Effects of Several Types of Insecticides on the Mite Predator, *Stethorus punctum* (Coleoptera: Coccinellidae), Including Insect Growth Regulators and Abamectin

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ABSTRACT Abamectin, methomyl, several types of insect growth regulators (IGRs), and several organophosphate insecticides were evaluated for toxicity to the egg, larval, pupal, and adult stages of the coccinellid mite predator, *Stethorus punctum* (LeConte). Field-collected eggs were dipped into aqueous solutions to evaluate ovicidal activity; field collected mid-instars and adults were tested by a 24-h dry film exposure method. Multiple mortality readings were done over time. Pupal mortality was evaluated in field plots that received biweekly applications. Fenoxycarb was ovicidal in the laboratory and disrupted the larval-pupal molt in the field. Abamectin was toxic to *S. punctum* larvae and adults in the laboratory and methomyl was toxic to adults. Teflubenzuron was toxic to the pupal stage in the field; along with fenoxycarb, this IGR caused late-season increases of phytophagous mite populations in field trials. Tebufenozide was not toxic to all *S. punctum* stages in the laboratory and field. *S. punctum* was tolerant to all organophosphate insecticides tested.

KEY WORDS *Stethorus punctum*, Coccinellidae, insecticides

SINCE THE INTRODUCTION of neurotoxic insecticides and acaricides in the mid-1940s, various species of tetranychid spider mites have become annual pests of orchards in virtually all fruit growing regions in North America (Tanigoshi et al. 1983). Spider mite populations in unsprayed orchards are usually regulated to low levels by a wide variety of mite and insect predators (Tanigoshi et al. 1983). Some important pests, such as the McDaniel spider mite, *Tetranychus mcdanieli* McGregor, and European red mite, *Panonychus ulmi* (Koch), are often virtually absent in unsprayed plots (Knisley & Swift 1972, Tanigoshi et al. 1983, Strickler et al. 1987). Various other phytophagous mites such as the apple rust mite, *Aculus schlechtendali* (Nalepa), are present at low densities in unsprayed orchards. Frequent applications of broad-spectrum insecticides and acaricides in commercial orchards have eliminated many phytophagous mite species and mite predators. Only a few phytophagous and predaceous mite species could adapt to these pesticides and are now commonly found in commercially treated orchards. Many orchard integrated pest management programs for spider mite control in the United States are based on the conservation of pesticide adapted predaceous mites of the Phytoseiidae, i.e., *Typhlodromus pyri* Scheuten and *Amblyseius fallacis* (Garman), and Stigmaeidae, i.e., *Zetzellia mali* (Ewing) and *Agistemus fleschneri* Summers (Croft 1990). One exception has been the conservation of an organophosphate insecticide resistant coccinellid predator, *Stethorus punctum* (LeConte), for phytophagous mite control in apple orchards of the Cumberland–Shennandoah Valley region (Hull & Beers 1985).

Control of pests that directly attack the apple fruit, i.e., *Cydia pomonella* L., *Conotrachelus nenuphar* (Herbst), and several species of leafrollers, is integrated with the biological control of the European red mite in this region by the use of organophosphates or organophosphate-carbamate mixtures applied at reduced rates as alternate-row middle sprays applied every 7–10 d (Hull et al. 1983). This integrated pest management program has reduced acaricide use in Pennsylvania by 80% since the mid-1970s and resulted in an estimated savings to growers of $20 million and a reduction of 0.8 million kilograms of pesticide (L.A.H., unpublished data). Recently, however, the tufted apple bud moth, *Platynota idaeusalis* (Walker), has become increasingly resistant to organophosphate insecticides (Knight & Hull 1989a, b; Biddinger 1993) and has become the most important direct pest of apples. Control of this species now requires multiple applications of the carbamate methomyl, which is highly toxic to *S. punctum* and disruptive to integrated pest control (David & Horsburgh 1985).

A change in orchard management to include selective insecticides, such as insect growth regulators (IGRs) or abamectin could enhance current integrated pest management programs. Abamectin is moderately selective toward phytoseiid predatory mites because of its short residual life (Graf-

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ton-Cardwell & Hoy 1983, Hoy & Cave 1985). Although its effects on predators and parasitoids have not yet been thoroughly investigated (Lasota & Dybas 1991), Morse et al. (1987) found that field rates of abamectin for mite control did not cause significant residual mortality to an aphelinid wasp parasitoid of scale nor to a coccinellid mealybug predator. Croft's (1990) extensive review of insecticidal effects on biological control agents showed that IGRs were generally safe to predaceous mites either through physiological or ecological selectivity, although some IGRs had detrimental effects on insect predators and parasitoids.

