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Acute and long-term effects of selective insecticides on *Micromus tasmaniae* Walker (Neuroptera: Hemerobiidae), *Coccinella transversalis* F. (Coleoptera: Coccinellidae) and *Nabis kinbergii* Reuter (Hemiptera: Miridae)

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Abstract The impacts of six selective insecticides on three predatory insect species, Tasman's lacewing (*Micromus tasmaniae*), the transverse ladybird (*Coccinella transversalis*) and the damsel bug (*Nabis kinbergii*), were tested by acute and long-term bioassay. Acute bioassays measured mortality 72 h after exposure to wet sprays and dry residues, and long-term bioassays measured mortality and sublethal effects over a generation. The acute bioassays were not consistently reliable indicators of the harmfulness of insecticides that did not induce high short-term mortality. Pymetrozine caused very low mortality (≤20%) to the larvae of *C. transversalis* in acute tests, but the long-term test showed that 97.6% of individuals were killed before maturity. Similarly, pirimicarb adversely affected reproduction of *C. transversalis* even though there was only a minor effect on short-term mortality. Imidacloprid caused low mortality (≤20%) of *M. tasmaniae* in acute tests, but reproductive capacity was reduced by about two-thirds in long-term tests. Therefore, in some cases, acute bioassays were poor indicators of overall effects of insecticides on populations. Our results support previous studies by other authors that long-term mortality and sublethal effects of selective insecticides on predatory species need to be determined to facilitate their effective use alongside biological control in integrated pest management programs.

Key words bioassay, integrated pest management, IPM, population, selective insecticide.

INTRODUCTION

Successful adoption of integrated pest management (IPM) practices in horticulture has required improved knowledge of the role of beneficial species in crops and the use of pest control methods that support their preservation (Lewis *et al.* 1997). The manufacture of modern insecticides (those claimed by their manufacturers to have at least some selectivity) has provided growers with pest management options that are far more compatible with IPM than have previously been available. Most insecticides developed in the early days of synthetic pesticides were broad-spectrum (such as the organochlorine, organophosphate and carbamate insecticides) as they killed a wide range of pests and also non-target organisms including beneficial species (Perkins 1982). Modern insecticides are generally more selective in their toxicity and so more closely target pest organisms (Hainzl *et al.* 1998). They are commonly referred to as 'soft' or 'selective' insecticides (Galvan *et al.* 2005; Koss *et al.* 2005; Wang *et al.* 2005).

This new group of insecticides provide valuable pest control options in crops because they are not always lethal to the many predatory and parasitic insect species that have the potential to control pest populations to manageable levels (Beddington *et al.* 1978; Biever *et al.* 1994). That is, the combined use of selective chemicals and biological control in IPM programs can provide highly effective long-term pest management (Kogan 1998; Horne & Page 2008). The availability of these insecticides has made an enormous contribution towards the adoption of IPM in Australian crop protection, but their impacts on the array of beneficial species that provide biological control of pest species are still not well understood. Even these more selective insecticides can cause negative acute or sublethal effects on a range of predatory and parasitoid species (Galvan *et al.* 2005; Van Driesche *et al.* 2006). Thus, selective insecticides still need to be evaluated for their effects on beneficial species.

Analysis of the impact of selective insecticides on non-target species is more complex than for broad-spectrum insecticides. Acute tests that measure the effects of exposure on mortality are useful for determining harmful insecticide effects, but they are less reliable at predicting the overall impact of selective chemicals on populations. When chemicals

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do not cause acute mortality of individuals, they may in fact be harmful to the population of that species through longer-term sublethal effects. The life history traits of beneficial species, such as generation time, fecundity, fertility and behaviour, can be adversely affected by chemicals that do not cause substantial mortality to individuals of the species (Stark & Banks 2003; Desneux *et al.* 2007). For example, Hattingh (1996) showed that pyriproxyfen, while not being lethal to individuals, caused complete sterility of coccinellid and hymenopteran biocontrol agents, and so destroyed populations of these species. Similarly, Van Driesche *et al.* (2006) found that spinosad reduced oviposition by 76% for *Neoseiulus cucumeris* Oudemans predatory mites but it was not acutely lethal to individuals. In addition to insecticides, some fungicides cause mortality and reduce fecundity in beneficial arthropods (Bernard *et al.* 2004).

