Toxicity of novel and conventional insecticides to selected beneficial insects

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ABSTRACT

We tested several novel and commercial insecticides for contact toxicity to selected beneficial insects to evaluate potential compatibilities in integrated pest management programs. Insecticides were sprayed on potted citrus plants at recommended field rates. Leaves were excised and presented to beneficial insects in Petri dishes. Tests often were repeated several days post spray to evaluate residual toxicity. Insecticide classes tested were: insect growth regulators (IGRs) (tebufenozide, methoxyfenozide, diflubenzuron, and pyriproxyfen), pyrethroids (fenpropathrin and cyfluthrin), antibiotics (abamectin and milbemectin), pyridazinone (pyridaben), inorganic (sulfur), chloronicotinyl (imidacloprid), novel pyrrole insecticide (chlorfenapyr), and organophosphates (oxydemeton-methyl, chlorpyrifos, methidathion, azinphosmethyl, and ethion). Beneficial insects tested were: *Cotesia flavipes* (Cameron), and *Allorhogas pyralophagus* Marsh (braconid parasitoids of sugarcane stalkborers); *Catolaccus grandis* (Burks) (Hymenoptera: Pteromalidae) (endoparasitoid of the cotton boll weevil); and, the twice-stabbed ladybeetle (*Chilocorus cacti* L. [Coleoptera: Coccinellidae]). All IGRs tested were nontoxic to *A. pyralophagus*. Methoxyfenozide also was nontoxic to *C. flavipes*. Abamectin and milbemectin showed no toxicity against *C. cacti* and *C. flavipes*. Abamectin also was safe to *C. grandis* and *A. pyralophagus*. Fenpropathrin was toxic to *A. pyralophagus*, but cyfluthrin was not to *C. flavipes*. *C. cacti*, *C. flavipes* and *A. pyralophagus* were not affected by chlorfenapyr. Pyridaben was nontoxic to *C. grandis*, *C. cacti*, and *A. pyralophagus* but was toxic to *C. flavipes* even 7 d post spray. Imidacloprid gave mixed results, being toxic to *A. pyralophagus*, but not to *C. grandis* or *C. cacti*. Measurable toxicity was found against *C. flavipes*. Organophosphates were toxic to most of the insects tested. Oxydemeton-methyl was toxic to *C. flavipes*. Methidathion was toxic to *A. pyralophagus* and showed residual toxicity to *C. flavipes* up to 7 d post spray. Azinphosmethyl and ethion showed residual toxicity to *C. flavipes* up to 3 and 4 d post spray, respectively. Chlorpyrifos was toxic to *C. grandis*, *C. flavipes*, and *A. pyralophagus*, but not to *C. cacti*. In summary, the IGRs and antibiotic insecticides appear compatible with the use of biological control agents, pyrethroids, pyridazinone and chloronicotinyl gave mixed results, and the organophosphates appear incompatible with biological control.

RESUMEN

Se probaron varios insecticidas nuevos y comerciales en lo referente a la toxicidad por contacto a varios insectos benéficos para evaluar su potencial de compatibilidad con programas de manejo integrado de plagas. Los insecticidas se asperjaron en plantas de cítricos en maceta en las dosis recomendadas de campo. Las hojas fueron cortadas y se pusieron en contacto con los insectos benéficos en cajas de Petri. Las evaluaciones frecuentemente se repitieron varios días después de la aspersión para evaluar la toxicidad residual. Los tipos de insecticida probados fueron: reguladores de crecimiento de insectos (IGRs) (tebufenozide, methoxyfenozide, diflubenzuron, y pyriproxyfen), piretroides (fenpropathrin y cyfluthrin), antibióticos (abamectin y milbemectin), pyridazinone (pyridaben), compuestos inorgánicos (azufre), chloronicotinyl (imidacloprid), el nuevo insecticida pirrólico (chlorfenapyr), y los organofosforados (oxydemeton-metilo, chlorpyrifos, methidathion, azinphosmethyl, y ethion). Los insectos benéficos probados fueron: *Cotesia flavipes* (Cameron), y *Allorhogas pyralophagus* Marsh (parasitoides bracónidos del barrenador de la caña de azúcar); *Catolaccus grandis* (Burks) (Himenóptera: Pteromalidae) (endoparasitoide del picudo del algodón); y *Chillocorus cacti* L. C (Coleoptera: Coccinellidae). Ninguno de los reguladores de crecimiento de insectos probados fueron tóxicos a *A. pyralophagus*. Methoxyfenozide tampoco fue tóxico para *C. flavipes*. Abamectin y milbemectin no mostraron ninguna toxicidad contra *C. cacti* y *C. flavipes*. Abamectin también fue inocuo para *C. grandis* y *A. pyralophagus*. Fenpropathrin resultó tóxico a A. pyralophagus, pero cyfluthrin no lo fue para *C. flavipes*. Ni *C. cacti*, *C. flavipes* o *A. pyralophagus* fueron afectados por chlorfenapyr. Pyridaben no resulto tóxico a *C. grandis*, *C. cacti* y *A. pyralophagus* pero fue tóxico a *C. flavipes* aun 7 días después de la aspersión. Imidacloprid brindó resultados contradictorios, siendo tóxico para *A. pyralophagus*, pero no para *C. grandis* o *C. cacti*. Se encontró toxicidad cuantificable para *C. flavipes*. Los organofosforados resultaron tóxicos a la mayoría de los insectos estudiados. Oxydemeton-methyl fue tóxico a *C. flavipes*. Methidathion fue tóxico a *A. pyralophagus*

