Effects of Pesticides on Pecan Aphid Predators *Chrysoperla rufilabris* (Neuroptera: Chrysopidae), *Hippodamia convergens*, *Cycloneda sanguinea* (L.), *Olla v-nigrum* (Coleoptera: Coccinellidae), and *Aphelinus perpallidus* (Hymenoptera: Encyrtidae)

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ABSTRACT The pecan aphid predators, *Chrysoperla rufilabris* (Burmeister) (egg, larva, and adult); adult *Hippodamia convergens* (Guerin-Meneville); *Cycloneda sanguinea* (L.); *Olla v-nigrum* (Say); and pupae of the parasite, *Aphelinus perpallidus* Gahan, were tested in the laboratory for mortality to registered commercial pesticides. Concentration–response lines were estimated for *C. rufilabris* larvae and adults. Only the half and full concentrations (recommended on label for pecan) were tested on the other species. Fungicides and acaricides caused <50% mortality to all the species, indicating compatibility of the predators and the parasite with chemicals used for disease and mite control. Endosulfan and phosalone were least toxic, but none of the insecticides was safe for all of the species tested. Pyrethroids were not toxic to larvae and adult *C. rufilabris* but organophosphates and carbamates were. We observed differences in response by the egg, larva, and adult *C. rufilabris* to fenvalerate, cypermethrin, phosalone, endosulfan, lindane and dicofol. Pyrethroids were toxic to *O. v-nigrum* but phosalone, methidathion, ethion, lindane, and malathion were not. Only lindane was not toxic to adult *H. convergens*. All chemicals tested caused >70% mortality to *C. sanguinea*. Phosalone, lindane, fluvalinate, endosulfan, and azinphos-methyl were not toxic to *A. perpallidus*. Results from this study provide information about the selectivity of pesticides for integration of biological and chemical controls for pecan aphids.

KEY WORDS Insecta, pecan aphids, predators, pesticides

PECAN, *Carya illinoensis* (Wang.) K. Koch, is an important crop in the southern United States and Mexico. The average annual production of pecan in the United States from 1976 to 1980 was 91,000,000 kg with an estimated value of $127,000,000 (Dutcher 1981). Pecan, a long-season crop, requires ≈7 mo to mature. One or more of a complex of direct and indirect pests, including diseases and mites, attack pecan at most times throughout the season. Among the most important pests of pecan are the foliar-feeding aphids.

Three species of pecan aphids feed on and damage pecan. These are black pecan aphid, *Melanocallis caryaefoliae* (Davis); yellow pecan aphid, *Monelliopsis pecanis* Bissell; and blackmargined aphid, *Monellia caryella* (Fitch). Pecan aphids feed on the vascular system of the leaves and damage the leaf veins at the site of feeding; black pecan aphids kill large amounts of leaf tissue (Tedders & Thompson 1981). Pecan aphids deplete leaf carbohydrates and proteins. They also reduce leaf chlorophyll (Wood & Tedders 1982, Tedders et al. 1982, Wood et al. 1987) and net photosynthesis (Wood et al. 1985). All species cause premature defoliation (Tedders 1978), decrease tree vigor (Dutcher 1985), and reduce yield (Dutcher et al. 1984, Tedders & Wood 1985).

Traditional control of pecan aphids with pesticides has resulted in development of resistance (Dutcher & Htay 1985), outbreaks of secondary pests (Ball 1981), and resurgence of aphids and mites after the use of pesticides to control other pests (Dutcher 1983). Furthermore, many previously registered pesticides are no longer available. Clearly, alternative tactics are needed to reduce pesticide use, retain the efficacy of available pesticides, and enhance the effects of natural enemies.

Biological control of aphids in pecan appears promising (Tedders 1983, Liao et al. 1984, Edelson & Estes 1987, Mizell & Schiffhauer 1987b, Bugg & Dutcher 1989). Many natural enemies that prey upon pecan aphids and other pests may be adversely affected by pesticide use (Dutcher 1983, Dutcher & Payne 1983). However, use of pesticides cannot be totally eliminated because of the need to control pests, including aphids.