Walthall and Stark (1997) argued that reliance on lethal dose tests, such as LD_{50} tests, to measure pesticide toxicity has shortcomings. They found that lethal concentration estimates by Probit Analysis were not consistent predictors of pesticide impact on population growth. Indeed, several authors have argued that long-term and sublethal effects of pesticides are a better indication of impacts on beneficial organisms than acute tests alone. Stapel *et al.* (2000) found that insecticides affected the foraging ability and lifespan of a parasitoid, and thus argued for the importance of sublethal testing of pesticides. Similarly, measures of population growth rate of the pea aphid *Acyrthosiphon pisum* Harris were more predictive than lethal concentration estimates when determining the impact of imidacloprid exposure to the population (Walthall & Stark 1997).

As pests in crops are controlled by beneficial arthropods at the population level rather than by individual organisms (Holt 1977) the effect of pesticides on beneficial species should be investigated in terms of their effects on populations and not solely for their impact on individuals of a species. A pesticide that causes rapid mortality to most individuals can reliably be considered as harmful to a population of that species. However, a pesticide that causes low or moderate mortality to individuals in the short term needs to be investigated further before its lethality at the population level can be determined (Wennergren & Stark 2000; Forbes & Calow 2002).

Analysis of the long-term effects of selective insecticides on beneficial arthropods is required for those insecticides to be used appropriately and with confidence in crop protection. For IPM systems to gain the highest possible benefit from the biological control organisms present there needs to be a clear understanding of the impacts of all pesticides that are applied.

Here, we tested six selective insecticides for their acute and long-term effects on three predatory species: Tasman's lacewing *Micromus tasmaniae* Walker (Neuroptera: Hemerobiidae), the transverse ladybird *Coccinella transversalis* F. (Coleoptera: Coccinellidae) and the Pacific damsel bug *Nabis kinbergii* Reuter (Hemiptera: Nabiidae). The reliability of acute toxicological effects of the insecticides is discussed and compared with the long-term impacts on species population parameters.

MATERIALS AND METHODS

Insect colonies

Colonies of *M. tasmaniae, C. transversalis* and *N. kinbergii* were maintained at 26°C (± 1.0°C) and 14L : 10D photoperiod. Additionally, colonies of *Lipaphis pseudobrassicae* Davis (turnip aphid) and *Plutella xylostella* L. (diamondback moth) were maintained as sources of prey for the predators.

Field-collected specimens of *M. tasmaniae, C. transversalis* and *N. kinbergii* were regularly introduced to laboratory colonies to maintain genetic diversity. Juveniles of each species were housed in ventilated polypropylene containers ($27 \times 19 \times 8$ cm) and provided with paper towel, water-saturated cotton wool and prey; *M. tasmaniae* and *C. transversalis* were provided twice weekly with aphids and *N. kinbergii* were provided with moth larvae. Adult stages were maintained similarly, with the addition of a 50:50 honey-yeast paste for *M. tasmaniae*, honey-water for *C. transversalis* and substrates on which to lay eggs. These were: strips of cotton wool pads for *M. tasmaniae*, red flagging tape for *C. transversalis* and snow pea pods for *N. kinbergii*.

Acute mortality bioassays

Acute bioassays determined mortality rates caused by seven insecticides (six selective and one broad-spectrum) currently registered for control of pests in various vegetable crops in Australia. They were applied at concentrations within the registered label rates in the state of Victoria (InfoPest 2008): 0.048 g/L spinosad (0.4 mL/L Success®), 0.022 g/L emamectin benzoate (0.5 g/L Proclaim®), 0.10 g/L indoxacarb (0.25 g/L Avatar®), 0.20 g/L pymetrozine (0.4 g/L Chess®), 0.05 g/L imidacloprid (0.25 mL/L Confidor®), 1.0 g/L pirimicarb (2.0 g/L Pirimor®) and 0.75 g/L chlorpyrifos (1.5 mL/L Lorsban 500EC®) (broad-spectrum).

