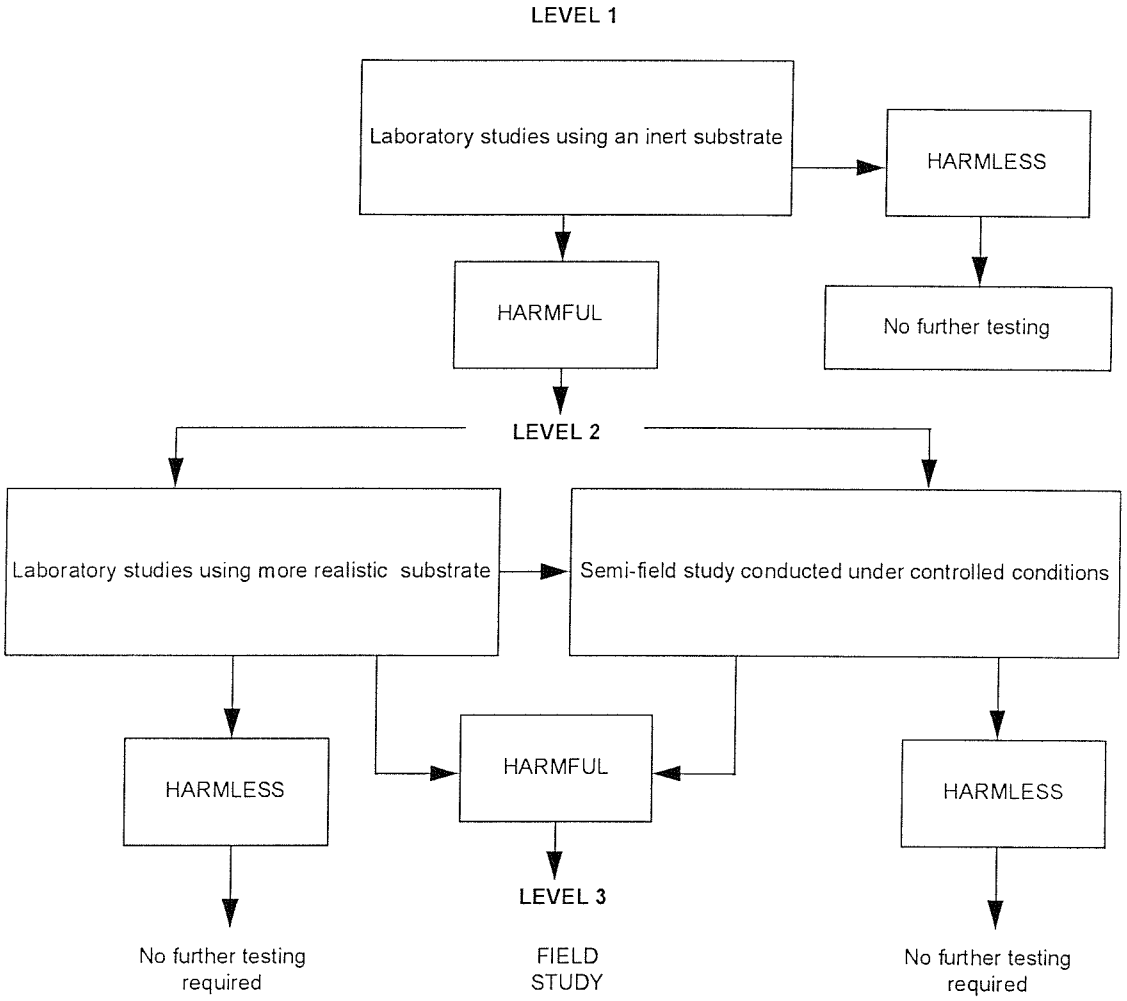


Figure 8.4. Simplified flow chart for adopting a sequential testing regime for beneficial predators (after Barrett (1992) and Carter (1992)).

be considered. Polyphagous and aphid specific predators are important in regulating aphid numbers (Wratten & Powell, 1991). When aphid numbers are low, natural enemies may be able to control populations, if an aphicide application becomes necessary a reduced dose-rate application may alter the pest:predator ratio in favour of the predator, but sub-lethal effects that reduce the predatory potential of natural enemies could significantly affect subsequent pest control. The result of Chapter 7 suggest that these would also be reduced from the application of reduced dose-rates however.

From an economic and ecological viewpoints, insecticides should only be used when necessary and in a way that minimises side-effects on, and maximises potential control by aphid antagonists. Reducing dose-rates appears to give equal control of pests as a full rate application and could enhance natural control in the field.

