

Susceptibility of *Aphis gossypii* (Glover) to Insecticides as Affected by Host Plant Using a Rapid Bioassay¹

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J. Entomol. Sci. 29(3): 289-301 (July 1994)

ABSTRACT The susceptibility of *Aphis gossypii* (Glover) reared on watermelon or cotton to seven insecticides was determined using a Petri dish bioassay. Baseline susceptibility values to each insecticide for susceptible laboratory *A. gossypii* colonies varied between host plants, but aphids reared on cotton were generally more tolerant to insecticides than aphids from watermelon. The ratio of relative susceptibility of cotton aphids to melon aphids was as much as 1000 with dimethoate or 415 with bifenthrin, however, no significant differences in susceptibility was observed with chlorpyrifos between aphid populations from the two host plants. Orders of toxicity for the seven insecticides varied between host plant, but on watermelon, the order of toxicity was bifenthrin > oxydemeton-methyl > methomyl > diclorophos > dimethoate > chlorpyrifos > endosulfan. Because of the wide range of response to insecticide doses observed with bifenthrin on melon aphid and with dimethoate and endosulfan against cotton aphid, use of the Petri dish bioassay method as a discriminating-dose field bioassay for these insecticides may not provide consistent estimations of the resistant nature of field populations. Bioassay data taken at 3 h were generally more consistent and provided a more predictive mortality model than those taken at 2 or 4 h for most insecticides. LC₅₀ values estimated for dimethoate with melon aphids using leaf-spray or leaf residue bioassays differed little from LC₅₀ values estimated with the Petri dish bioassay. Because Petri dish bioassays cost less than half as much as plant-based bioassays, provide comparable results, and require less assay time, this method is more suitable for use in monitoring for insecticide resistance in melon aphid.

KEY WORDS Bioassay, cotton, watermelon, *Aphis gossypii*, insecticide, resistance monitoring.

The melon aphid, *Aphis gossypii* Glover, is considered one of the most destructive aphids in the United States, attacking at least 64 plant species, including several cucurbit crops and cotton (Blackman and Eastop 1985). Watermelon (*Citrullus lanatus* L.) is an important cucurbit crop in the South, comprising 22,000 to 32,500 ha annually in Texas and Oklahoma and over 81,000 ha nationwide (Allred and Lucier 1990). Worldwide, melon aphid historically has been the most destructive pest of watermelons, lowering yield

¹ Accepted for publication 17 March 1994.

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and fruit quality (Cartwright 1992) and typically requiring 1-5 insecticide applications per season to prevent economic damage. Due to recent insecticidal control failures experienced nationwide with *A. gossypii* on cotton (Grafton-Cardwell 1991, Kerns and Gaylor 1992), our attention has been focused on development of resistance management strategies for this pest on watermelon. To date, *A. gossypii* infesting watermelons has not been shown to possess the wide spectrum of resistance found in *A. gossypii* infesting cotton, although control difficulties in watermelons suggest that some tolerance may exist (Cartwright, unpublished data). McKenzie et al. (1993, 1994) developed and validated a rapid bioassay method for assessing susceptibility to insecticides of *A. gossypii* on cotton. In order to adapt this bioassay method for use on watermelons and to establish basis for monitoring insecticide resistance in *A. gossypii* on watermelon, the following objectives were established for this study: 1) determine baseline toxicity levels for a laboratory population of melon aphid, 2) determine host plant effects on aphid susceptibility, 3) determine the effect of assay time on aphid mortality and efficacy of the Petri dish bioassay method, and 4) compare the Petri dish method with whole-plant assessments using dimethoate as a model of insecticide efficacy.

Materials and Methods

Insecticides. A laboratory colony of melon aphids was screened to determine susceptibility to seven commercially formulated insecticides representing organophosphate, pyrethroid, carbamate, and chlorinated bicyclic sulfite classes of insecticides. Insecticides were: bifenthrin (Capture 2 EC; FMC Corp., Philadelphia, PA), chlorpyrifos (Lorsban 4E; Dow-Elanco., Midland, MI), dicrotophos (Bidrin 8; E. I. DuPont De Nemours & Company, Wilmington, DE), dimethoate (Cygon 400; American Cyanamid Company, Wayne, NJ), endosulfan (Thiodan 3 E.C.; FMC Corp., Philadelphia, PA), methomyl (Lannate L; E. I. DuPont De Nemours & Company, Wilmington, DE), and oxydemeton-methyl (Metasystox-R; Miles Corp., Kansas City, MO). Because Kerns and Gaylor (1992) did not observe significant differences in toxicity between commercially formulated and technical grade insecticides to *A. gossypii*, we used formulated product for all bioassays.