Use of selective pesticides may help conserve natural enemies. Integration of chemical and biological control is not new (Ripper et al. 1951, van den Bosch & Stern 1962, Bartlett 1964, Croft &

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Table 1. Pesticides tested with pecan aphid predators and a parasite

Pesticide and rate	Manufacturer	Class[a]	Full registered rate mg (AI)/liter[b]
Triphenyltin hydroxide 4 L	Griffin	F, OT	223
Benomyl 50 WP	E. I. Dupont de Nemours	F, CA	222
Dodine 65 WP	American Cyanamid	F, AN	577
Hexakis 4 L	E. I. Dupont de Nemours	A, OT	228
Dicofol 1.6 E	Rohm and Haas	A, CH	239
Endosulfan 3 E	FMC	I, CH	900
Lindane 1.6 E	Chevron Chemical	I, OP	736
EPN 5 E	Helena Chemical	I, OP	554
Parathion 15 WP	Woolfolk Chemical Works	I, OP	365
Diazinon AG 500	CIBA-GEIGY	I, OP	1,773
Chlorpyrifos 4 E	Dow Chemical	I, OP	1,494
Demeton formulation	ICI Americas	I, OP	99
Malathion 4.3 E	C. J. Martin	I, OP	4,029
Dimethoate 2 E	Southern Agricultural Insecticide	I, OP	296
Phosalone 3 E	Rhone-Poulenc	I, OP	444
Azinphos-methyl 50 WP	Mobay Chemical	I, OP	608
Ethion 4 E	FMC	I, OP	1,109
Fenvalerate 4 E	Shell Chemical	I, PU	46
Cypermethrin 3 E	ICI Americas	I, PU	35
Fluvalinate 2 E	Sandoz	I, PU	44
Methomyl 1.8 E	E. I. Dupont de Nemours	I, CA	244
Carbaryl 80 S	Union Carbide	I, CA	1,331
Methidathion formulation	CIBA-GEIGY	I, OP	600

[a] F, fungicide; A, acaricide; I, insecticide; OP, organophosphate; PY, pyrethroid; CA, carbamate; OT, organotin; CH, chlorinated hydrocarbon; AN, aliphatic nitrogen.
[b] Represents median concentration of active ingredient from manufacturer's suggested range for pecan.

Strickler 1983). However, data on the toxicity of pesticides to natural enemies are not available for pecan and many other crops. *Chrysoperla rufilabris* (Burmeister) is reared commercially and released by growers who wish to control aphids after control failure with conventional pesticides. Our experiments were done to determine whether predators and other natural enemies might survive pesticide applications in pecan orchards.

Materials and Methods

Pesticides. The chemicals tested, their manufacturers, and formulations are listed in Table 1.

Chrysoperla rufilabris. Eggs, larvae, and adults were obtained from Rincon-Vitova Insectaries, Oak View, Calif. The culture was initiated from collections in pecan orchards on the USDA Southeastern Fruit and Tree Nut Laboratory, Bryon, Ga. The orchards had been sprayed for several years as recommended for commercial pecan by the Georgia Cooperative Extension Service. Test individuals were approximately the second through the fifth generation reared from these field-collected insects. Eggs were placed on slides with double-sided tape and held under ambient conditions of 60–80% RH and 24–29°C. Slides were dipped for 5 s in a solution of pesticide + water + sticker (Triton 1956, Rohm & Haas, Philadelphia, Pa., at 100 ppm). Eclosion was recorded 72 h after treatment. Eight replicates of 10 eggs each were tested for each concentration along with a water + sticker control. Data were corrected for control mortality with Abbott's (1925) formula. Differences in mortality between the half and full rates for each chemical were determined by a t test ($P = 0.05$).