y mostró toxicidad residual a *C. flavipes* hasta 7 días después de la aspersión. Azinphosmethyl y ethion mostraron toxicidad residual a *C. flavipes* hasta 3 y 4 días después de la aspersión, respectivamente. Chlorpyrifos resulto tóxico a *C. grandis*, *C. flavipes*, y *A. pyralophagus*, pero no a *C. cacti*. En resumen, los reguladores de crecimiento de insectos y los insecticidas antibióticos parecieron ser compatibles con el uso de agentes de control biológico; los piretroides, el pyridazinone y el chloronicotinyl brindaron resultados contradictorios, y los organofosforados parecieron incompatibles con el control biológico.

Key words: insect growth regulator, novel insecticide, nontarget

The adoption of integrated pest management (IPM) strategies saves U. S. agricultural producers more than $500 million annually (Rajotte et al. 1987). In the 1990's, close to 70% of U. S. crop acreage was under some form of IPM management. However, in many crops, only rudimentary IPM was practiced (USDA 1994a). Government, industry, academia and public interests groups recognized the need for implementing more sophisticated IPM strategies with a greater

Table 1. Insecticides tested against selected beneficial insects.

Brand Name	Common Name	Class	Chemistry	Company
Confirm® 70W	tebufenozide (RH-5992)	IGR	benzoic acid, 3,5-dimethyl, 1-(1,1-dimethylethyl) 2-(4-ethylbenzoyl) hydrazide	Rohm and Haas, Philadelphia, PA
Intrepid®	methoxyfenozide (RH-2485)	IGR	N'-Tert-butyl N'-(3,5-dimethylbenzyl)-3-methoxy-2-methyl-bennzaldehyde	Rohm and Haas
Micromite® 25W	diflubenzuron	IGR	N-[[(4-chlorophenyl) amino] carbonyl]-2,6-difluorobenzamide	Uniroyal, Middlebury, CT
Knack® 0.86 EC	pyriproxyfen (V-71639)	IGR	2-(1-methyl-2-(4-phenoxyphenoxy) ethoxy) pyridine	Valent, Walnut Creek, CA
Danitol® 2.4EC	fenpropathrin	Pyd	α-cyano-3-phenoxybenzyl 2,2,3,3-tetramethyl-cyclopropanecarboxylate	Sumitomo, NY, NY
Baythroid 2L®	cyfluthrin	Pyd	cyano (4-fluoro-3-phenoxyphenyl) methyl-3-(2,2-dichloroethenyl)-2,2-dimethylcyclopropanecarboxylate	Bayer, Kansas City, MO
Agri-Mek® 0.15 EC	abamectin	Anti	80% avermectins B_1a and B_1b	Novartis, Greensboro, NC
CM-006 1.0EC	milbemectin	Anti	(Chemistry not released)	Sankyo, Tokyo, Japan
Nexter® 75 WP	pyridaben	Pzn	2-tert-butyl-5-(4-tert-butylbenzylthio)-4-chloropyridazin-3(2H)-one	BASF, Research Triangle Park, NC
Orchex® 796		POil		Exxon, Houston, TX
Kumulus® DF	sulfur	InOrg	sulfur	BASF
Admire® 2F	imidacloprid	CN	1-[(6-chloro-3-pyridinyl)methyl]-N-nitro-2-imidazolidinimine	Bayer
Provado® 1.6F	imidacloprid	CN	1-[(6-chloro-3-pyridinyl)methyl]-N-nitro-2-imidazolidinimine	Bayer
Alert® 2SC	chlorfenapyr (AC 303,630)	Pye	14-bromo-2-(4-chlorophenyl)-1 (ethoxymethyl)-5-(trifluoromethyl) pyrrole-3-carbonitrile	American Cyanamid, Parsippany, NJ
Metasystox-R®	oxydemeton-methyl	OP	S-[2-(ethylsulfinyl)ethyl] 0,0-dimethyl phosphorodithioate	Gowan, Yuma, AZ
Lorsban* 4E	chlorpyrifos	OP	0,0-diethyl-0-(3,5,6-trichloro-2-pyridinyl)	Dow Agrosciences, Indianapolis, IN
Supracide® 25WP	methidathion	OP	0,0-dimethyl-S-[5-methoxy-2-oxo-1,3, 4-thiadiazol-3(2H)-yl-methyl]-dithiophosphate	Gowan
Guthion® 2L	azinphosmethyl	OP	0,0-dimethyl S-[(4-oxo-1,2,3-benzotriazin-3(4H)-yl)methyl]-phosphorodithioate	Bayer
(Ethion 4 Miscible)	ethion	OP	0,0,0',0'-tetraethyl S,S'-methylene bisphosphoro-dithioate	FMC, Philadelphia, PA

IGR = insect growth regulator; Pyd = pyrethroid; Anti = antibiotic; Pzn = pyridazinone; POil = petroleum oil; InOrg = inorganic; Ni = chloronicotinyl; Pye = pyrrole; OP = organophosphate