Selective insecticides would also enhance biological control of other arthropod pest species with endemic biological control agents. Presently, many biological control agents are not effective because they do not tolerate broad-spectrum insecticides. Many IGRs are specific to lepidopteran pests such as leafrollers and may allow wasp and fly predators and parasitoids for many orchard pests to become established (Reede et al. 1984, Charmillot & Brunner 1990). A total of 37 species of dipteran and hymenopteran parasitoids of *P. idaeusalis* exist in Pennsylvania apple orchards (Hull et al. 1993). In orchards in which pheromone disruption, rather than a broad-spectrum insecticide is used for tufted apple bud moth control, rates of parasitism increased from an average of 7.7% in conventionally managed orchards to 19.4% in pheromone disruption blocks over a 3-yr period (Hull et al. 1993). Similar results may be obtained with IGRs for tufted apple bud moth control, although none have yet been registered in the United States.

We examined the toxicity of abamectin and several types of IGRs to the coccinellid mite predator *S. punctum*. This predator is a key component in tree fruit integrated pest management programs for mite control in the Cumberland–Shenandoah region. Although *S. punctum* is of economic importance in a rather restricted area of the United States, it serves as a model species to measure the effects of IGRs and abamectin on other predators in many different cropping systems.

Materials and Methods

All pesticides tested were formulated product in aqueous solution. The materials were azinphosmethyl (Guthion 35 WP [wettable powder] and Guthion 3 F [flowable], Miles, Kansas City, MO); tebufenozide (RH-5992 2 F, Rohm & Haas Company, Philadelphia, PA); fenoxycarb (Insegar 25 WP, Ciba, Greensboro, NC); teflubenzuron (CME-13406 0.15 EC [emulsifiable concentrate], EM Industries, Hawthorne, NY); abamectin (Agri-Mek 0.15 EC, Merck, Sharp, & Dohme, Rahway, NJ); malathion (Malathion 0.66 EC, Ciba); diflubenzuron (Dimilin 25 WP, UniRoyal, Middlebury, CT); methomyl (Lannate 1.8 L [liquid], DuPont, Wilmington, DE); micro-encapsulated methyl parathion (Penncap M 2 FM, Atochem North America, Tifton, GA).

Ovicide Bioassay. *S. punctum* eggs were collected from experimental research plots at the Penn State Fruit Research Laboratory, Biglerville, PA, in 1992. Eggs were collected from trees that were treated only with the fungicides captan 50 WP (ICI Americas, Wilmington, DE) and benomyl 50 DF (dry flowable) (DuPont) and no azinphosmethyl applications 7 d before collection. The eggs were located on the apple leaves by using Opti-Visor (Donegan Optical, Kansas City, MO) binocular headpiece magnifiers. Leaves with eggs were picked and brought into the laboratory. The eggs were treated by dipping the leaf into aqueous solutions of each compound for 5 s. The leaves were allowed to air dry in the laboratory for 1 h and were then placed separately into plastic petri dishes (100 by 15 mm) with a damp paper towel to maintain high humidity. All dishes were placed into large plastic bags and stored at 20 ± 2°C and a photoperiod of 16:8 (L:D) h. Eggs were evaluated every 2–3 d for hatch. Larval mortality was assessed by determining if the larva was alive at the next evaluation. A randomized complete block design (i.e., five collection dates) was used. On each collection date, at least 10 eggs were treated with each pesticide; each egg served as a replicate. At least 50 eggs were treated per concentration. The concentrations selected were the proposed field rates for experimental compounds or the maximum label rate for azinphosmethyl.

Larval and Adult Bioassays. Mid-instars and adults of *S. punctum* were collected from experimental research plots at the Penn State Fruit Research Laboratory in 1991 for dry film contact bioassays in petri dishes. None of the trees used for the collection of these adults and larvae had been treated with insecticides within 10 d before collection; only the fungicides captan 50 WP and benomyl 50 DF were applied during the collection period.