Acute mortality of the insecticides was determined by two types of bioassay on each species: (1) direct application, where juveniles were sprayed directly with the chemical; and (2) dried residue, where juveniles were exposed to dried residues of the chemicals on leaf surfaces. The direct application and dried residue bioassays were performed separately.

For the direct application tests, replicates of first-instar larvae or nymphs (24–48 h old) were placed onto blotting paper in a fume cupboard and sprayed directly with a hand-held pressurised atomiser at a distance of 25 cm to the point of saturation. The average dose of spray deposit was 4.9 μL/cm^2. There were 10 replicates of three to four individuals each for the treatments and control. Treated insects were placed into ventilated 70 mL plastic containers on an unsprayed cabbage (*Brassica oleracea* var. *Capitata*) leaf with a food source of *L. pseudobrassicae*. The containers with treated insects were incubated at 24°C (± 1°C) and 14L : 10D photoperiod for 72 h. Mortality was then assessed. Moribund individuals unable to walk were considered to be dead.

For the residue tests, ten cabbage leaf disks of 50 mm diameter were sprayed separately on both surfaces to the point of

insipient run-off (average 4.9 μL/cm^2 spray deposit) with the insecticide suspensions and 0.01% non-ionic surfactant (Wetter 600). Control replicates were sprayed with water and the non-ionic surfactant. They were then air dried for 2 h at 24°C and placed into the ventilated containers. Three to four larvae of *M. tasmaniae* or *C. transversalis* or nymphs of *N. kinbergii* were then placed into each of the containers and supplied with a food source and water as for the direct application bioassays. The containers with treated insects were incubated for 72 h before the assessment of mortality.

For all bioassays, treatment mortalities were adjusted for control mortality (Abbott 1925). Control mortality was less than 10% for both bioassays reported in this study. Data were analysed by ANOVA and Tukey tests (StatsDirect 2006) to determine the percentage mortality observed for each treatment and to separate differences between treatment means at the 5% level of significance.

Long-term and sublethal bioassays

In addition to acute tests, longer-term fecundity tests were performed on each species. Treatment application was the same as for direct application acute bioassays, except that three replicates of 13–14 first-instar individuals were sprayed separately. The replicates of treated juveniles were housed in three ventilated polypropylene containers (27 × 19 × 8 cm) and were reared through to adults using the same methods as for the laboratory colonies. Adults remained in the containers for 5 days and were then separated into male–female pairs in 400 mL ventilated plastic containers. In addition to food sources, suitable egg-laying strata were provided.

The following observations were recorded: life-stage mortality, the pre-oviposition period (egg hatch to first egg lay), fecundity (number of eggs laid), fertility (number of juveniles produced) and duration of reproductive period. The data were incorporated into abridged life tables (Carey 1993) for each replicate and used to calculate the net reproductive rate, R_o (average number of female offspring produced by a female in a cohort over her lifetime), mean generation time, T (time taken for a female in a cohort to produce a number of offspring equal to the net reproductive rate) and intrinsic rate of increase, r_m (daily rate of natural increase in a closed stable population) (Carey 1993; Walthall & Stark 1997). The relationship between these parameters is summarised as (Carey 1993):

$$r_m = \frac{(\log_e R_o)}{T}$$

For each species and insecticide combination, the life-table parameters (R_o, T and r_m) generated for each replicate were analysed by ANOVA and Tukey tests (StatsDirect 2006) to separate differences between treatments.