Aphid Colony. A melon aphid colony was established in 1989 from untreated field-grown watermelons at the Wes Watkins Agricultural Research & Extension Center Lane, OK. Before testing, the colony had been held in continuous culture without introduction of a field population for over 4 years. The melon aphid colony was reared on watermelon cv. 'Jubilee' and maintained in an environmentally controlled culture room at $27 \pm 5^\circ\text{C}$ and a photoperiod of 16:8 (L:D). Plants were grown under greenhouse conditions without foliar insecticide applications before transfer to screened culture cages in the culture room. However, the greenhouse was fumigated during the growing season with dithio insecticidal smoke (Fulex; Fuller System Inc., Woburn, MA) at 7-10 day intervals to avoid introduction of "wild" aphids on culture plants. Fresh watermelon plants were added weekly. Old plants were removed when the aphids had moved to the new plants.

Petri Dish Bioassay Method. Commercially formulated insecticides were dissolved in denatured proprietary ethanol (95.5%) to make desired stock solutions based on each

insecticide's formulation. Aphids were tested with a wide range (0.001-1000 ppm) of log concentrations to determine the mortality response range. Serial dilutions of 6-8 concentrations targeted at producing mortality between 2 and 99% were used to define the response range. Dosages producing 0 to 100% mortality were considered to be outside the effective response range of the aphid. Serial dilutions were in increments of either 33% for insecticides that elicit a narrow response range or 75% for insecticides that elicit a broader response range from aphids. Plastic Petri dishes (50 mm diam) with tight fitting lids were used to prevent aphid emigration. Petri dishes were treated with 500 μ l of each concentration of insecticide applied to the inside of both the lid and bottom dish (1 ml total per dish). Three dishes per concentration were treated and then gently rotated to deposit residue evenly over all surfaces. Petri dishes were placed in a fume hood for ca. 2 h to allow the ethanol to completely evaporate.

Determination of Petri Dish Baseline Toxicity Levels. Apterous adult aphids of similar size and age were transferred with a soft camel hair brush from culture plants to petri dishes. Aphids were carefully removed from glabrous cotyledons because it was more difficult to transfer aphids without damage from true leaves which were more hairy. Twenty to 30 aphids per dish were used for each concentration starting with the untreated control and loading in ascending order of concentration. Sufficient numbers of alate aphids to bioassay a full range of concentrations for all insecticides were difficult to obtain. Therefore, only dimethoate was used with the Petri dish bioassay to compare the effective response range for alate and apterous adult aphid forms reared on watermelon. Alate aphids were handled the same as in the Petri dish bioassay with the exception of reducing the number of aphids per dish to ± 10 aphids. When all dishes had been loaded, any aphids killed by handling were removed, beginning with the control. Aphid mortality was assessed at 2, 3, and 4 h after treatment under an industrial fluorescent magnifying glass. Aphids were considered dead when no movement was detected after the aphid had been gently probed with a camel hair brush. Bioassays were replicated at least 5 times for each insecticide to produce sufficient observations to obtain stable probit curves. Failure of 95% fiducial limits (FL) to overlap was used as the criterion for identifying significant differences among LC_{50} values of replicated tests for each insecticide and between LC values of different host plants for each insecticide. Tests in which mortality exceeded 5% in control dishes were not included in analyses. Data from replicated tests with overlapping FLs for LC_{50} values were pooled for probit analyses. The total sample size for each insecticide tested for pooled probit analyses ranged from 1849 to 3613 for watermelon and 1859 to 4154 for cotton. Relationships between mortality and concentration of insecticide were evaluated by probit analysis (Sparks and Sparks 1987).