The surfactant Tween-20 (polyoxy ethylene sorbitan monolaurate, ICI Americas) was added to all treatments at a rate of 300 ppm to increase adhesion of insecticides to the plastic petri dishes and to uniformly distribute the residues. All compounds were evaluated at full, half, and one-tenth of recommended field rates. One milliliter of solution was dispensed into tightly fitting clear plastic petri dishes (50 by 9 mm) with a Labsystems Finnpipette repeating dispenser (Labsystems Oy, Helsinki, Finland). The petri dishes were then closed and the solutions swirled within for 10 s. The dishes were then separated and the remaining solution was divided evenly between the top and bottom halves. The halves were allowed to air dry in the laboratory (3–4 h). During the drying period, the remaining solution was redistributed every 15 min to ensure uniform deposition of the insecticide residues. Petri dishes were either used immediately or stored in a freezer (<−5°C) for up to 3 d.

Table 1. Contact activity of various IGRs and azinphosmethyl on the eggs and hatching larvae of *S. punctum*

Treatment	Concentration, ppm	*n*	Mean % egg mortality (SD)	No. larvae that hatched	Mean % larval mortality (SD)
Azinphosmethyl 35 WP	300	50	32.0b (7.0)	34	44.1b (8.6)
Teflubenzuron 0.15 SC	30	50	20.0ab (6.0)	40	37.5b (7.8)
Tebufenozide 2 F	92	50	26.0ab (6.0)	37	27.0b (7.4)
Fenoxycarb 25 WP	40	52	51.9c (7.0)	25	36.0b (9.8)
Control	0	55	12.7a (5.0)	48	8.3a (4.0)

Means within each column followed by the same letter are not significantly different ($P < 0.05$; Fisher's Protected LSD test [Abacus Concepts 1989]).

Aspirators were used to collect adults directly from the orchard. The adults were temporarily transferred to ventilated, clear plastic containers with apple foliage for transport back to the laboratory. Mid-instars were collected in the orchard and transferred directly into treated petri dishes with camel's-hair brushes to reduce handling. Adults were transferred to treated petri dishes only in the laboratory because of their mobility. The collected larvae and treated petri dishes were stored in coolers during collection and transport. Five *S. punctum* adults or larvae were placed into each petri dish for evaluation. At least 50 adults or larvae were evaluated for each concentration in 10 replicates of five individuals. A minimum of 60 adults or larvae were used as controls in dishes treated with only 300 ppm of Tween-20. All petri dishes were held at 20 ± 2°C and a photoperiod of 16:8 (L:D) h. A second bioassay was done with adults from the same orchard. Concurrently, three to five additional concentrations of azinphosmethyl, methomyl, and abamectin and controls were tested and the results were combined with those from the previous bioassay to estimate concentration–mortality curves dose–mortality curves using POLO (LeOra Software, 1987).

After 24 h, larval or adult mortality was assessed; surviving individuals were transferred by brush to a pair of clear plastic Plantcon (Flow Laboratories, McLean, VA) plant tissue culture container bottoms (9.5 by 9.5 by 7 cm) that had been taped together to form a plastic cage. Airflow was ensured by a 2.5-cm^2 hole covered with very fine mesh cloth screen in the top. An apple spur with four to six leaves heavily infested with European red mites from an unsprayed orchard were added as food for the larvae or adults. Mortality was assessed a second time 3 d after first exposure.

Field Evaluations. Various IGRs and azinphosmethyl were applied to two tree plots each consisting of a 'Golden Delicious' and a 'Rome Beauty' tree and were replicated four times. The trees were 30 yr old and planted at a spacing of 7.3 by 10.7 m. Treatments were blocked according to a precount of the European red mite populations on the 'Golden Delicious' tree on 10 July. Treatments were applied with a truck-mounted John Bean sprayer equipped with a handgun to thoroughly wet the trees. Approximately 23 liters of spray were applied per tree (3.75 kiloliter/ha). Treatments were applied at 14-d intervals (13 and 28 July, and 11 and 25 August). Regular maintenance fungicides (captan 50 WP and benomyl 50 DF) were applied to all trees. European red mite populations were evaluated weekly by counting 25 leaves per tree (100 leaves per treatment from the four replicate 'Golden Delicious' trees only) with a leaf brushing machine (JGH. Edwards, Llanfair Orchards, RR1 Okanagan Falls, BC, Canada). The effect of treatments on *S. punctum* were evaluated only on the 'Golden Delicious' trees by separate weekly 3-min counts around the periphery of the tree for the adult, larval and pupal stages. Only pupae that had not yet eclosed were counted. Pupal mortality was evaluated by collecting leaves with ≈200 noneclosed pupae from the 'Rome Beauty' trees of each treatment on 28 August. Pupae were returned to the laboratory and maintained under natural light conditions in large plastic containers during the evaluation period. The pupae were observed every 2–3 d during a 14-d period for adult eclosion or mortality. Each pupa from each treatment, regardless of the collection tree, was considered a replicate for analysis.