RESULTS

Acute mortality bioassays

Chlorpyrifos caused 100% mortality to all species by both direct and residue bioassays. Imidacloprid caused complete mortality to *C. transversalis* and *N. kinbergii* and emamectin benzoate was lethal to *N. kinbergii* through both routes of exposure (Tables 1,2). Long-term bioassays could subsequently not be carried out in these cases. Variable mortalities were obtained for other insecticide–species combinations. Pymetrozine had a low mortality impact (≤20%) by both methods of exposure on all three species, and by these tests appeared to be harmless to individuals of these species. Similarly, pirimicarb also caused low levels of mortality in *M. tasmaniae* and *C. transversalis*. Spinosad induced a moderate level of mortality (39%) to *M. tasmaniae* following exposure to residues.

There were several instances where dried residues caused significantly higher mortality rates than corresponding control mortality, while direct spray application of the same insecticide to the same species was not significantly different from the corresponding control mortality. These treatments were indoxacarb exposure to *C. transversalis* (direct: d.f. = 19, $F = 0.101$, $P = 0.754$; residues: d.f. = 19, $F = 117.8$, $P < 0.0001$) and *N. kinbergii* (direct: d.f. = 19, $F = 1.20$, $P = 0.288$; residues: d.f. = 19, $F = 64.57$, $P < 0.0001$), spinosad exposure to *M. tasmaniae* (direct: d.f. = 19, $F = 3.82$, $P = 0.632$; residues: d.f. = 19, $F = 40.33$, $P = 0.0002$) and pirimicarb exposure to *N. kinbergii* (direct: d.f. = 19, $F = 1.00$, $P = 0.331$; residues: d.f. = 19, $F = 29.56$, $P < 0.0001$) (Tables 1,2).

Long-term and sublethal tests

The long-term investigations over a generation of each species revealed impacts of some insecticides that were not evident in

Table 1 Adjusted mortalities (%) of predatory insects 72 h after direct spray application of insecticides

Insecticide	Concentration (g/L)	Adjusted mortality (%) 72 h after treatment		
		Micromus tasmaniae	*Coccinella transversalis*	*Nabis kinbergii*
Indoxacarb	0.10	3.3 a	0 a	6.9 a
Pymetrozine	0.20	0 a	6.7 ab	4.7 a
Emamectin benzoate	0.022	24.2 b	0 a	100 b
Spinosad	0.048	0 a	7.8 ab	0 a
Imidacloprid	0.05	20.0 b	100 c	100 b
Pirimicarb	1.0	3.3 a	13.0 b	8.6 a
Chlorpyrifos	0.75	100 c	100 c	100 b

Numbers in the same column followed by the same letter are not significantly different ($P \geq 0.05$).

Table 2 Adjusted mortalities (%) of predatory insects following exposure to dried residues of insecticides for 72 h

Insecticide	Concentration (g/L)	Adjusted mortality (%) after 72 h exposure		
		Micromus tasmaniae	*Coccinella transversalis*	*Nabis kinbergii*
Indoxacarb	0.10	4.5 a	80.0 c	71.4 b
Pymetrozine	0.20	0 a	20.0 b	13.9 a
Emamectin benzoate	0.022	9.8 a	7.1 a	97.4 c
Spinosad	0.048	39.2 b	0 a	0 a
Imidacloprid	0.05	3.3 a	100 d	100 c
Pirimicarb	1.0	12.5 a	7.4 a	64.8 b
Chlorpyrifos	0.75	100 c	100 d	100 c

Numbers in the same column followed by the same letter are not significantly different ($P \geq 0.05$).