Host Plant Influences on Aphid Susceptibility. The relative susceptibility of *A. gossypii* to each of seven insecticides using the Petri dish bioassay was calculated by dividing the LC_{50} value from a susceptible Texas A&M University laboratory colony reared on cotton (McKenzie et al. 1993) by the LC_{50} value from a laboratory colony of *A. gossypii* reared on watermelon.

Effects of Assay Time on Aphid Mortality and Petri Dish Bioassay Efficacy. Regression analysis was used to determine relationships between % mortality calculated at 2 or 3 h and dose of Petri dish bioassay for each insecticide (SAS Institute Inc. 1988).

Comparison of Plant-based Bioassay Methods. Leaf residue activity was compared with the Petri dish method in a greenhouse study. Watermelon (cv. 'Jubilee') plants were transplanted into 15.0 cm pots and maintained in the greenhouse at $27 \pm 5^\circ\text{C}$. Plants were at the first true leaf stage when the bioassay was initiated. An experimental unit consisted of one watermelon plant covered with a ventilated cylindrical cage made of polycarbonate plastic. Serial dilutions of 6 dimethoate concentrations known to produce an effective response range with the Petri dish bioassay were used to treat watermelon plants. Each concentration was replicated 3 times, with one plant representing a replicate. Dimethoate was dissolved in distilled water to make the desired concentration range. All insecticide treatments were applied with a spray bottle until the watermelon leaves were thoroughly covered and began to drip (approx. 60 ml per plant). Controls were sprayed with distilled water only. Plants were allowed to dry for 1-2 h. Mixed aphid forms from the laboratory population were transferred to watermelon plants after the plants had dried. Thereafter, the aphids were handled as in the laboratory bioassay. Times were noted after all replicates of each concentration had been infested. Aphid mortality in the highest concentration was assessed every hour until $> 50\%$ mortality was observed. At that time, initial aphid counts for apterous adult, alate nymph and alate aphids for all concentrations were taken and were repeated at twice the initial aphid count time.

In the greenhouse, contact activity of dimethoate also was compared with the Petri dish method in a manner similar to the leaf residue method. Dimethoate was chosen for comparison because we felt that if differences were going to occur between LC estimations, this insecticide would be the best candidate for discrepancies in methods because it showed the greatest difference between host plants. However, in this experiment, aphids were allowed to colonize bioassay plants before dimethoate was sprayed. Plants, aphids, and mortality counts were handled similarly to the leaf residue bioassay using the same serial dilutions to treat the watermelon plants. Prior to spraying, watermelon plants were infested with a mixed sample of aphid forms and allowed to acclimate to the plant for 1-2 h. All insecticide treatments were applied with a spray bottle until the watermelon leaves were thoroughly covered and began to drip (approx. 60 ml per plant). Controls were sprayed with distilled water only. Times were noted after all replicates of each concentration had been sprayed. Mortality was assessed at 6 and 12 h. Failure of 95% fiducial limits (FL) to overlap was used as the criterion for identifying significant differences between LC values for time and aphid forms for both plant-based bioassays. Relationships between mortality and concentration of insecticide were evaluated with probit analysis (Sparks and Sparks 1987).

Results and Discussion

Determination of Petri Dish Baseline Toxicity Levels. Bifenthrin was the most toxic to *A. gossypii* reared on watermelon, closely followed by oxydemeton-methyl, methomyl, and dicrotophos (LC_{50} values ranged from 0.28-0.51 ppm) (Table 1). Although bifenthrin was the most toxic to melon aphid, the aphid's response to bifenthrin also spanned the broadest range of concentrations (slope