Mortality and population field counts were analyzed by analysis of variance (ANOVA): treatment means were separated by Fisher's protected least significant differences (LSD) multiple range test (SuperAnova, Abacus Concepts 1989). Mean separations were done by log $(x + 1)$ transformed values.

Results

Ovicide Bioassay. The juvenile hormone analog fenoxycarb was the most toxic compound tested on *S. punctum* eggs; this IGR also affected the survival of larvae from eggs that did hatch (Table 1). Azinphosmethyl (organophosphate) was less toxic to eggs than fenoxycarb, but it also reduced survival of larvae. The chitin synthesis inhibitor teflubenzuron and the ecdysone agonist tebufenozide

Table 2. Contact activity of abamectin, various IGRs, and azinphosmethyl on field collected mid-instar *S. punctum*

Treatment	Rate, ppm	*n*	Mean % mortality	
			24 h	3 d
Azinphosmethyl 35 WP	300	50	2ab	32d
	150	50	0a	20bcd
	30	50	14bc	28cd
Tebufenozide 2 F	90	55	9ab	24bcd
	45	60	0a	12ab
	9	55	0a	15abc
Abamectin 0.15 EC	7.5	50	46e	94f
	3.75	50	34de	86f
	0.75	50	22cd	64e
Fenoxycarb 25 WP	40	50	2ab	10ab
	20	50	0a	10ab
	4	50	0a	2a
Check (Tween 20)	300	220	2ab	11ab

Means within each column followed by the same letter are not significantly different ($P < 0.05$; Fisher's protected LSD test [Abacus Concepts 1989]).

were not toxic to eggs, but larval survivorship was reduced after treatment with these compounds.

Larval Bioassay. Abamectin caused more larval mortality than in the control at most concentrations and both evaluation periods (Table 2). Abamectin was the only material that caused appreciable mortality during the 24-h exposure period. Mortality increased after the initial exposure period; by 3 d after treatment, it was extremely toxic at all rates. Probit analysis indicated that abamectin was 6.3-fold more toxic to *S. punctum* larvae 3 d after initial exposure compared with toxicity after 24 h (Table 3). Azinphosmethyl did not cause significant mortality during the 24-h exposure period, but larval survival was reduced at most concentrations by 3 d (Table 2). Fenoxycarb and tebufenozide did not reduce larval survival.

Adult Bioassay. Methomyl and abamectin caused significant adult mortality at most concentrations and at both evaluation periods (Table 4). All concentrations of both compounds were very toxic at 3 d and the highest two rates of both compounds (full and half rates) caused high adult mortality during the first 24 h. Probit analysis indicated that methomyl and abamectin continued to cause additional adult mortality after exposure ended (Table 3). Methomyl was almost 15-fold more toxic at the LC_{50} for 3 d than at 1 d and abamectin was 14-fold more toxic after 3 d than after 1 d. Of the organophosphate insecticides, only microencapsulated methyl parathion at the full and half rates caused appreciable adult mortality; this occured only after 24 h (Table 4). Diflubenzuron was slightly toxic at all concentrations after 3 d. Fenoxycarb and tebufenozide did not affect adult survivorship during the entire bioassay period. Low relative humidity in the growth chambers may have stressed the adults and contributed to the higher control mortality during the August trial.

Field Evaluations. Fenoxycarb was very toxic to *S. punctum* pupae brought into the laboratory after being treated in the field (Table 5). Teflubenzuron and azinphosmethyl were somewhat less toxic, and teflubenzuron was more toxic than azinphosmethyl. Tebufenozide did not cause pupal mortality.

Evaluations of the effects of the various compounds on *S. punctum* adult and pupal populations were inconclusive when based on field counts. Live and dead pupae were indistinguishable when populations on the trees were evaluated; movement of the highly mobile adults into and out of these small plots made evaluation of treatment effects difficult.