Concentration–responses of *C. rufilabris* larvae (first and second instars) and adults were determined as described by Grafton-Cardwell & Hoy (1985). Residues of pesticides + sticker were coated on plastic Petri dishes (adult) or microtiter plates (larva). The insects were added along with food and held at ambient conditions in the laboratory (24–31°C, 60–80% RH). Petri dishes containing the adults were inverted over tissue paper on a pegboard shelf to prevent fumigation. Eight replicates of 10 insects were tested at each concentration. Their responses were compared with responses of a control group treated with water + sticker. Further concentrations were chosen based on the response to half and full concentrations; lower or higher (minimum of four, usually more) concentrations were tested to provide a good fit of the probit model. Concentration–response lines for each pesticide were estimated with POLO (Robertson et al. 1980).

Coccinellids. Adult *Hippodamia convergens* (Guerin-Meneville) were collected from plantings of vetch and clover adjacent to the sprayed orchards previously described at Bryon, Ga. *Olla v-nigrum* (Say) and *Cycloneda sanguinea* (L.) (fewer in number) were collected from a planting of crape myrtle, *Lagerstroemia indica* L., at Monticello, Fla. All coccinellids were collected in areas near pecan orchards which had various histories of pesticide use. After collection and before testing, they were held for ≤24 h in cages with abundant aphids and a water source. Five replicates of eight

Table 2. Toxicity of pesticides to eggs of *C. rufilabris* in a laboratory bioassay

Pesticide and rate	$\bar{x}$% Mortality ± SEM
Triphenyltin hydroxide 4 L	8.7 ± 4.8
Benomyl 50 WP	20.3 ± 6.8
Dodine 65 WP	35.0 ± 7.8[a]
Hexakis 4 L	31.7 ± 4.0
Dicofol 1.6 E	40.6 ± 9.3
Endosulfan 3 E	11.3 ± 3.5
Lindane 1.6 E	42.9 ± 1.8
EPN 5 EC	86.3 ± 4.6
Parathion 15 WP	72.0 ± 7.6
Diazinon AG 500	71.3 ± 7.8[a]
Chlorpyrifos 4 E	100.0 ± 0
Demeton 6 EC	30.0 ± 4.2
Malathion 4.3 E	50.0 ± 5.0[a]
Dimethoate 2 E	53.8 ± 5.0[a]
Phosalone 3 E	25.0 ± 3.3
Azinphos-methyl 50 WP	45.0 ± 8.0
Ethion 4 EC	25.0 ± 4.2
Fenvalerate 2.4 E	90.0 ± 3.3
Cypermethrin 3 E	100 ± 0
Fluvalinate 2 E	57.9 ± 2.6
Carbaryl 80 S	72.5 ± 4.5[a]

[a] Chemicals for which mortality at half concentration was significantly lower than at full concentration as determined by a *t* test. Mortality at the half and full concentrations for all other chemicals was not significantly different.

adult coccinellids were tested for each species at the recommended full rate and half rate; responses at these concentrations were compared with responses to a water + sticker control. Plastic cups (30 ml) with tops were dipped in the pesticide + water + sticker for 5 s and dried. Adult coccinellids along with wheat and a small, moist cotton ball were then placed in each cup. The cups were ventilated on the top and bottom and held under a fume hood. This arrangement provided airflow to prevent fumigation. Mortality was assessed at 72 h after treatment. Percentage mortality was corrected for control mortality by Abbott's (1925) formula. Differences in mortality between the half and full rate for each chemical were determined by a *t* test ($P = 0.05$).

***Aphelinus perpallidus* Gahan.** Parasite pupae (aphids turn black when the parasite pupates) in mummies of the pecan aphids *Monellia caryella* and *Monelliopsis pecanis* were collected from pecan orchards at Monticello, Fla. Leaf disks bearing one mummy each were cut from the leaves with a small corkborer and affixed to slides with double-sided tape. The slides were dipped in the pesticide + water + sticker at the half or full rate as described above. Eight replicates of 10 mummies each were tested with each concentration; responses were compared with that of a water + sticker control. Mortality (based on the criterion of the presence of a parasite emergence hole in each mummy) was tallied after 7 d. Data were corrected for control mortality with Abbott's (1925) formula. Differences in mortality between the half and full rate for each chemical were determined by a *t* test ($P = 0.05$).