= 0.66 ± 0.02) suggesting toxicity of formulated bifenthrin is variable among aphids reared on watermelon when tested with this bioassay method. Biologically, the slope of the probit regression line estimates the changes in activity per unit change in concentration. Use of formulations that elicit a flat slope in the bioassay severely limit the validity of the results because FL of LC values are inversely related to slope (low slope = wide FL and broad response range). For example, although the LC₅₀ FLs for watermelon and cotton do not overlap, the LC₉₀ FLs for watermelon are so wide (4.06-1,716) they encompass the FLs for cotton (193.50-259.19) which diminishes the bioassay's ability to predict accurate bifenthrin lethal concentrations for melon aphid. When cotton was used as the host, bifenthrin toxicity could more precisely be estimated because the aphid responded to a much narrower range of concentrations (slope = 4.68 ± 0.23) with tightened corresponding FLs for both LC values presented. Apparently, bifenthrin formulation is not the most crucial factor contributing to the flat slope associated with testing *A. gossypii* reared on watermelon using this bioassay. Response ranges for chlorpyrifos, dimethoate, dicotophos, endosulfan, methomyl, and oxydemeton-methyl were narrower than bifenthrin, consequently corresponding slopes were higher, ranging from 2.17 to 7.23, suggesting that the accuracy of the Petri dish bioassay in predicting lethal concentrations for these insecticides is increased. For field monitoring purposes, insecticides generating slopes >2 (bifenthrin for melon aphid; dimethoate and endosulfan for cotton aphid) may not be conducive to this bioassay and the accuracy of a discriminating dose differentiating between susceptible and potentially resistant *A. gossypii* may be compromised.

Host Plant Influences on Aphid Susceptibility. Studies reported by McKenzie et al. (1993) of *A. gossypii* reared on cotton using the Petri dish bioassay were conducted simultaneously with the present study. Responses of susceptible laboratory populations of *A. gossypii* to seven insecticides appear to be mediated by the host plant (Table 1). *A. gossypii* LC values for each insecticide were significantly different for cotton and watermelon for all insecticides bioassayed with the exceptions of methomyl LC₉₀ value for watermelon and LC₅₀ value for cotton, bifenthrin LC₉₀ values and chlorpyrifos LC values which were found to be equally toxic to both cotton and melon aphid (FL overlap). *A. gossypii* reared on watermelon was innately more susceptible to insecticides than *A. gossypii* reared on cotton, with 3.7, 4.5, 20, 61, 415, and 1000 times more susceptibility detected between baseline LC₅₀ values to methomyl, dicotophos, endosulfan, oxydemeton-methyl, bifenthrin, and dimethoate, respectively. The order of insecticide toxicity was also influenced by host plant. Insecticides in descending order of toxicity when aphids were reared on watermelon were as follows: bifenthrin > oxydemeton-methyl > methomyl > dicotophos > dimethoate > chlorpyrifos > endosulfan. In contrast, the order of insecticide toxicity when cotton was the host were as follows: methomyl > dicotophos > chlorpyrifos > oxydemeton-methyl > bifenthrin > endosulfan > dimethoate. Thus, toxicity to *A. gossypii* appears to be strongly influenced by the host plant, but the level of host plant influence depends upon the individual insecticide and not necessarily the insecticide class (apterous adult aphid responses to OP insecticides varied from not significantly different to 1000-fold difference in relative susceptibility).

Table 1. Response of susceptible laboratory populations of adult apterous *A. gossypii* to seven insecticides as mediated by host plant using a Petri dish bioassay.

Insecticide	Host*	n	Slope \pm SE	LC ₅₀ (95% FL)**	LC ₉₀ (95% FL)**	RS†	OT‡
Bifenthrin	WM	3613	0.66 \pm 0.02	0.28 (0.08-1.14)	24.46 (4.06-1,716)	415	1
	COT	2456	4.68 \pm 0.23	116.06 (104.75-126.55)	218.05 (193.50-259.19)		5
Chlorpyrifos	WM	3040	2.27 \pm 0.10	14.96 (14.06-15.99)	54.77 (47.65-64.45)	NSD	6
	COT	3675	3.05 \pm 0.09	13.90 (11.98-16.03)	36.55 (29.80-48.76)		3
Dicrotophos	WM	1846	7.23 \pm 0.32	0.51 (0.41-0.58)	0.76 (0.66-0.97)	4.5	4
	COT	1859	3.08 \pm 0.15	2.30 (1.93-2.68)	6.00 (4.80-8.52)		2
Dimethoate	WM	2193	2.24 \pm 0.11	4.28 (4.00-4.55)	15.93 (14.09-18.45)	1000	5
		171§	4.69 \pm 0.58	3.16 (2.67-3.65)	5.92 (5.03-7.38)	1355	
	COT	2151	1.62 \pm 0.11	4,282 (3,693-5,143)	26,467 (18,838-41,056)		7
Endosulfan	WM	1849	3.01 \pm 0.15	117.81 (111.48-124.88)	313.77 (280.32-358.74)	20	7
	COT	3622	1.02 \pm 0.04	2,362 (1,072-12,535)	42,087 (9,138-3,483,749)		6

Table 1. Continued.