Table 3. Response of field-collected adult and mid-instar *S. punctum* to various insecticides after contact exposure to dry film residues for 24 h

Stage	Chemical	Time of mortality reading[a]	*n*[b]	*c*[c]	Slope ± SEM	LC_{50}, ppm (95% CL)	LC_{90}, ppm (95% CL)
Adults	Azinphosmethyl 35 WP	1 d	300	1.0	—	—	—
		3 d	300	4.0	6.85 ± 1.48	576 (494–655)	886 (743–1,600)
	Methomyl 1.8 L	1 d	300	15.5	0.63 ± 0.13	34.4 (10.9–95.2)	3,710 (646–8.6×10^5)
		3 d	300	33.7	1.53 ± 0.25	2.3 (0.6–4.5)	16.0 (8.7–44.1)
	Abamectin 0.15 EC	1 d	300	14.7	1.52 ± 0.47	6.1 (4.0–19.0)	42.4 (15.5–8,780)
		3 d	385	30.0	1.83 ± 0.23	0.43 (0.24–0.64)	2.2 (1.5–3.7)
Larvae	Abamectin 0.15 EC	1 d	200	4.0	3.04 ± 0.59	7.5 (5.6–14.5)	19.7 (11.4–114.2)
		3 d	300	16.0	1.80 ± 0.24	1.2 (0.8–1.7)	6.2 (4.1–12.0)

[a] Mortality observations made at 1 and 3 d after initial exposure.
[b] Number of individuals tested.
[c] Control mortality.
[d] 95% CL not reported because $g > 0.50$ (LeOra Software 1987).

Table 4. Contact activity of insecticides on field-collected adult of *S. punctum*

Treatment	Concentration, ppm	*n*	Mean % mortality 24 h	Mean % mortality 3 d
	July			
Azinphosmethyl 35 WP	300	50	6abc	12abc
	150	50	10abc	20bc
	30	50	8abc	14abc
Malathion 0.57 EC	1,500	50	6abc	26c
	750	50	2ab	16abc
	150	50	4abc	16abc
Tebufenozide 2 F	90	50	2ab	8ab
	45	50	10abc	10ab
	9	50	4abc	10ab
Abamectin 0.15 EC	7.5	50	72e	98c
	3.75	50	36d	98e
	0.75	50	12bc	62d
Fenoxycarb 25 WP	40	50	14c	20bc
	20	50	0a	4a
	4	50	12bc	22bc
Control (Tween 20)	300	225	6abc	12abc
	August			
Diflubenzuron 25 WP	63	50	22abc	58c
	32	50	18ab	50bc
	6	50	32bcd	52bc
Methomyl 1.8 L	270	50	98e	100d
	135	50	48d	98d
	27	50	40cd	98d
Microencapsulated methyl parathion 2 FM	300	50	24abc	46bc
	150	50	32bcd	44bc
	30	50	28abcd	40ab
Control (Tween 20)	300	60	15a	28a

Means within each column followed by the same letter are not significantly different ($P < 0.05$; Fisher's protected LSD test [Abacus Concepts 1989]).

Fenoxycarb was the only compound that reduced numbers of *S. punctum* larvae in the orchard (Fig. 1). The ovicidal activity of fenoxycarb resulted in reduced larval populations; many larvae that survived died as last instar larvae undergoing the pupal molt or as pupae (Table 5). Teflubenzuron did not appear to cause larval mortality (Fig. 1) in the field. Larval mortality caused by azinphosmethyl was not significant in the field, and counts of larvae were actually higher than the control late in the season (Fig. 1). The reductions in larval and pupal populations of *S. punctum* by fenoxycarb and the pupal mortality caused by teflubenzuron resulted in higher European red mite populations by mid-August (Fig. 2). By 14 August, mite populations were higher in these two treatments than in the tebufenozide treatment and by 24 August, mite populations were higher than in any of the other treatments.

Discussion

Organophosphate and Carbamate Insecticides. All stages of *S. punctum* were tolerant to the widely used organophosphate, azinphosmethyl. The LC_{50} of the adults was almost twice the maximum field rate (300 ppm) for this compound 3 d after initial exposure. Mortality at 24 h was low and dose–mortality regressions could not be estimated. Colburn (1971) found a much higher adult LC_{50} of 2,364 ppm in a 5-s dip evaluation with azinphosmethyl, but this difference in toxicity may have resulted from the use of different methods in the two studies. Microencapsulated methyl parathion was also slightly toxic to the adults at the higher rates and was not tested on other life stages.