Table 2. Experiment 1 – Percentage mortality in *C. grandis*.*

	% Mortality					
Treatments	0 d	1 d	2 d	3 d	4 d	9 d
pyridaben	0.0b	0.0b	–	–	–	–
pyridaben + sulfur	0.0b	0.0b	–	–	–	–
abamectin	5.3b	0.0b	–	–	–	–
imidacloprid	8.0b	4.0b	–	–	–	–
chlorpyrifos	88.0a	42.7a	14.7a	5.3a	2.7a	0.0a
control	0.0b	1.3b	0.0a	0.0a	0.0a	0.0a

* – test not conducted because of low parasite mortality on bioassay of previous day (n = 75, 5 Petri dishes x 15 insects / dish); means followed by the same letter within each column are not significantly different (DMRT, P = 0.05)

emphasis on biologically-based methods and a reduction in reliance on broadly toxic chemical pesticides (CSREES 1998). In response, the U. S. Department of Agriculture (USDA) announced an initiative calling for the adoption of biologically-based IPM methods on 75% of U. S. crop acreage by the year 2000 (USDA 1994b). Furthermore, with the passage of the Food Quality Protection Act (FQPA; H. R. 1627) in August 1996, the use of entire classes of insecticides will be severely curtailed, resulting in the need for development and evaluation of safer classes of insecticides.

Compliance with the USDA initiative and the FQPA will require the development and evaluation of insecticides with very specific host ranges, making them compatible with biological control agents and reducing the risks to human health. Many conventional broad-spectrum insecticides are extremely toxic to non-target organisms, especially natural enemies. Parasitic Hymenoptera often are far more susceptible to insecticides than their hosts. Target host insects usually possess detoxification mechanisms adapted to a wide range of plant toxins, whereas parasitoids are adapted only to those of a specific host or narrow range of hosts (Baker et al. 1995). Several workers have evaluated lethal and sublethal effects of conventional commercial insecticides on natural enemies (e.g. Bayoun et al. 1995, Baker and Throne 1995, Baker et al. 1995, Jones et al. 1995, Rathman et al. 1995, Rumpf et al. 1997).

Recently, interest has focused on the evaluation of the toxicities of 'biorational' insecticides (i.e. those based on natural products or synthesized analogues of naturally-occurring biochemicals, Bentz and Neal 1995). Evaluations have been performed on: insect growth regulators (IGRs) (Biddinger and Hull 1995, Legaspi et al. 1999a), neem (Spollen and Isman 1996), an avermectin derivative (Biddinger and Hull 1995, Kok et al. 1996), and an extract from *Nicotiana gossei* Domain (Bentz and Neal 1995). The biorational insecticides usually were less toxic to natural enemies than conventional insecticides. Dhadialla et al. (1998) report that the IGR insecticides tebufenozide (RH-5992, tradenames Mimic®, Confirm®, and Romdan®) and methoxyfenozide (RH-2485, tradename Intrepid®, Rohm and Haas, Philadelphia, PA) are very selectively toxic to lepidopteran pests and are safe to beneficial insects. Studies on the safety of tebufenozide to non-target insects are reported by Heller et al. (1992), Brown (1994) and Biddinger and Hull (1995). Less is known on the safety of methoxyfenozide because it is a newer insecticide. However, in a recent paper, both IGRs caused zero mortality in *Allorhogas pyralophagus* Marsh (Hymenoptera: Braconidae), a parasitoid of the Mexican rice borer, *Eoreuma loftini* (Dyar) (Lepidoptera: Pyralidae) (Legaspi et al. 1999a).

We studied the toxicities of insect growth regulators on selected insect natural enemies and compared the effects against other novel and conventional chemical insecticides.

MATERIALS AND METHODS

Insecticidal treatments. The complete list of insecticides tested are presented in Table 1, and listed by brand name, common name, class of insecticide, chemistry and supplier. When necessary, additives were used in conjunction with the insecticides. Latron CS-7® is an adjuvant (60% blend alkylaryl polyethoxylate and sodium salt of alkyl sulfonated alkylate; Rohm and Haas, Philadelphia, PA). The surfactant Silwet® L-77 (99.5% polyalkyleneoxide modified heptamethyltrisiloxane; Loveland, Greeley, CO) was used at a rate of 0.0125% (v/v). Kinetic®, a nonionic adjuvant (99% blend of polyalkylenoxide modified polydimethylsiloxane and nonionic surfactants; Helena, Memphis, TN), was used at a rate of 0.0125% (v/v). The petroleum oil, Narrow range (NR) 435 (Sun Oil, Philadelphia, PA), was used at a rate of 0.5% (v/v).

Beneficial insects tested. Cotesia flavipes (Cameron) (Hymenoptera: Braconidae), imported from India, is credited with successful biological control of the sugarcane borer, *Diatraea saccharalis* (F.) (Lepidoptera: Pyralidae) in south Texas (Fuchs et al. 1979). Parasitoids tested were obtained from a laboratory colony being reared at the Texas Agricultural Experiment Station (TAES) in Weslaco using *D. saccharalis* as the host insect. Smith et al. (1987) imported the braconid parasitoid *Allorhogas pyralophagus* Marsh from Mexico as a

Table 3. Experiment 4 – Percentage mortality in *C. flavipes*.*

	% Mortality		
Treatments	0 d	1 d	2 d
pyridaben	95.0a	90.0a	100.0a
chlorfenapyr	8.3b	3.3c	–
methoxyfenozide	3.3b	5.0b	–
abamectin	1.7b	21.7b	0.0b
ethion	91.7a	96.7a	100.0a
control	1.7b	3.3b	0.0a

* – test not conducted because of low parasite mortality on bioassay of previous day (n = 60, 3 Petri dishes x 20 insects / dish at 1- and 1-d post spray; n = 30, 3 Petri dishes x 10 insects / dish at 2-d post spray); means followed by the same letter within each column are not significantly different (DMRT, P = 0.05).