Insecticide	Host*	n	Slope \pm SE	LC ₅₀ (95% FL)**	LC ₉₀ (95% FL)**	RS†	OT ‡
Methomyl	WM	2469	2.17 \pm 0.11	0.44 (0.35-0.52)	1.71 (1.36-2.37)	3.7	3
	COT	2807	4.15 \pm 0.13	1.63 (1.42-1.85)	3.32 (2.84-4.12)		1
Oxydemeton-methyl	WM	2446	4.24 \pm 0.20	0.30 (0.26-0.33)	0.59 (0.51-0.73)	61	2
	COT	4154	2.48 \pm 0.09	18.40 (14.96-23.44)	60.63 (41.93-116.57)		4

* WM = watermelon cv. 'Jubilee', COT = cotton cv. 'Stoneville'; Data for aphids reared on cotton was adapted from McKenzie et al. (1993).

** LC values expressed in ppm active ingredient of formulated insecticide; mortality was calculated at 3 h.

† RS = Relative susceptibility was calculated by dividing the LC₅₀ obtained from a susceptible Texas A&M University laboratory colony of *A. gossypii* reared on cotton by the LC₅₀ obtained from a laboratory colony of *A. gossypii* reared on watermelon; NSD indicates estimated LC₅₀ values from each host plant are not significantly different (FL overlap).

‡ OT = Order of insecticide toxicity for each host plant as indicated by LC₅₀ values.

§ Only adult alate forms of *A. gossypii* were tested with the Petri dish bioassay.

Mounting evidence that responses of phytophagous insects to pesticides are significantly affected by host plant continue to accumulate (Moldenke et al. 1992, Robertson et al. 1990, Seigfried and Mullin 1989, Yu et al. 1979). Juneja and Sharma (1973) reported the relative susceptibility and relative toxicity of *A. gossypii* to each of ten insecticides varied considerable when reared on six cucurbitaceous hosts: bottle gourd (source population), pumpkin, round gourd, cucumber, watermelon, and muskmelon. Furk and Vedjhi (1990) found all populations of *A. gossypii* reared on chrysanthemum survived a discriminating dose of pirimicarb which was highly toxic (100% mortality) to *A. gossypii* reared on cucumber. In Japan, pesticide susceptibilities of *A. gossypii* also varied significantly from different host plants (chrysanthemum, aubergine, strawberry, and cucumber) and correlations between high esterase activity and organophosphate resistance were found (Inoue 1987). Saito (1991) found *A. gossypii* infesting Cucurbitaceae (melon and cucumber) showed higher enzyme activity than aphids reared on Solanaceae (eggplant and potato) crops, however host plants did not affect aphid aliesterase activity when transferring populations from one host plant to another. Elevated esterase activity has been correlated to insecticide resistance and apparently the cotton aphid utilizes multiple mechanisms of insecticide resistance (Sun et al. 1987, Takada and Murakami 1988, O'Brien et al. 1992), even within a single class of insecticide (Kerns and Gaylor 1992). Currently, the cotton aphid has developed tolerance to all major classes of insecticides and mechanisms conferring insecticide resistance have been associated with acetylcholinesterase insensitivity (Silver 1984), altered levels of carboxylesterases, gene amplification (O'Brien et al. 1992), mutations in the germ line cell, and dissociations or rearrangements of chromosomes (Furk and Vedjhi 1990). Inferences made from these studies suggest various concentrations of insecticides depending upon the susceptibility to each insecticide and each host plant may prove economical in the control of aphids on watermelon and cotton when applied judiciously.