Table 5. Effect of various IGRs and azinphosmethyl on laboratory reared pupae of *S. punctum* when treated as larvae in the field

Treatment	Rate, ppm	No. pupae reared	% mortality
Tebufenozide 2 F + Latron B-1956	60 312	229	24.0a
Fenoxycarb 25 WP	40	149	85.2d
Teflubenzuron 15 SC	30	212	67.9c
Azinphosmethyl 3 F	300	158	56.3b
Control		205	28.8a

Means within each column followed by the same letter are not significantly different ($P < 0.05$; Fisher's protected LSD test [Abacus Concepts 1989]).

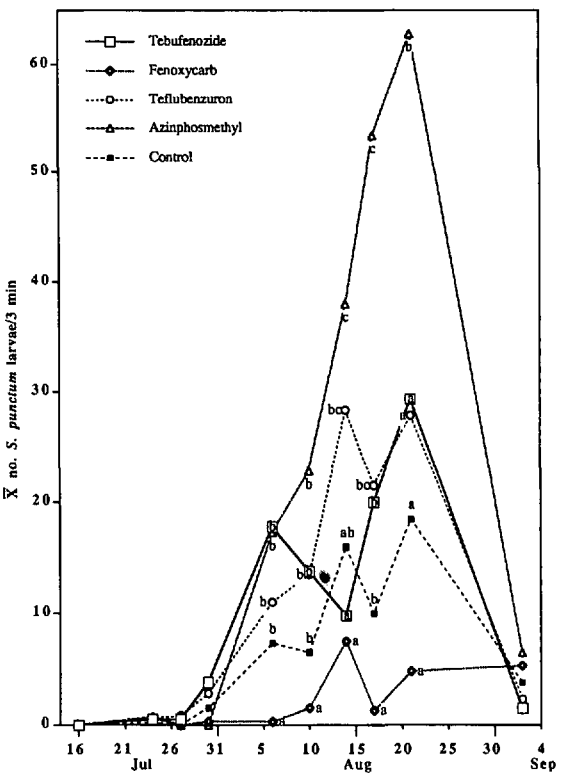

Fig. 1. Effect of insect growth regulator insecticides and azinphosmethyl on field populations of *S. punctum* larvae. Means from the same evaluation date followed by the same letter were not statistically different ($P < 0.05$; Fisher's protected LSD test [Abacus Concepts 1989]). Evaluation dates with means not followed by a letter were not significantly different from each other.

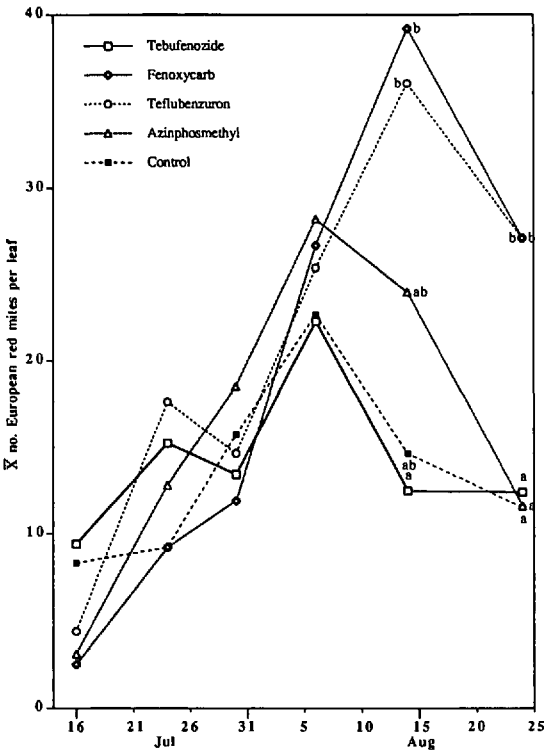

Fig. 2. Effect of insect growth regulator insecticides and azinphosmethyl on field populations of the European red mite. Means from the same evaluation date followed by the same letter were not statistically different ($P < 0.05$; Fisher's protected LSD test [Abacus Concepts 1989]). Evaluation dates with means not followed by a letter were not significantly different from each other.

The eggs and neonate larvae of *S. punctum* appear to be the most susceptible stages to azinphosmethyl. Mortality to neonate larvae probably resulted from contact with residues on the leaf surface rather than delayed mortality from exposure during the egg stage. Only a few studies have examined the ovicidal effects of insecticides on *S. punctum*. In a similar dip assay of field collected eggs, Hull & Biddinger (1991) also found that azinphosmethyl was slightly ovicidal at a field rate of 300 ppm and that survival of hatching larvae was reduced. Colburn (1972) indicated that eggs were the most susceptible stage of *S. punctum* to azinphosmethyl. His laboratory studies at 150 and 300 ppm rates indicated only a 20% reduction in egg hatch after 7 d. Other organophosphates including phosmet and phosalone were also evaluated in his study, but egg susceptibility varied with each compound.