Table 4. Experiment 5 – Percentage mortality in *C. flavipes*.*

	% Mortality					
Treatments	0 d	1 d	2 d	3 d	6 d	7 d
pyridaben	98.0a	96.0a	98.0a	100.0a	90.0a	100.0a
methoxyfenozide	0.0b	4.0b	6.0d	0.0d	–	–
chlorpyrifos	100.0a	100.0a	52.0c	20.0c	0.0b	–
methidathion	98.0a	100.0a	82.0b	70.0b	14.0b	5.0b
control	0.0b	4.0b	0.0d	2.0a	0.0b	2.0a

* – test not conducted because of low parasite mortality on bioassay of previous day (n = 50, 5 Petri dishes x 10 ♀ / dish); means followed by the same letter within each column are not significantly different (DMRT, P = 0.05).

Table 5. Experiment 7 – Percentage mortality in *C. flavipes*.*

	% Mortality					
Treatments	1 d	2 d	3 d	7 d	8 d	10 d
chlorpyrifos	83.0a	75.0a	32.5a	–	1.8a	3.9a
azinphosmethyl	48.0ab	41.5a	15.8a	–	2.1a	–
imidacloprid (foliar)	5.0c	0.0b	15.0a	–	1.8a	–
oxydemeton-methyl	17.6c	0.0b	0.0a	–	3.6a	0.0a
control	3.1c	0.0b	4.2a	0.0a	4.0a	0.0a

* – test not conducted; (4 Petri dishes, 9-15 insects / dish); means followed by the same letter within each column are not significantly different (DMRT, P = 0.05).

Table 6. Experiment 8 – Percentage mortality in *A. pyralophagus*.*

	% Mortality								
Treatments	0 d	1 d	3 d	6 d	9 d	14 d	22 d	29 d	30 d
fenpropathrin + surfactant	100ab	50a	93a	93a	100a	100a	90a	87a	100a
pyriproxyfen	0d	0b	0b	–	–	–	–	–	–
pyriproxyfen + 0.5% N435 oil	0d	0b	3b	–	–	–	–	–	–
imidacloprid (0.03 kg ai/ha)	20c	3b	0b	–	–	–	–	–	–
imidacloprid (0.06 kg ai/ha)	30c	10ab	17b	12b	5b	30b	2b	–	–
methidathion	60b	0b	0b	–	–	–	–	–	–
control	0d	0b	0b	0c	0b	0c	5b	0b	0b

* – test not conducted because of low parasite mortality on bioassay of previous day; (3 Petri dishes, 10-15 insects / dish); means followed by the same letter within each column are not significantly different (DMRT, P = 0.05)

biological control agent against the Mexican rice borer, *Eoreuma loftini* (Dyar) (Pyralidae), currently the key pest of sugarcane in south Texas (Legaspi et al. 1999b). The parasitoid is recovered consistently in the field (Legaspi et al. 1997), but is unable to exert complete control of the rice borer, perhaps because it is unable to attack hosts that have tunneled deep into the cane stalk (Hawkins et al. 1987). *Catolaccus grandis* (Burks) (Hymenoptera: Pteromalidae) is an important parasitoid of the boll weevil, *Anthonomus grandis* Boheman (Coleoptera: Curculionidae). The boll weevil is the key pest in much of the cotton growing areas in the United States (Bottrell 1976). *C. grandis* was introduced from Mexico to the U. S. in the early 1970's (Morales-Ramos et al., 1996). Although *C. grandis* possesses many of the desired biological attributes of a successful control agent (Morales-Ramos et al. 1995), it is unable to overwinter and establish in the field (Johnson et al. 1973). *C. grandis* was obtained from colonies maintained at the USDA ARS Beneficial Insects Research Unit, Weslaco, TX, using *A. grandis* as the host insect. Larvae and adults of the twice-stabbed ladybeetle (*Chilocorus cacti* L. (Coleoptera: Coccinellidae)) were collected from trifoliate orchard trees in Weslaco, Texas.

Experimental procedure. A common experimental protocol was followed for all the toxicity tests. Two potted citrus (grape fruit and sour orange) plants (Expts. 1-6, and 8) and orchard trees (Expts. 7 and 9) in Weslaco, Texas were sprayed with the labeled and experimental insecticides following recommended rates. Controls were sprayed with water only. Unless stated otherwise, the insecticides were applied using a CO_2 koke cap back sprayer at 2.1 kg/sq cm. In 3-year old orchard trees ('Rio red' grapefruit), foliar materials were sprayed using a Hy-Pro 5200 handgun sprayer (HyPro Sprayers, St. Paul, MN) at 5.6 kg/sq cm and 1870.7 liters of water per ha until run-off. The parasitoids to be tested were anesthetized using CO_2 gas for 3-5 s to facilitate transfer to Petri dishes where they were exposed to 2-3 treated leaves, excised from the potted plants. Predators were not anesthetized. Exposure times of 4-6 h were used, depending on the parasitoid being tested. The initial bioassay conducted immediately after spraying is designated as 0 d post spray. To measure residual effects of the insecticides, new sets of leaves were excised from the potted plants at different days after spraying, and presented to new sets of test insects. In most experiments, testing for residual toxicity was halted if the insecticide caused insignificant parasitoid mortality. Each experiment had a control check consisting of parasitoids