Effects of Assay Time on Aphid Mortality and Petri Dish Bioassay Efficacy. Generally, Petri dish mortality assessed at 3 h was the most consistent; mortality assessed at 2 h was less consistent, and if the bioassay was extended to 4 h (data not presented), mortality often exceeded 5% in untreated dishes. Regression analysis of mortality data calculated at 3 h gave reduced CV values for all seven insecticides bioassayed and greater R^2 values where generally observed, compared with R^2 values calculated for data taken at 2 h (Table 2). A minimal decrease in the CV observed of mortality data calculated at 3 h for oxydemeton-methyl and dicrotophos corresponded with slight decreases R^2 values; therefore, reducing the bioassay time to 2 h may be appropriate, considering the minimal difference in bioassay accuracy realized for the additional assay time for these insecticides.

Comparison of Plant-based Bioassays. A good relationship was observed between the direct leaf spray and leaf residue bioassay when determining the toxicity of dimethoate to melon aphid (Table 3). No significant differences were detected between LC_{50} values of plant-based methods at 6 or 12 h after treatment and apterous adult or mixed aphid forms, however significant differences were detected between time periods for both plant-based methods. Estimates of LC_{50} values for dimethoate using the Petri dish bioassay were not significantly different from LC_{50} values calculated at 12 h using a direct leaf spray or leaf

Table 2. Regression of % mortality calculated at 2 or 3 h and dose of Petri dish bioassay for seven insecticides.

Insecticide	Hour	Regression Equation	CV	R ²	F*	df
Bifenthrin	2	% mortality = 34.69 + 0.31 (± 0.05)* dose	68.9	0.18	36.2	1,160
	3	% mortality = 45.01 + 0.36 (± 0.06)* dose	58.8	0.20	40.0	1,160
Chlorpyrifos	2	% mortality = -2.23 + 1.32 (± 0.07)* dose	79.4	0.72	403.8	1,157
	3	% mortality = -0.63 + 3.03 (± 0.08)* dose	36.1	0.89	1268.6	1,157
Dicrotophos	2	% mortality = 13.10 + 35.12 (± 2.25)* dose	45.3	0.66	243.8	1,124
	3	% mortality = 35.98 + 29.36 (± 2.47)* dose	38.3	0.53	141.5	1,124
Dimethoate	2	% mortality = -0.48 + 2.27 (± 0.20)* dose	82.4	0.54	125.6	1,106
	3	% mortality = 12.41 + 6.39 (± 0.31)* dose	30.6	0.80	434.8	1,106
Endosulfan	2	% mortality = -1.58 + 0.16 (± 0.01)* dose	77.6	0.66	184.7	1,94
	3	% mortality = 5.11 + 0.26 (± 0.01)* dose	45.3	0.77	317.8	1,94
Methomyl	2	% mortality = 14.80 + 25.81 (± 1.81)* dose	43.8	0.64	202.4	1,115
	3	% mortality = 26.82 + 29.67 (± 2.03)* dose	35.6	0.65	212.9	1,115
Oxydemeton-methyl	2	% mortality = 7.26 + 42.43 (± 2.44)* dose	43.4	0.76	301.4	1,94
	3	% mortality = 37.39 + 36.79 (± 3.68)* dose	40.2	0.51	99.7	1,94

* F values calculated for each regression were significant at *P* < 0.0001 level.

Table 3. Effect of time and aphid form on toxicity of dimethoate to *A. gossypii* reared on watermelon using plant-based bioassays.

Bioassay Method	Hour	Aphid Form Ratio*	n	Slope ± SE	LC ₅₀ (95% FL)**	LC ₉₀ (95% FL)**
Leaf Spray	6	100:0:0	421	1.78 ± 0.28	12.97 (10.23-19.06)	67.89 (38.06-197.18)
		91:5:4	462	2.41 ± 0.26	11.68 (9.89-14.58)	39.76 (28.23-66.38)
	12	100:0:0	421	1.60 ± 0.24	5.23 (4.29-6.37)	33.24 (21.11-74.66)
		91:5:4	462	1.69 ± 0.23	5.52 (4.63-6.63)	31.76 (21.03-63.41)
Leaf Residue	6	100:0:0	307	2.13 ± 0.32	9.82 (7.98-13.34)	39.28 (24.69-89.68)
		89:8:3	345	1.92 ± 0.29	9.48 (7.68-12.91)	44.23 (26.82-109.10)
	12	100:0:0	307	2.20 ± 0.31	5.39 (4.53-6.47)	20.66 (14.67-36.89)
		89:8:3	345	1.91 ± 0.28	5.32 (4.42-6.47)	24.85 (16.85-48.45)

* Aphid form ratio = % apterous adult: % alateform nymph: % alate forms of total *A. gossypii* used to calculate mortality.