Table 3 Long-term and sublethal effects of insecticides on *Coccinella transversalis*

Insecticide	Concentration (g/L)	Survival at maturity (% ± SEM)	Pre-oviposition period (days ± SEM)	Net reproductive rate (R_o)	Generation time (T) (days)	Intrinsic rate of increase (r_m)
Control (water)	–	79 ± 10.9	29.8 ± 1.75	42.4 ab	44.2 a	0.081 a
Indoxacarb	0.10	43 ± 23.0	31.8 ± 1.14	32.1 a	44.8 a	0.077 a
Pymetrozine	0.20	2.4 ± 2.4	–	–	–	–
Emamectin benzoate	0.022	86 ± 7.2	29.7 ± 2.28	24.6 ac	40.5 a	0.074 a
Spinosad	0.048	91 ± 2.4	26.4 ± 1.78	51.4 b	39.5 a	0.098 a
Pirimicarb	1.0	76 ± 4.8	46.8 ± 2.79	13.8 c	57.8 b	0.044 b

Numbers in the same column followed by the same letter are not significantly different ($P \geq 0.05$).

Table 4 Long-term and sublethal effects of insecticides on *Micromus tasmaniae*

Insecticide	Concentration (g/L)	Survival at maturity (% ± SEM)	Pre-oviposition period (days ± SEM)	Net reproductive rate (R_o)	Generation time (T) (days)	Intrinsic rate of increase (r_m)
Control (water)	–	60 ± 4.6	25.0 ± 1.00	35.4 a	40.0 a	0.093 a
Indoxacarb	0.10	35 ± 2.9	23.5 ± 1.32	26.2 a	43.4 a	0.072 a
Pymetrozine	0.20	57 ± 3.3	22.6 ± 0.27	36.2 a	41.9 a	0.082 a
Emamectin benzoate	0.022	48 ± 3.5	23.6 ± 1.57	35.2 a	46.2 a	0.074 a
Spinosad	0.048	57 ± 3.3	24.5 ± 0.55	40.5 a	45.9 a	0.077 a
Imidacloprid	0.05	27 ± 1.7	23.0 ± 0.00	11.4 b	41.8 a	0.058 b
Pirimicarb	1.0	65 ± 10.4	28.2 ± 0.63	35.8 a	37.0 a	0.095 a

Numbers in the same column followed by the same letter are not significantly different ($P \geq 0.05$).

the acute bioassays. The most striking example was the effect of pymetrozine on *C. transversalis*, where acute mortalities were low, but the long-term survival of juveniles was severely affected and only one individual pupated and then reached maturity (Table 3). That is, one application of pymetrozine caused extinction of the test population in less than one generation. Development of larvae through successive instars was slower than untreated individuals and mortality of larvae occurred at a steady rate.

Pirimicarb had a negative impact on the population of *C. transversalis* to a lesser extent than pymetrozine. Pirimicarb caused a significant increase in the duration of the pre-oviposition period (d.f. = 5, $F = 5.34$, $P = 0.002$) and significantly reduced (d.f. = 5, $F = 26.75$, $P = 0.0003$) the net reproductive rate to less than half that of the untreated population (Table 3). The intrinsic rate of increase of the population was significantly reduced (d.f. = 5, $F = 188.5$, $P < 0.0001$) by about half.

The survival at maturity of *M. tasmaniae* was significantly reduced by imidacloprid (d.f. = 5, $F = 29.17$, $P = 0.0003$) and indoxacarb (d.f. = 5, $F = 19.37$, $P = 0.004$) (Table 4). Imidacloprid also reduced the net reproductive rate of females to less than one-third of the rate of untreated individuals, thus the value of r_m for *M. tasmaniae* exposed to imidacloprid was reduced to 62% of the untreated population. The pre-oviposition period of *M. tasmaniae* was not affected by the treatments, except for an increase caused by pirimicarb (d.f. = 5, $F = 1.87$, $P = 0.015$) (Table 4).

The net reproductive rate and r_m of *N. kinbergii* were significantly reduced (d.f. = 5, $F = 11.67$, $P = 0.014$; d.f. = 5, $F = 7.53$, $P = 0.034$, respectively) by pirimicarb (Table 5). The survival at maturity of *N. kinbergii* was reduced by pirimicarb (d.f. = 5, $F = 12.27$, $P = 0.018$) and there was a slightly significant (d.f. = 5, $F = 3.22$, $P = 0.048$) increase in survival for the population treated with pymetrozine.