exposed to 2 untreated leaves. Each Petri dish was fitted with a plastic tubing connected to a vacuum pump (2.1 kg/sq cm) for aeration. All dishes were kept in a controlled temperature environment [26 ± 2ºC, 65 ± 5% RH, 12:12 (L:D) h]. Treatments were arranged in a complete randomized design with 3-5 replicates, each replicate consisting of 10-20 insects. Percentage mortality data were analyzed by analysis of variance (ANOVA; SAS Institute, Cary, NC). Separate ANOVA was performed for each post spray test. For clarity of presentation, we show only the results of the means separations tests (DMRT) following significant ANOVA, i.e., individual *F*, df, and *P* values are not presented. Percentage data were transformed prior to statistical analysis (arc sine - square root method), but are presented as untransformed means.

Experiment 1. *C. grandis* was exposed to: pyridaben (0.45 kg ai/ha), pyridaben + sulfur (0.18 kg ai/ha + 1.8 kg/ha), abamectin (0.013 kg ai/ha), imidacloprid (0.18 kg ai/ha), chlorpyrifos (3.36 kg ai/ha) and an untreated check. In addition to the initial test at 0 d post spray, residual effects were tested at 1 d post spray for pyridaben, pyridaben + adjuvant, abamectin, and imidacloprid. For chlorpyrifos, residual effects were tested at 1, 2, 3, 4, and 9-d post spray.

Experiment 2. *C. cacti* was exposed to the treatments described in Expt. 1. Additional treatments consisted of chlorfenapyr (0.22 kg ai/ha), and milbemectin (0.03 kg ai/ha). Residual effects of the insecticides were not tested (*n* = 40, 4 Petri dishes x 10 insects / dish).

Experiment 3. *C. flavipes* received the same treatments as described in Expt. 1 for *C. grandis*. Residual effects of the insecticides were not tested.

Experiment 4. *C. flavipes* was tested using: pyridaben (0.22 kg ai/ha), chlorfenapyr (0.22 kg ai/ha), methoxyfenozide (0.22 kg ai/ha), abamectin (0.013 kg ai/ha), ethion (2.8 kg ai/ha), and an untreated check. Residual effects were tested at 1 d post spray for chlorfenapyr and methoxyfenozide. Residual effects were tested at 1, 2, and 4 d post spray for the remaining treatments.

Experiment 5. *C. flavipes* was treated with: pyridaben (0.22 kg ai/ha), methoxyfenozide (0.22 kg ai/ha), chlorpyrifos (1.12 kg ai/ha), methidathion (1.12 kg ai/ha), and an untreated check. Residual effects were tested at 1, 2, 3, 6, and 7-d post spray for all treatments.

Experiment 6. *C. flavipes* was treated with: milbemectin (0.03 kg ai/ha), milbemectin + oil (0.03 kg ai/ha), milbemectin (0.03 kg ai/ha)+ adjuvant, abamectin (0.013 kg ai/ha) + oil, and an untreated check. Residual effects were tested at 1 and 2-d post spray (*n* = 50, 5 Petri dishes x 10 ♀ / dish).

Experiment 7. Insecticides were applied until run-off using a Hy-Pro 5200 handgun sprayer at 5.6 kg/sq cm in 3-year old orchard trees. *C. flavipes* was exposed to the following insecticide treatments: chlorpyrifos (1.7 kg ai/ha), azinphosmethyl (1.1 kg ai/ha), imidacloprid (0.112 kg ai/ha), oxydemeton-methyl (0.84 kg ai/ha), and an untreated control.

Experiment 8. *A. pyralophagus* was exposed to the following treatments: fenpropathrin (0.34 kg ai/ha) + surfactant, pyriproxyfen (0.074 kg ai/ha), pyriproxyfen (0.074 kg ai/ha + oil), imidacloprid (0.03 and 0.059 kg ai/ha), methidathion (0.56 kg ai/ha), and an untreated control. Residual toxicities were tested at 1, and 3-d post spray for pyriproxyfen and pyriproxyfen + oil, and imidacloprid (0.03 kg ai/ha), and methidathion. Imidacloprid (0.059 kg ai/ha) was tested at 1, 3, 6, 9, 14 and 22-d post spray. Fenpropathrin was tested 1, 3, 6, 9, 14, 22, 29 and 30 d post spray.

Experiment 9. Insecticides were applied in orchard trees. *A. pyralophagus* was treated with: diflubenzuron (0.34 kg ai/ha), diflubenzuron (0.34 kg ai/ha) + oil, chlorfenapyr (0.22 kg ai/ha), chlorfenapyr + oil (0.34 kg ai/ha), imidacloprid (0.06 kg ai/ha) + adjuvant, abamectin (0.013 kg ai/ha) and an untreated control. Residual effects were tested at 1 d post spray for chlorfenapyr and abamectin treatments. The chlorfenapyr + oil treatment was tested 1 and 5-d post spray (*n* = 45, 3 Petri dishes x 15 insects / dish).