** LC values expressed in ppm active ingredient of commercially formulated dimethoate.

residue method. Plant-based methods were nearly identical with LC_{50} values separated by only 0.16 and 0.20 ppm for apterous adult aphid and mixed aphid forms, respectively. For comparing Petri dish bioassay predictions of dimethoate toxicity to melon aphid, 12 h appeared to be the most appropriate time for correlating mortality to plant-based bioassay methods.

In comparing the response of only apterous adult aphids with the response of a mixed population of apterous adult (89-91% of total aphids), alate nymph (5-8%) and alate aphids (3-4%) tested with dimethoate, no significant differences were observed between aphid forms for either plant-based bioassay method. Comparison of melon aphid forms tested with the Petri dish method determined alate adults were significantly less tolerant to dimethoate than were apterous adult aphids reared on watermelon. In contrast, Grafton-Cardwell (1991) found alate nymphs and adults infesting cotton grown in California to be generally more tolerant to pesticides tested with a discriminating dose dipped-leaf contact bioassay. Although differences may occur when alate and apterous forms are tested separately with dimethoate using the Petri dish bioassay, no differences are apparent when mixed populations were compared between plant-based bioassays with apterous aphids alone. Bioassays based only on apterous aphids, which are easier to obtain in culture and from field collections, appear to be representative of mixed populations treated with dimethoate.

The Petri dish bioassay was relatively inexpensive. Although materials and labor may vary, supplies averaged \$6.50 per bioassay and labor averaged 3.5 h to complete a bioassay, including Petri dish preparation time. The plant based bioassays cost more than twice the amount in supplies (\$14.25) and labor (7.5 h). Supply costs was calculated on those items that were not reuseable. Labor was calculated on the amount of time required to perform each task and did not consider time between tasks. The Petri dish bioassay was economical, reliable and yielded quick results and could easily be incorporated into IPM programs.

By developing a rapid insecticide resistance bioassay system which can be used to predict insecticide efficacy against *A. gossypii* Glover both in cotton and melons, selection of insecticide products can be made based on known susceptibility of aphids present in individual fields. These data provide an estimate of the relative susceptibility of watermelon and cotton aphids using a Petri dish bioassay method. Discriminating doses extrapolated from LC_{90} values of the Petri dish bioassay could be used to monitor the status of *A. gossypii* susceptibility to various insecticides in watermelon and cotton and provide a more complete basis for categorizing insecticides for their resistance status. In addition, knowledge of how the susceptibility of aphids to insecticides is affected by host plant will provide a basis for a comprehensive resistance management strategy especially where cotton and melons are grown in close proximity. Identification of field populations of aphids in which the bioassay indicates potential resistance could greatly improve the selection of insecticides by predicting *a priori* the expected efficacy and delay the development of insecticide resistance by reducing repeated exposure to ineffective insecticides. The ease of using the Petri dish bioassay lends itself for use in field monitoring of insecticide resistance and could improve control of this pest, improve crop profitability and help eliminate unnecessary insecticide use.

Acknowledgments

The technical assistance by R. Sheffield and M. Cook and the secretarial assistance of Kelly James Miller is gratefully acknowledged. Approved for publication by the Director, Oklahoma Agricultural Experiment Station. This research was supported under Hatch Project #OKLO-2040 and USDA-CSRS-Southern Region IPM Grant Program #91-34103-5844.