Results

Chrysoperla rufilabris. The fungicides triphenyltin hydroxide, benomyl, and dodine, the acaricides dicofol and hexakis, and the insecticides dimethoate, demeton, malathion, phosalone, endosulfan, azinphos-methyl, lindane, and ethion caused <50% mortality (Table 2) to eggs. Based on a comparison between the LC_{50} and the full concentration, the fungicides (except for triphenyltin hydroxide), the acaricides, and the insecticides lindane, phosalone, endosulfan, fenvalerate, cypermethrin, and fluvalinate were not toxic to the larvae. The carbamate insecticides carbaryl and methomyl and all other insecticides were highly toxic to larvae (Table 3). *C. rufilabris* adults and larvae responded to the pesticides similarly with a few exceptions (Table 4). The fungicide triphenyltin hydroxide was not toxic to adults. The insecticides endosulfan, phosalone, dicofol, and lindane were toxic to adults, but not to larvae. Pyrethroid insecticides were not toxic to either stage.

Hippodamia convergens. All pesticides were toxic to adults of this coccinellid with the exception of the fungicides benomyl, dodine, and triphenyltin hydroxide, the acaricides hexakis and dicofol and the insecticide lindane (Table 5).

Cycloneda sanguinea. Triphenyltin hydroxide caused 60% mortality to adults of this coccinellid (Table 5). Mortality from all other chemicals tested was >70%.

Olla v-nigrum. Adults of this coccinellid responded very differently from the other two species tested (Table 5). The fungicides and acaricides and the insecticides lindane, methidathion, malathion, phosalone, and ethion caused <50% mortality. Lower mortality was observed at the half rate to dimethoate, EPN, parathion and chlorpyrifos, although these compounds caused >50–100% mortality at the full rate.

Aphelinus perpallidus. The fungicides and acaricides and the insecticides azinphos-methyl, phosalone, and lindane caused <50% mortality to the pupae of the parasite (Table 5). Mortality from methomyl and carbaryl was only 57 and 51%, respectively. Only fenvalerate, phosalone, endosulfan, methomyl, and dimethoate caused significantly less mortality at half rate compared with mortality at full rate.

Discussion

Our results agree closely with those of Lawrence (1974), who applied pesticides topically to *C. rufilabris*, and with those of Grafton-Cardwell & Hoy (1985) and literature therein for *C. carnea* (Stephens). The toxicity of pesticides to *Chrysopa oculata* Say (Pree & Hagely 1985) and *C. scelestes* Banks (Krishnamoorthy 1985) has been reported, although the experimental methods were different. The responses to pesticides reported for the four species mentioned above are similar with some mi-

Table 3. Toxicity of pesticides to larvae of *C. rufilabris* in a laboratory bioassay