Experiment 10. *A. pyralophagus* was treated with tebufenozide (0.29 kg ai/ha), chlorpyrifos (3.37 kg ai/ha), pyridaben (0.44 kg ai/ha), petroleum oil (2% v/v) and a control. Residual toxicity was tested 1, 2, and 3-d post spray.

RESULTS

Experiment 1. Only chlorpyrifos caused significant mortality in *C. grandis* (Table 2). Tests for residual mortality continued until 9-d post spray. In all other insecticide treatments, tests were halted after 1 d.

Experiment 2. None of the insecticides tested caused significant mortality in *C. cacti*. For this reason, no tests for residual toxicity were conducted.

Experiment 3. Pyridaben and chlorpyrifos caused the highest mortality in *C. flavipes* (87.5% and 98%, respectively), followed by pyridaben + sulfur (45.1%), imidacloprid (11.3%), and abamectin (3.6%). Mortality due to abamectin did not differ significantly from the control mortality of 0.0%.

Experiment 4. The novel insecticides pyrrole and methoxyfenozide did not cause mortality in *C. flavipes* significantly greater than mortality recorded in the control (Table 3). Tests for residual activity in these two compounds was halted after 1 d post spray. Pyridaben and ethion showed significant parasitoid mortality even 4 d post spray.

Experiment 5. As in Expt. 4, pyridaben proved highly toxic to *C. flavipes*, causing 100% mortality up to 7 d post spray (Table 4), exceeding toxicity even of the organophosphates methidathion and chlorpyrifos. In contrast, mortality due to methoxyfenozide did not differ significantly from the control at all times tested.

Experiment 6. Milbemectin and abamectin did not cause mortality in *C. flavipes* significantly higher than control mortality. Parasitoid mortality using milbemectin alone was 0, 0 and 2% at 0, 1, and 2 d post spray. Mortality was 0% on all 3 test days using milbemectin and an oil. With the adjuvant, mortalities were 2, 2, and 4% on the 3 test days, respectively. Abamectin and oil caused parasitoid mortalities of 0, 0, and 4%, respectively. Control mortality was 0% on all three days.

Experiment 7. One d post spray, the organophosphates chlorpyrifos and azinphosmethyl caused higher mortalities in *C. flavipes* than did imidacloprid and oxydemeton-methyl (also an organophosphate, Table 5). Residual toxicity was found in chlorpyrifos and azinphosmethyl 2 d post spray. Imidacloprid caused no significant parasitoid mortality.

Table 7. Summary of insecticide effects on beneficial insects.

Common Name	Class[1]	Not affected	Deleterious[2]
tebufenozide	IGR	*A. pyralophagus*	
methoxyfenozide	IGR	*C. flavipes*	
diflubenzuron	IGR	*A. pyralophagus*	
pyriproxyfen	IGR	*A. pyralophagus*	
abamectin	Anti	*C. grandis, C. cacti, C. flavipes, A. pyralophagus*	
milbemectin	Anti	*C. cacti, C. flavipes*	
fenpropathrin	Pyd		*A. pyralophagus* (30)
cyfluthrin	Pyd	*C. flavipes*	
pyridaben	Pzn	*C. grandis, C. cacti, A. pyralophagus*	*C. flavipes* (7)
imidacloprid	CN	*C. grandis, C. cacti, C. flavipes*	*A. pyralophagus* (6)
chlorfenapyr	Pye	*C. cacti, C. flavipes, A. pyralophagus*	
oxydemeton-methyl	OP		*C. flavipes* (1)
chlorpyrifos	OP	*C. cacti*	*C. grandis, C. flavipes* (3), *A. pyralophagus* (2)
methidathion	OP		*C. flavipes* (7), *A. pyralophagus* (1)
azinphosmethyl	OP		*C. flavipes* (3)
ethion	OP		*C. flavipes* (4)

[1]IGR = insect growth regulator; Pyd = pyrethroid; Anti = antibiotic; Pzn = pyridazinone; CN = chloronicotinyl; Pye = pyrrole; OP = organophosphate

[2]Numbers in parentheses indicate residual toxicity (d)

Experiment 8. Imidacloprid (12 g ai/ac) and the experimental insecticide pyriproxyfen caused no significant mortality in *A. pyralophagus* at 0, 1, or 3-d post spray (Table 6). However, residual toxicity of imidacloprid at the higher application rate of 24 g ai/ac caused significant parasitoid mortality as long as 14 d post spray. Methidathion was highly toxic at the time of application, but showed no residual effects at 1, and 3 d post spray. The pyrethroid fenpropathrin caused very high parasitoid mortalities as long as 30 d post spray.

Experiment 9. No significant toxicity effects were found in any of the treatments against *A. pyralophagus*. All treatments and the control showed 0% mortality, except the chlorfenapyr + oil treatment at 5-d post spray which was 7% ($P > 0.05$).

Experiment 10. Using the standard 4-h exposure time, no treatments caused significant parasitoid mortality. However, at 24-h exposure, chlorpyrifos showed significant toxicity. Parasitoid mortality was 24, 14, 18 and 6% at 0, 1, 2, and 3-d post spray, respectively.