Even though azinphosmethyl seems slightly toxic to *S. punctum* eggs and neonate larvae under laboratory conditions, our sampling techniques indicated no affect on *S. punctum* or European red mite populations in the field (Figs. 1 and 2). All *S. punctum* stages remained at levels equivalent to or higher than those found on unsprayed sprayed trees, and European red mite populations did not differ from the control trees throughout the season. The tolerance of *S. punctum* toward organophosphate insecticides in the field has been well-documented since the early 1970s (Colburn & Asquith 1970, 1971, 1973). This tolerance has resulted in a highly successful integrated pest management program for apples in Pennsylvania (Hull & Beers 1985). *S. punctillum* has also been shown to be somewhat tolerant to azinphosmethyl and other organophosphate insecticides in apple orchards in Italy (Croft 1990). *S. punctillum* did not develop resistance to broad-spectrum insecticides in parts of Europe and Canada and its importance as a predator of mites in apple has declined greatly since the introduction of these insecticides (Putnam 1955, McMurtry et al. 1970, Alford 1984). Several Australian and New Zealand species of *Stethorus* were also important predators of phytophagous mites in tree and small fruits before the introduction of organophosphate and pyrethroid insecticides (Collyer 1964, Croft 1990). Because of their extreme susceptibility to these compounds (Edwards & Hodgson 1973; Walters 1976a, b; Wearing & Thomas 1977; Bower & Kaldor 1980; Charles et al. 1985), biological control of mites now depends on laboratory selected strains of predatory mites that have been released into orchards (Croft 1990).

The carbamate methomyl was extremely toxic to adult *S. punctum* even after 24 h. Mortality increased with time, so that methomyl was almost 15-fold more lethal at the LC_{50} after 3 d compared with 1 d. Carbamates are recommended only at low rates for tufted apple bud moth control in Pennsylvania because of their extreme toxicity to *S. punctum* (Biddinger et al. 1993). Colburn & Asquith (1970) and David & Horsburgh (1985) have shown carbamate insecticides such as carbaryl, carbofuran, and methomyl to be extremely toxic to the eggs, larvae, and adults of this predator.

Insect Growth Regulators. The juvenile hormone analog fenoxycarb was the most toxic IGR to the eggs, larvae, and pupae of *S. punctum*. Fenoxycarb was ovicidal to field-collected eggs and reduced the survivorship of neonate larvae. Hull & Biddinger (1991) also found that fenoxycarb was ovicidal to *S. punctum* eggs dipped into aqueous solutions, but they did not demonstrate a reduction in larval survivorship from eggs that survived treatment. Other juvenile hormone analogs (hydroprene and methoprene) are ovicidal to *Coccinella septempuncta* L. (Kismali & Erkin 1984a, b).

Titers of juvenile hormone are very important in the growth and reproduction of insects at various stages in their development. Excess levels of juvenile hormone in the egg could block embryonic development and cause egg mortality, but residues of a juvenile hormone analog on the leaf surface should not affect young larvae upon hatching because juvenile hormone titers are already high

(Retnakaran et al. 1985). Possibly, some of these neonate larvae died from delayed sublethal effects that occurred during embryogenesis.

Fenoxycarb was not toxic to mid-instars in the laboratory, but it was very toxic to late-instars and pupae in the field. More pupae that had been continually exposed to fenoxycarb in all previous life stages died compared with those exposed to any of the compounds evaluated. Larval populations were extremely low from July to September on trees treated with fenoxycarb because of its ovicidal activity; many larvae that survived to late instar died as larval/pupal intermediates. To initiate a pupal molt, the level of juvenile hormone in the last instar must be low compared with levels of the molting hormone, ecdysone (Blum 1985). Larvae receiving an excessive dose of juvenile hormone or a juvenile hormone analog such as fenoxycarb during this stage, molt to another supernumerary larval instar or to abnormal larval/pupal intermediates. Mortality is often high, but delayed in expression in the larval and pupal stages or in the form of deformed adults (Retnakaran et al. 1985, Mauchamp et al. 1989). Kismali & Erkin (1984a, b) also found that methoprene and hydroprene to be toxic to last-instar *C. septempuncta.*

In the laboratory bioassays, adults were not killed by fenoxycarb and the number of adults in the field were generally high, probably a result of immigration from adjacent trees or orchards. Regardless of the cause of mortality, the most important effect from the standpoint of orchard pest management was the significant increase in European red mite numbers during mid- to late August. Mite populations in the control and azinphosmethyl treatments were declining because of higher *S. punctum* populations. *S. punctum* populations on trees treated with azinphosmethyl were actually higher than the control because of the lack of competition with predaceous mites that survived in the untreated trees. Hull (1988) and Jacobs et al. (1988) have also shown greatly reduced numbers of *S. punctum* larvae on trees treated with fenoxycarb in experimental field trials, in addition to late season increases of mite populations.