Future work

1. Improving forecasts of aphid control

Chapter 2 highlighted a 'crude' relationship between laboratory generated data and extrapolation to a field situation, this needs to be further investigated to improve this relationship. This could be achieved through a mechanistic approach of studying all the factors that contribute i.e. investigating, the intrinsic toxicity of topical, residual, fumigant and systemic modes of could be investigated as follows;

Depending on the insecticide being used improving knowledge about the following routes of exposure is required, i) topical exposure could be investigated using the Potter tower or for greater accuracy a microapplicator to generate dose response data for use in extrapolating to the field, ii) residual exposure could be investigated using glass or a more natural substrate i.e. leaf from wheat plant to generate dose-response data, iii) fumigant toxicity of an insecticide could be investigated using the apparatus shown in Fig 8.4., and iv) systemic exposure using a soil applied drench to whole plants or individual ears or leaves in a suitable medium. If the same doses were applied for all exposure routes the mortality observed would give an indication of the

importance of that route of exposure and the model could be adjusted to include all the relevant routes of exposure.

A model from dose-response data could be generated by using 'extended laboratory studies' and then extrapolating the data for a field situation. Ears and flag leaves from individual wheat plants could be removed together from a plant and placed in suitable containers. After infesting with known numbers of aphids they would be sprayed individually using a Potter tower at rates to generate a more crude dose-response curve. the dose-response data would then be used to generate the data for extrapolation to a field situation.

2. Improving deposition knowledge

To improve the relationship between laboratory and field generated data there is a need to improve our current knowledge of pesticide deposition on the plant. Rather than use data on ear deposition, data on deposition on aphids on different parts of the plant could be generated. Small glass marbles or similar, the size of an adult aphid could be attached to different parts of a wheat plant and the fluorescent tracer technique of Cilgi & Jepson (1992) used to study the dose impinging upon aphid in the field. Carefully removing the marbles from the plant using forceps and washing the marble would produce a distribution on aphids through the canopy.

The fluorescent tracer technique was applied at a full field rate and using it assumes a direct extrapolation between full field rate and reduced dose-rate applications. The technique should be used to confirm that such an extrapolation is correct and to what dosage reduction this is true.

3. Influence of temperature on toxicity

The study showed that temperature did influence the toxicity of an insecticide in the laboratory, this needs to be verified under more realistic conditions. This is difficult to do in the field but could be done in large environment controlled chambers. Studies using whole plants to observe if temperature does affect the toxicity of an insecticide

field could done and allow extrapolation a field situation. The efficacy of reduced doses at different temperatures could also be investigated to see if there is any potential for cutting rates of autumn applications.

4. Sub-lethal effects on aphids

Further studies on sub-lethal doses, using doses that were found to penetrate the crop at field and reduced dose-rate applications from the distribution studies (4.), could be applied to individual aphids and measurements of their growth and development recorded. These measures could be inserted into aphid development models to see what consequences there were for aphid population development after an insecticide application.

5. Consequences for field populations of aphids

In either a natural or infested field population, the effect of spraying reduced dose-rates on aphid populations at different growth stages needs to be investigated. This will show whether a single reduced spray can give control of aphids for the season if an early population were sprayed or whether a second spray would be necessary.

6. Models for predicting economic benefits of reduced dose-rate applications

Models already exist that can predict the benefits of using reduced dose-rate insecticide applications e.g. Mann & Wratten, 1988. The above experiments allow the calculation of variables such insecticide efficacy and aphid population development to be calculated. This could be inserted into these type of model to better predict the benefit of using reduced dose-rate insecticides.

7. Mechanistic approaches to effects on predators

Residual and dietary toxicity are the two routes through which predators will be exposed to an applied insecticide (Croft, 1990). In order to further understand what

effects reduced dose-rate applications might have on predators experiments need to investigate further i) differences in toxicity between the soil and crop to observe if the hazard posed to insect on the crop is the same as predators on the ground. This could be achieved by exposing predators to residual deposit applied to leaves in the laboratory or whole plants in the field at different dose rates. Effects on predatory potential also have to be investigated, especially what effect on feeding exposure to aphids contaminated with reduced dose-rates of insecticides compared to untreated controls. Experiments are also need to see if predators show any preference between treated and untreated aphids and if there is a threshold for the rejection of an insecticide contaminated aphid.

In comparison with a field rate application, the effects of reduced dose-rate applications on predators in the field need to be addressed. This would involve pit-fall trapping of ground active predators to look for differences in numbers between the applications. effects on predation in the field also need to be investigated through the use of bait cards that would measure differences in predation between the different insecticide rates. In 'extended laboratory experiments the effects of topical, residual and dietary exposure to an insecticide could be investigated by containing predators in suitable containers and substrates with a known amount of prey and then applying an insecticide at different doses. Treating each route of exposure with the same dose of insecticide would also allow the importance of each route of exposure to be investigated and attempt to reduce the hazard that this route poses by changing the insecticide applied or its timing.

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