Pesticide and rate	*n*	Slope ± SE	LC_{50}	
			mg (AI)/liter	(95% CL)
Triphenyltin hydroxide 4 L	292	1.5 ± 0.26	101	(55.7–1,506)
Benomyl 50 WP	186	0.9 ± 0.25	46,940	(23,366–2.9 × 10^5)
Dodine 65 WP	200	—	—[a]	(—)
Hexakis 4 L	254	0.6 ± 0.11	885	(364–3,009)
Dicofol 1.6 E	200	—	—	(—)
Endosulfan 3 E	318	1.5 ± 0.23	14,776	(9,273–24,753)
Lindane 1.6 E	319	2.4 ± 0.47	2,207	(1,591–2,930)
EPN 5 E	214	3.3 ± 0.49	2.7	(2.1–3.48)
Parathion 15 WP	208	5.0 ± 1.2	9.8	(7.1–12.1)
Diazinon AG 500	169	2.8 ± 0.56	11.4	(7.8–15.1)
Chloropyrifos 4 E	328	3.3 ± 0.54	10.4	(6.6–13.9)
Demeton 6 L	280	3.2 ± 0.62	0.7	(0.42–1.0)
Malathion 4.3 E	370	3.8 ± 0.44	10.1	(6.1–13.2)
Dimethoate 2 E	229	1.6 ± 0.29	5.4	(2.9–8.3)
Phosalone 3 E	256	2.5 ± 0.76	9,287	(5,408–16,584)
Azinphos-methyl 50 WP	227	2.4 ± 0.34	4.4	(3.3–5.7)
Ethion 4 E	338	3.0 ± 0.48	1,094	(748–1,452)
Fenvalerate 2.4 E	160	1.9 ± 0.36	156	(98.9–226)
Cypermethrin 3 E	327	1.3 ± 0.39	2,211	(123–9,480)
Fluvalinate 2 E	337	0.4 ± 0.09	2,661	(810–42,869)
Carbaryl 80 S	224	2.3 ± 0.29	26.8	(17.9–39)
Methomyl 1.8 E	244	1.2 ± 0.28	59.5	(29.5–115)

[a] —, No mortality.

nor differences in the responses by specific life stages or to specific pesticides. In general, *Chrysopa* spp. and *Chrysoperla* spp. are tolerant of chlorinated hydrocarbons, pyrethroids, acaricides, and fungicides, and intolerant of carbamates and organophosphates. Of the organophosphates, phosalone is a notable exception because it is not toxic to either the larvae or adults of all four species except adult *C. rufilabris*. Carbaryl is uniformly toxic to these four Chrysopidae. *C. rufilabris* larvae and adults respond differently to methomyl and ethion, but generally exhibit similar responses to other pesticides tested. Because of their low toxicity to most life stages, phosalone and endosulfan may be used where *Chrysoperla* spp. are important predators. Based on this comparison of four species, we doubt if *C. rufilabris* has developed resistance to pesticides in pecan and therefore, we conclude that our responses reflect inherent pesticide tolerance by these species.

The coccinellids were collected from ground covers or trees that had received no recent pesticide

Table 4. Toxicity of pesticides to adult *C. rufilabris* in a laboratory bioassay

Pesticide and rate	*n*	Slope ± SE	LC_{50}	
			mg (AI)/liter	(95% CL)
Triphenyltin hydroxide 4 L	273	1.7 ± 0.27	296	(105–528)
Benomyl 50 WP	200	—	—[a]	(—)
Dodine 65 WP	237	0.7 ± 0.17	19,223	(6,316–7.8 × 10^7)
Hexakis 4 L	387	0.8 ± 0.13	6,210	(1,568–1.3 × 10^5)
Dicofol 1.6 E	200	2.1 ± 0.31	21.7	(10.9–45.7)
Endosulfan 3 E	196	1.5 ± 0.31	91.2	(18.9–173.0)
Lindane 1.6 E	398	3.8 ± 0.38	16.2	(11.8–20.5)
EPN 5 E	310	3.3 ± 0.37	0.5	(0.3–0.7)
Parathion 15 WP	200	3.1 ± 0.77	1.0	(0.21–1.5)
Diazinon AG 500	206	7.3 ± 1.5	23.0	(18.9–26.7)
Chloropyrifos 4 E	243	1.9 ± 0.27	3.6	(1.8–5.7)
Demeton 6 L	243	1.8 ± 0.28	9.8	(3.4–19.0)
Malathion 4.3 E	244	3.8 ± 0.57	36.7	(26.7–48.9)
Dimethoate 2 E	198	7.5 ± 1.2	6.3	(5.2–7.7)
Phosalone 3 E	441	1.4 ± 0.15	13.7	(3.9–29.6)
Azinphos-methyl 50 WP	248	1.3 ± 0.22	1.3	(0.24–2.5)
Ethion 4 E	362	1.3 ± 0.20	238	(58.7–528)
Fenvalerate 2.4 E	320	0.7 ± 0.14	698	(286–2,697)
Cypermethrin 3 E	307	1.3 ± 0.14	72.6	(35.9–131.2)
Fluvalinate 2 E	239	1.0 ± 0.19	841	(307–2,218)
Carbaryl 80 S	244	1.9 ± 0.37	7.5	(3.1–11.1)
Methomyl 1.8 E	280	3.2 ± 0.42	41.2	(28.1–55.7)