DISCUSSION

The results of the various insecticide bioassays are summarized in Table. 7. The IGRs, tebufenozide and methoxyfenozide, mimic the action of the molting hormone ecdysterone (20-hydroxyecdysone, Dhadialla et al. 1998), thus accounting for its specificity against the target pests. Pyriproxyfen is a juvenile hormone analogue known to inhibit insect metamorphosis, embryogenesis, reproduction and larval development in target pests. However, pyriproxyfen has also shown measurable toxicity to several natural enemies of scale insects (Mendel et al. 1994). Pyrethroids disrupt the transmission of nerve impulses in insects and mammals (Sparks 1996). Most pyrethroids are much less toxic to mammals than the organophosphates they replaced. The antibiotic insecticides abamectin and milbemectin are derived from natural products. Abamectin is comprised of two avermectins, which are complex natural products synthesized by the soil fungus, *Streptomyces avermitilus*. Avermectins may also be valuable as veterinary products because of toxicity to internal vertebrate parasites, but not to the host (Miller 1996). Imidacloprid is a nicotinic insecticide based on a natural toxin extracted from marine worms (Miller 1996). Insects resistant to organophosphates appear to lack resistance to imidacloprid due to differences in their modes of action. The insecticide is being developed for control of sucking insects, including aphids, thrips and whiteflies (Sparks 1996). Pyridazinones inhibit mitochondrial respiration and are effective pesticides against mites, aphids, whiteflies, and thrips of tree fruits. These compounds are generally safe to mammals and birds. Pyrroles are a relatively novel type of insecticide based on a product of *Streptomyces*, which suggests they are a form of antibiotic insecticide. Unlike many insecticides that target the insect nervous system, pyrroles disrupt insect ATP production (Sparks 1996). Organophosphates represent a large and diverse group of insecticides that target the nervous system. These compounds deactivate the essential enzyme acetylcholinesterase which breaks down the neurotransmitter acetylcholine. Subsequent accumulation of acetylcholine results in overstimulation followed by exhaustion and disruption of neural transmission. Organophophates are extremely toxic to nontarget organisms, including birds, mammals and fish.

The results of these bioassays reiterate that broad predictions can be made regarding the ecotoxicological effects of certain classes of insecticides, but specific nontarget or beneficial insect species can display a range of tolerances to particular insecticides. Host specificity bioassays are likely to be necessary in many cases. The insecticide classes will be discussed in an order roughly approximating increasing toxicity to nontarget or beneficial insects.

The IGRs, tebufenozide, diflubenzuron, and pyriproxyfen, were found to induce insignificant mortalities against *A. pyralophagus*. Methoxyfenozide was nontoxic to *C. flavipes*. Tebufenozide has previously been found to cause zero mortality in *A. pyralophagus* at 1- and 4-d post spray, and to have insignificant effects on adult parasitoid survivorship (Legaspi et al. 1999a). Other studies indicate that both tebufenozide (e.g. Biddinger and Hull 1995, Smagghe and Degheele 1995, Rumpf et al. 1997) and methoxyfenozide (Dhadialla et al. 1998) are safe to beneficial insects. French et al. (1997) reported no mortality in *A. pyralophagus* when exposed for 6-8 h to field-collected leaves from trees sprayed with chlorphenapyr, imidacloprid, diflubenzuron or abamectin.

Diflubenzuron is the most thoroughly studied compound in a class known as benzoylphenyl ureas, which are recognized to be highly effective IGR insecticides (Butaye and Degheele 1995). With few exceptions, diflubenzuron has shown no appreciable effects on hymenopteran or dipteran parasitoids (Butaye and Degheele 1995, and references cited). Diflubenzuron caused no deleterious effects through direct or residual contact in the predatory stinkbug, *Podisus maculiventris* (Say) (Heteroptera: Pentatomidae), but was highly toxic when ingested (De Clercq et al. 1995). Diflubenzuron can also cause significant larval mortality in *Eulophus pennicornis* (Nees) (Hymenoptera: Eulophidae), a larval ectoparasite of *Mamestra brassicae* L. (Lepidoptera: Noctuidae) (Butaye and Degheele 1995).

Of the IGRs we tested, perhaps the most mixed results have been reported for pyriproxyfen. Although we found pyriproxyfen safe to *A. pyralophagus*, the phenoxy juvenile hormone analog has shown toxicity against several dipteran, coleopteran and hemipteran natural enemies of scale insects (Mendel et al. 1994). Whether administered by contact or ingestion, pyriproxyfen also caused severe deformities in *P. maculiventris* during ecdysis (De Clercq et al. 1995). Against aphelinid (Hymenoptera) parasitoids of the silverleaf whitefly, *Bemisia argentifolii* Bellows and Perring (Homoptera: Aleyrodidae), pyriproxyfen was nontoxic to *Encarsia pergandiella* Howard, relatively safe to *E. transvena* (Timberlake), but relatively toxic to *E. formosa* Gahan (Liu and Stansly 1997). Nontoxic effects were also found in another aphelinid, *Aphytis holoxanthus* DeBach, an ectoparasitoid of citrus scales (Peleg 1988).