Table 5 Long-term and sublethal effects of insecticides on *Nabis kinbergii*

Insecticide	Concentration (g/L)	Survival at maturity (% ± SEM)	Pre-oviposition period (days ± SEM)	Net reproductive rate (R_o)	Generation time (T) (days)	Intrinsic rate of increase (r_m)
Control (water)	–	57 ± 5.6	26.0 ± 0.26	19.0 a	53.1 a	0.054 a
Indoxacarb	0.10	60 ± 3.1	30.0 ± 2.16	8.2 b	55.2 a	0.036 b
Pymetrozine	0.20	80 ± 5.5	26.3 ± 0.33	14.0 a	44.7 b	0.056 a
Spinosad	0.048	55 ± 5.2	24.5 ± 0.87	11.6 ab	43.0 b	0.053 a
Pirimicarb	1.0	33 ± 6.0	28.8 ± 0.92	8.4 b	50.8 ab	0.028 b

Numbers in the same column followed by the same letter are not significantly different ($P \geq 0.05$).

DISCUSSION

Our results here support the need for long-term and sublethal testing of selective insecticides that are not acutely lethal to beneficial arthropod species. We can conclude from our acute toxicity tests that the broad-spectrum insecticide chlorpyrifos is lethal to all three species tested, that imidacloprid is lethal to *C. transversalis* and *N. kinbergii* and that emamectin benzoate is lethal to *N. kinbergii*. Thus, no further testing for long-term effects was required or possible at the rates tested. In contrast, the acute bioassays of several selective insecticides indicated that these insecticides were harmless in terms of short-term mortality (<30% mortality) (Hassan 1992). However, long-term tests showed that some of the insecticides were harmful at the population level by reducing reproductive capacity or by causing less immediate mortality. The combined effect of mortality and reduction in fecundity on some arthropod populations is discussed by Stark *et al.* (2007).

An important example of long-term impact detected in this study is that of pymetrozine on *C. transversalis*. This insecticide did not cause significant mortality in acute direct and residual exposures, but the long-term bioassay showed its destructiveness to the population. Acute tests therefore erroneously predicted that pymetrozine was safe to *C. transversalis*, while it is in fact lethal to the population by causing mortality to late-instar larvae. Sechser *et al.* (2002) found that pymetrozine was harmless to a species of the same genus, *Coccinella septempunctata* L., after 10-day exposure, but did not report on the survival of individuals to maturity.

The reduction in survival at maturity of *M. tasmaniae* and the decrease in fecundity following imidacloprid exposure were also not predicted by acute tests on this species. Cole and Horne (2006) previously observed high mortality of *M. tasmaniae* larvae that had ingested aphids feeding on lettuce seedlings treated with a high rate of imidacloprid. This observation contrasts with the harmlessness to larvae observed in these acute bioassays and supports the long-term and sublethal effects that imidacloprid is harmful to *M. tasmaniae*.

The harmfulness of indoxacarb to *N. kinbergii* was shown by high mortality following exposure to residues and by a reduction in the net reproductive rate. The acute tests showed that residues were harmful to *N. kinbergii* but exposure to wet spray was safe. These types of variable mortalities suggest the importance of performing a range of tests for each species–insecticide combination before drawing an overall conclusion about the impact of the insecticide on the species.

Our bioassays showed comparative effects of the pesticides on each species following exposure to a discrete age class, newly hatched juveniles. Other factors that could affect the overall impact on field populations are age-specific toxicity and population density and structure (Stark & Banken 1999; Forbes *et al.* 2001). Indeed, we have tested the impact of direct exposure of imidacloprid to adult *M. tasmaniae* (unpubl. data 2008) and observed 100% mortality at the recommended foliar application rate of 0.05 g/L. Further, variable field conditions may alter the effects of insecticides on non-target organisms. For instance, the rate of degradation of insecticide residues may alter their impact on individuals. In addition, selective insecticides may impact negatively on beneficial invertebrates by modifying behaviour or movement (e.g. reduced feeding or avoidance) (Shaw *et al.* 2006), which were not directly tested in this study.