[a] —, No mortality.

Table 5. Toxicity of pesticides at full-registered rates to adult *H. convergens*, adult *O. v-nigrum*, adult *C. sanguinea* and pupae of *A. perpallidus* in a laboratory bioassay

Pesticide	x̄% Mortality ± SEM			
	H. convergens	*O. v-nigrum*	*C. sanguinea*	*A. perpallidus*
Triphenyltin hydroxide 4 L	0 ± 10	7 ± 11	60 ± 20[a]	0 ± 9
Benomyl 50 WP	0 ± 8	0 ± 6	*[b]	9 ± 6
Dodine 65 WP	50 ± 19[a]	0 ± 6	*	1 ± 10
Hexakis 4 L	0 ± 4	0 ± 0	*	0 ± 5
Dicofol 1.6 E	45 ± 14[a]	0 ± 4	*	0 ± 7
Endosulfan 3 E	74 ± 11	66 ± 13	70 ± 11	19 ± 6
Lindane 1.6 E	42 ± 18	0 ± 6	*	2 ± 16
EPN 5 E	94 ± 4	92 ± 6[a]	100 ± 0	85 ± 3
Parathion 15 WP	100 ± 0	63 ± 13[a]	100 ± 0	96 ± 2
Diazinon AG 500	100 ± 0	100 ± 0	100 ± 0	100 ± 0
Chlorpyrifos 4 E	100 ± 0	68 ± 11[a]	100 ± 0	94 ± 2
Malathion 4.3 E	100 ± 0	47 ± 12	100 ± 0	90 ± 3
Dimethoate 2 E	100 ± 0	84 ± 8	100 ± 0	67 ± 9
Phosalone 3 E	88 ± 3	15 ± 7	87 ± 10	52 ± 8
Azinphos-methyl 50 WP	100 ± 0	100 ± 0	*	31 ± 8[a]
Ethion 4 E	86 ± 5	0 ± 3	*	61 ± 13[a]
Fenvalerate 2.4 E	94 ± 4	97 ± 3	76 ± 11	96 ± 2
Cypermethrin 3 E	91 ± 6	100 ± 0	*[b]	74 ± 11
Fluvalinate 2 E	100 ± 0	84 ± 6	*[b]	84 ± 5[a]
Methomyl 1.8 E	100 ± 0	100 ± 0	*[b]	57 ± 7[a]
Carbaryl 80 S	100 ± 0	100 ± 0	100 ± 0	51 ± 6
Methidathion 2 E	*	45 ± 8	*[b]	86 ± 4

[a] Chemicals for which mortality at half concentration was significantly lower than at full concentration ($P = 0.05$) as determined by a t test. Mortality at the half and full concentrations for all other chemicals was not significantly different.

[b] Not tested.

treatments, although some pesticide had been used in surrounding areas. Our test populations thus had probably not developed resistance to pesticides.

Our data for *H. convergens* agree with previous laboratory research on this predator collected from peach (Moffitt et al. 1972), cotton (Wilkinson et al. 1979), and celery (Jones et al. 1983) and field research in pecan (Dutcher 1983). *H. convergens* would probably succumb when insecticides are used.