The chitin synthesis inhibitor teflubenzuron was not toxic to the eggs of *S. punctum,* but it reduced the survivorship of hatching neonates. Pupal mortality in the field was also higher when previous life stages had been exposed to teflubenzuron. Diflubenzuron was only slightly toxic to the adults. Weekly counts of *S. punctum* on trees treated with teflubenzuron did not indicate reduced numbers of larvae, pupae, or adults. European red mite populations on these trees continued to build late in the season, whereas treatments other than fenoxycarb caused populations to decline. This response by mites may indicate that teflubenzuron inhibits *S. punctum* development. Chitin synthesis inhibitors such as diflubenzuron and teflubenzuron show ovicidal activity toward many species of Lepidoptera (Elliot & Anderson 1982, Moffit et al. 1988), but they do not appear to be as toxic to the eggs of many species of Coleoptera (Croft 1990). Various species of *Stethorus* in Australia and New Zealand, however, are very susceptible to diflubenzuron as eggs (Wearing & Thomas 1977, Bower 1989).

The ecdysone agonist tebufenozide appears to be the least disruptive and toxic compound tested on *S. punctum.* It was nontoxic to both egg and adult stages and only slightly toxic to neonate and mid-instars. In the field, this compound did not cause additional mortality in the pupal stage and did not affect adult and larval populations. Mite populations remained at levels equal to those found in the unsprayed control throughout the season, indicating that any affects on larvae found in the laboratory are not significant in the field using current sampling methods.

Ecdysone agonists are a new class of IGRs (Aller & Ramsey 1988) about which relatively little is known. Tebufenozide induces a premature lethal molt in Lepidoptera soon after ingestion; it has some effect on selected Diptera and scale insects, but not Coleoptera (Heller et al. 1992). Its selectivity toward Lepidoptera and lack of contact activity on other insect orders and mites make this compound ideal for controlling leafroller pests like tufted apple bud moth without disrupting the biological control of mites by *S. punctum.*

Avermectins. Abamectin is an ecologically selective compound relying on a short residual and low-contact activity rather than physiological selectivity. As an agonist of the neurotransmitter GABA, it is toxic to almost all insect orders, but it is especially effective as a miticide (Lasota & Dybas 1991). Dry film bioassays, however, have shown that abamectin is extremely toxic to *S. punctum* adults and mid-instars. Abamectin continued to cause additional mortality to *S. punctum* even after the exposure period as individuals died from starvation cause by the onset of paralysis. Mid-instars were slightly less susceptible than the adults at 3 d. This difference may be caused by increased exposure through ingestion in the adults because of a tarsal and antennal grooming behavior that is not found in the larvae. Abamectin is probably less toxic to the life stages of this predator in the field because the half life of residues in sunlight is only ≈12 h (Lasota & Dybas 1991), and unsprayed refugia are available for the adults. The effect of abamectin on *S. punctum* populations in the field has been difficult to determine because of its strong miticidal activity. This activity quickly reduces mite populations which, in turn, affects the predator populations.

Our experiments illustrate the importance of determining the effects of selective insecticides such as IGRs and abamectin on potential and known biological control agents before the wide scale use of these compounds into existing crop management systems is completed. Although many of these compounds can possess greater selectivity for

certain orders of insects than most neurotoxic compounds, the various classes of IGRs and the avermectins can still exhibit serious negative effects on biological control agents like *S. punctum*. The widely differing modes of actions for many of these selective compounds may make generalizations about their effects on beneficials very difficult. Investigations on their activity against biological control agents may be needed on a case-by-case basis for each compound or class of compounds. Previous studies have shown that these compounds are almost always more toxic to biological control agents in the laboratory than in the field (Croft 1990). Final evaluation of the utility and selectivity of these compounds should, therefore, be reserved until large-scale field trials have been conducted.

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