Walthall and Stark (1997) argued that population growth rate factors, such as the intrinsic rate of increase (r_m) and more recently (Stark *et al.* 2007) the delay in population growth index, are superior predictors of population response to toxicant exposure than acute mortality estimates. Accordingly, the population growth estimates calculated in this study may indicate insecticide and species combinations for which field investigations on their impacts should be performed. Examples of reductions in r_m observed here are the impact of pirimicarb on *C. transversalis* and indoxacarb on *N. kinbergii*. The question is whether or not these observed reductions in r_m in the laboratory would translate into reduced ability of the beneficial species to control pest species in the field following an application of the particular insecticide. It is possible that factors such as natural recruitment from outside the sprayed crop and presence of various age classes may compensate for the losses due to the impact of the insecticide on juveniles.

Our results suggest that several of these selective insecticides will not harm populations of the predatory species tested here, and therefore are compatible with their biological control of pest species in crops. For example, pymetrozine is unlikely to harm field populations of *M. tasmaniae* or *N. kinbergii*, emamectin benzoate is unlikely to harm *C. transversalis* and spinosad is likely to be safe to *C. transversalis* and *N. kinbergii*.

Critical to IPM programs is that the pesticides applied should cause minimal disruption to the resident biological control organisms in crops. Knowledge of the effects of pesticides on beneficial species is therefore an essential component in their selection and use. This study has shown that

selective insecticides must be screened for their long-term impacts on beneficial species so that they can be used judiciously in crop protection alongside biological control options.

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REFERENCES

Abbott WS. 1925. A method of computing the effectiveness of an insecticide. *Journal of Economic Entomology* **18**, 265–267.

Beddington JR, Free CA & Lawton JA. 1978. Characteristics of successful natural enemies in models of biological control of insect pests. *Nature* **273**, 513–519.

Bernard MB, Horne PA & Hoffmann AA. 2004. Developing an ecotoxicological testing standard for predatory mites in Australia: acute and sublethal effects of fungicides on *Euseius victoriensis* and *Galendromus occidentalis* (Acarina: Phytoseiidae). *Journal of Economic Entomology* **97**, 891–899.

Biever KD, Hostetter DL & Kern JR. 1994. Evolution and implementation of a biological control-IPM system for crucifers: 24-year case history. *American Entomologist* **40**, 103–108.

Carey JR. 1993. *Applied Demography for Biologists*. Oxford University Press, New York, USA.

Cole PG & Horne PA. 2006. The impact of aphicide drenches on *Micromus tasmaniae* (Walker) (Neuroptera: Hemerobiidae) and the implications for pest control in lettuce crops. *Australian Journal of Entomology* **45**, 244–248.

Desneux N, Decourtye A & Delpuech J-M. 2007. The sublethal effects of pesticides on beneficial arthropods. *Annual Review of Entomology* **52**, 81–106.

Forbes VE & Calow P. 2002. Population growth rate as a basis for ecological risk assessment of toxic chemicals. *Philosophical Transactions of the Royal Society of London* **357**, 1299–1306.

Forbes VE, Sibly RM & Calow P. 2001. Toxicant impacts on density-limited populations: a critical review of theory, practice, and results. *Ecological Applications* **11**, 1249–1257.

Galvan TL, Koch RL & Hutchinson WD. 2005. Effects of spinosad and indoxacarb on survival, development and reproduction of the multicolored Asian lady beetle (Coleoptera: Coccinellidae). *Biological Control* **34**, 108–114.

Hainzl D, Cole LM & Cassida JE. 1998. Mechanisms for selective toxicity of fipronil insecticide and its sulfone metabolite and desulfinyl photoproduct. *Chemical Research in Toxicology* **11**, 1529–1535.