O. v-nigrum overwinters in the pecan orchard (Mizell & Schiffhauer 1987a) and is important during the entire season for aphid control. *O. v-nigrum* shows much promise for use in integrated control programs for pecan aphids. The lower mortality to *O. v-nigrum* observed at the half rate of dimethoate, EPN, chlorpyrifos, or parathion suggests that this predator may benefit from reduction in spray concentrations in an integrated approach. Unfortunately, as was also shown by Dutcher (1983), carbaryl (which was very toxic to *O. v-nigrum*) is also the most commonly used pesticide for weevil control late in the season. Therefore, use of carbaryl would disrupt aphid biological control dependent upon *O. v-nigrum* and Chrysopidae, or both.

Aphelinus perpallidus is the only known native primary parasite of pecan aphids. The *A. perpallidus* used in the tests were probably exposed to some pesticide. The parasite displayed some tolerance (50%) to carbaryl. Therefore, surviving parasites may become particularly important in the field against pecan aphids when carbaryl, which kills most of the other natural enemies, is used to control pecan weevil.

Biological control of aphids appears compatible with control of plant diseases and mites because all the species tested were tolerant to the fungicides and acaricides. Integrated use of natural enemies (particularly *C. rufilabris*, *O. v-nigrum*, and *A. perpallidus*) with pesticides for management of pecan aphids and other pests appears possible by use of selective pesticides (phosalone, endosulfan) or use of reduced concentrations of other pesticides so that at least one life stage of these natural enemies will be conserved. When specific species of predators (e.g., lacewings) are dominant in the field, application of pyrethroids may be feasible to conserve these specific predator populations and suppress aphids without side effects. Applications of reduced rates of certain pesticides for which predator mortality significantly declined at the half rate may provide control of pecan aphids as a result of the additional mortality from predation. These possibilities require further verification in the field.

Dutcher (1983) discussed aphid resurgence and Ball (1981) discussed mite outbreaks as related to pesticide toxicity to natural enemies. Currently, carbaryl is the only chemical that will control high populations of pecan weevil. Because carbaryl is toxic to most of the predators tested, its use to control pecan weevil disrupts biological control of aphids in late season. Application of either endosulfan or phosalone would conserve one or more life stages of all the natural enemies tested. These two insecticides may be used judiciously in a program to maximize the effect of biological control agents while retaining the efficacy of the chemicals and reducing total amounts of pesticide applied.

Because few pesticides are registered for use on pecan, we must retain the efficacy of available chemicals and try to manage their use to avoid development of resistance. The nature of the pest complex attacking pecan and the general lack of selectivity of the registered pecan pesticides discussed here will often make it necessary to use pesticides which are not selective of natural enemies. Therefore, we need information concerning the residual activity of chemicals on pecan. Such information would enable scheduling the safe release of natural enemies for augmentation or estimation of the mortality to immigrant natural enemies arriving in the orchard or moving into the trees from ground covers (Tedders 1983, Mizell & Schiffhauer 1987a, Bugg & Dutcher 1989) after pesticide application.

Much research needs to be done before we understand the role and management of natural enemies in pecan. Pesticide exclusion is one classical research approach (Debach et al. 1976). Pesticides which researchers might use to exclude single or multiple species of natural enemies in pecan can be gleaned from our tables. For example, malathion might be used to conserve adult *O. v-nigrum* and exclude most other predators.

We have discussed the tolerance to pesticides of at least one life stage of five important natural enemies of pecan aphids and other pests. These species were chosen because of their known importance as aphid predators and their availability for testing. As in other crops, immature stages of the coccinellid species (especially *O. v-nigrum*) and other predators (most notably the mirid, *Deraeocorus nebulosus* (Uhler)), syrphids, and other Chrysopidae and Hemerobiidae are undoubtedly adversely affected by use of pesticides in pecan. Most likely, other Chrysopidae respond to many insecticides similarly to *C. rufilabris*. The effects of pesticides on other species remain to be determined. Confirmation of our findings from field experiments is also needed.

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