In our tests, the antibiotic insecticides abamectin and milbemectin showed no toxicity against *C. cacti* and *C. flavipes*. Additionally, abamectin was safe to *C. grandis* and *A. pyralophagus*. Previous workers have reported varying degrees of abamectin toxicity to beneficial insects, including: the mite predator, *Stethorus punctum* (LeConte) (Coleoptera: Coccinellidae) (Biddinger and Hull 1995); *E. formosa* (Zchori-Fein et al. 1994); and, pupal parasitoids (Hymenoptera: Pteromalidae) of the house fly, *Musca domestica* L. (Diptera: Muscidae) (Geden et al. 1992). However, possibilities exist for abamectin to be used in conjunction with biological control agents such as: *E. formosa* to control the greenhouse whitefly, *Trialeurodes vaporariorum* Westwood (Homoptera: Aleyrodidae) (Zchori-Fein et al. 1994); *Aphelinus semiflavus* Howard (Hymenoptera: Aphelinidae) and *Diaeretiella rapae* (McIntosh) (Hymenoptera: Braconidae) against the green peach aphid, *Myzus persicae* Sulzer (Homoptera: Aphididae) (Shean and Cranshaw 1991); and the predatory mite *Phytoseilus persimilis* Anthias-Henriot (Acari: Phytoseiidae) to control the twospotted spider mite, *Tetranychus urticae* Koch (Acari: Tetranychidae) (Zhang and Sanderson 1990). No previous works were found regarding the toxicity of milbemectin to nontarget insects.

Because chlorfenapyr is a novel pyrrole insecticide, possessing a novel mode of action (Pimprale et al. 1997), relatively little is known about toxicity to nontarget insects. We found that all beneficial insects tested (*C. cacti*, *C. flavipes* and *A. pyralophagus*) were not affected by chlorfenapyr. These results indicate that chlorfenapyr is a relatively benign insecticide. The compatibility between insecticides and biological control agents is similar to that found in the antibiotic insecticides abamectin and milbemectin.

Pyrethroid insecticides in general have shown mixed results when evaluated for toxicity to nontarget and beneficial insects. Among recent examples, bifenthrin was toxic to all parasitoid stages of *E. pergandiella* attacking the silverleaf whitefly, *B. argentifolii* (Stansly and Liu 1997). Aphid parasitoids belonging to the genus *Aphidius* were at greater risk from exposure to deltamethrin than their aphid hosts (Longley and Jepson 1997). However, Cho et al. (1997) argue that among the available insecticides, pyrethroids are probably the safest to both parasitoids and predators. As a specific example, they cite bioassays using several pyrethroid insecticides against the aphid predator *Harmonia axyridis* Pallas (Coleoptera: Coccinellidae). In this study, fenpropathrin was toxic to *A. pyralophagus*, and cyfluthrin was not to *C. flavipes*.

Pyridaben is a new insecticide and little has been published regarding its effects on either target or nontarget organisms. French and Bruno (1996) evaluated pyridaben as a control for citrus mites, and suggested the insecticide might have benign toxicity against nontarget insects. Our findings show that pyridaben was nontoxic to *C. grandis*, *C. cacti*, and *A. pyralophagus*. However, *C. flavipes* showed residual mortality even 7 d post spray.

Toxicity of imidacloprid was evaluated against three phytoseiid mites: *Neoseiulus collegae* (de Leon), *Phytoseilus macropilis* (Banks), *Proprioseiopsis mexacanus* (Garman); and the predators *Deraecoris nebulosus* (Uhler) (Heteroptera: Miridae), *Olla v-nigrum* (Mulsant) (Coleoptera: Coccinellidae) and *Hippodamia convergens* (Guerin-Meneville) (Coccinellidae) (Mizell and Sconyers 1992). At recommended field rates, imidacloprid showed little toxicity to the predatory mites, but was toxic to most of the predators. Only *P. mexacanus* showed tolerance to imidacloprid. The use of imidacloprid to control the Russian wheat aphid, *Diuraphis noxia* (Kurdjumov) had no measurable effects on body weights and percentage emergence of its parasitoid, *D. rapae* (Burd et al. 1996). De Cock et al. (1996) found imidacloprid to be highly toxic to *P. maculiventris*, which they contrasted with the lack of toxicity reported against another predatory stinkbug, *Perillus bioculatus* (F.) (Hough-Goldstein and Whalen 1993). Our results reflect the mixed findings of other authors. We found imidacloprid to be toxic to *A. pyralophagus*, and nontoxic

to *C. grandis* and *C. cacti*. Measurable levels of toxicity were found against *C. flavipes*.

As expected, the broad spectrum organophosphates proved toxic to most of the beneficial insects tested. Oxydemeton-methyl was toxic to *C. flavipes*. Methidathion was toxic to *A. pyralophagus* and showed residual toxicity to *C. flavipes* up to 7 d post spray. Azinphosmethyl and ethion showed residual toxicity to *C. flavipes* up to 3 and 4 d post spray, respectively. Chlorpyrifos was toxic to *C. grandis*, *C. flavipes*, and *A. pyralophagus*, but curiously, not to *C. cacti*. The fact that *C. cacti* was not adversely affected by any class of insecticide tested suggests that it is an extremely tolerant predator.

We base our conclusions upon a common experimental protocol wherein mortality was evaluated through contact with treated plant material in the vast majority of the experiments. It is possible that different concentrations or application methods, e.g. ingestion of the insecticide, may produce different results (as in De Clercq et al. 1995). In summary, our findings are consistent with those reported in the literature. The IGRs and antibiotic insecticides were benign to the nontarget insects tested and appear to be broadly compatible with biological control due to either an endemic natural enemy complex, or the introduction of parasitoids or predators. Pyridaben, imidacloprid and the pyrethroids showed mixed results and should be used with caution in an integrated control program. With the exception of *C. cacti*, the organophosphates displayed the expected toxicity to all beneficial insects tested and must be used with caution in IPM programs incorporating biological control agents.

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