Hassan S. 1992. International Organization for Biological and Integrated Control of Noxious Animals and Plants/Working Group Pesticides and Beneficial Organisms 1992. Guidelines for testing the effects of pesticides on beneficial organisms: description of test methods. *IOBC/WPRS Bulletin* 1992/XV/3.

Hattingh V. 1996. The use of insect growth regulators – implications for IPM with citrus in southern Africa as an example. *BioControl* **41**, 513–518.

Holt RD. 1977. Predation, apparent competition, and the structure of prey communities. *Theoretical Population Biology* **12**, 197–229.

Horne PA & Page J. 2008. *Integrated Pest Management for Crops and Pastures*. Landlinks Press, Melbourne, Australia.

InfoPest. 2008. *InfoPest CD ROM* March 2008. Queensland Department of Primary Industries and Fisheries, Brisbane, Australia.

Kogan M. 1998. Integrated pest management: historical perspectives and contemporary developments. *Annual Review of Entomology* **43**, 243–270.

Koss AM, Jensen AS, Schreiber A, Pike KS & Snyder WE. 2005. Comparison of predator and pest communities in Washington potato fields treated with broad-spectrum, selective, or organic insecticides. *Environmental Entomology* **34**, 87–95.

Lewis WJ, van Lenteren JC, Phatak SC & Tumlinson JH. 1997. A total system approach to sustainable pest management. *Proceedings of the National Academy of Sciences* **94**, 12243–12248.

Perkins JH. 1982. *Insects, Experts, and the Insecticide Crisis: The Quest for New Pest Management Strategies*. Plenum Press, New York, USA.

Sechser B, Reber B & Bourgeois F. 2002. Pymetrozine: selectivity spectrum to beneficial arthropods and fitness for integrated pest management. *Journal of Pesticide Science* **75**, 72–77.

Shaw EM, Waddicor M & Langan AM. 2006. Impact of cypermethrin on feeding behaviour and mortality of the spider *Pardosa amentata* in arenas with artificial vegetation. *Pest Management Science* **62**, 64–68.

Stapel JO, Cortesero AM & Lewis WJ. 2000. Disruptive sublethal effects of insecticides on biological control: altered foraging ability and life span of a parasitoid after feeding on extrafloral nectar of cotton treated with systemic insecticides. *Biological Control* **17**, 243–249.

Stark JD & Banken JAO. 1999. The importance of population structure at the time of toxicant exposure. *Ecotoxicology and Environmental Safety.* **42**, 282–287.

Stark JD & Banks JE. 2003. Population-level effects of pesticides and other toxicants on arthropods. *Annual Review of Entomology* **48**, 505–519.

Stark JD, Vargas R & Banks JE. 2007. Incorporating ecologically relevant measures of pesticide effect for estimating the compatibility of pesticides and biocontrol agents. *Journal of Economic Entomology* **100**, 1027–1032.

StatsDirect. 2006. *StatsDirect Statistical Software*. Version 2.5.7. StatsDirect Ltd., Cheshire, UK.

Van Driesche RG, Lyon S & Nunn C. 2006. Compatibility of spinosad with predacious mites (Acari: Phytoseiidae) used to control western flower thrips (Thysanoptera: Thripidae) in greenhouse crops. *Florida Entomologist* **89**, 396–401.

Walthall WK & Stark JD. 1997. A comparison of acute mortality and population growth rate as endpoints of toxicological effect. *Ecotoxicology and Environmental Safety* **37**, 45–52.

Wang A-H, Wu J-C, Yu Y-S, Liu J-L, Yue J-F & Wang M-Y. 2005. Selective insecticide-induced stimulation on fecundity and biochemical changes in *Tryporyza incertulas* (Lepidoptera: Pyralidae). *Journal of Economic Entomology* **98**, 1144–1149.

Wennergren U & Stark J. 2000. Modeling long-term effects of pesticides on populations: beyond just counting dead animals. *Ecological Applications* **10**, 295–302.

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