References Cited

- Allred, A. J. and G. Lucier.** 1990. The U. S. Watermelon Industry. USDA-ARS, Staff Rep. No. AGES-9015. 73 pp.
- Blackman, R. L. and V. F. Eastop.** 1985. Aphids on the World's Crops: An Identification Guide. John Wiley and Sons, New York. 466 pp.
- Cartwright, B.** 1992. Assessment of damage to watermelons by *Aphis gossypii* (Homoptera: Aphididae). In: proceedings XIX International Congress of Entomology. p. 385.
- Furk, C. and S. Vedjhi.** 1990. Organophosphorus resistance in *Aphis gossypii* (Homoptera: Aphididae) on chrysanthemum in the UK. Ann. Appl. Biol. 116: 557-561.
- Grafton-Cardwell, E. E.** 1991. Geographical and temporal variation in response to insecticides in various life stages of *Aphis gossypii* (Homoptera: Aphididae) infesting cotton in California. J. Econ. Entomol. 84(3): 740-749.
- Inoue, M.** 1987. Relationship between esterase activity and susceptibility to organophosphorus insecticides of the cotton aphid. Japanese J. Applied Entomol. and Zool. 31: 404-406.
- Juneja, V. K. and J. C. Sharma.** 1973. Effect of host plants on the susceptibility of *Aphis gossypii* Glover to certain insecticides. Indian J. Entomol. 35: 179-186.
- Kerns, D. L. and M. J. Gaylor.** 1992. Insecticide resistance in field populations of the cotton aphid (Homoptera: Aphididae). J. Econ. Entomol. 85 (1): 1-8.
- McKenzie, C. L., B. Cartwright, and M. Karner.** 1993. Validation of a field bioassay to assess insecticide resistance in the cotton aphid. In Proc. Beltwide Cotton Conferences, 46th Cotton Insect Research and Control Conference. 2: 747-750.
- McKenzie, C. L., B. Cartwright, M. Karner, and C. T. Allen.** 1994. Progress on validation of field bioassay to predict efficacy of insecticides against cotton aphid. In Proc. Beltwide Cotton Conferences, 47th Cotton Insect Research and Control Conference. 2: (In press).
- Moldenke, A. F., R. E. Berry, J. C. Miller, R. G. Kelsey, J. G. Wernz, and S. Venkateswaran.** 1992. Carbaryl susceptibility and detoxication enzymes in gypsy moth (Lepidoptera: Lymantriidae): Influence of host plant. J. Econ. Entomol. 85: 1628-1635.
- O'Brien, P. J., Y. A. Abdel-Aal, J. A. Ottea, and J. B. Graves.** 1992. Relationship of insecticide resistance to carboxylesterases in *Aphis gossypii* Glover (Homoptera: Aphididae) from mid-South cotton. J. Econ. Entomol. 85(3): 651-657.
- Robertson, J. L., K. F. Armstrong, D. M. Suckling, and H. K. Preisler.** 1990. Effects of host plants on the toxicity of azinphosmethyl to susceptible and resistant light brown apple moth (Lepidoptera: Tortricidae) J. Econ. Entomol. 83: 2124-2129.
- Saito, T.** 1991. Insecticide resistance of the cotton aphid, *Aphis gossypii* Glover (Homoptera: Aphididae). V. Relationship between host preference and organophosphorus resistance. Japanese J. Applied Entomol. and Zool. 35: 145-152.
- SAS Institute Inc.** 1988. SAS/STAT User's Guide, Release 6.03 Edition. Cary, NC: SAS Institute Inc., pp. 1028.
- Seigfried, B. D. and C. A. Mullin.** 1989. Influence of alternative host plant feeding on aldrin susceptibility and detoxification enzymes in western and northern corn rootworms. Pestic. Biochem. Physiol. 35: 155-164.

- Silver, A. R. J.** 1984. The biochemical nature of primicarb resistance in two glasshouse strains of *Aphis gossypii* Glover. Ph.D. Thesis, University of Reading, UK.
- Sparks, T. C. and A. Sparks.** 1987. Micro Probit. DowElanco, Greenfield, IN.
- Sun, Y-Q., G-L. Feng, J-G Yuan, P. Zhu, and K-Y Gong.** 1987. Biochemical mechanism of resistance of cotton aphid to organophosphorus insecticides. Acta Entomol. Sin. 30: 13-20.
- Takada, H. and Y. Murakami.** 1988. Esterase variation and insecticide resistance in Japanese *Aphis gossypii*. Entomol. Exp. Appl. 48: 37-41.
- Yu, S. J., R. E. Berry, and L. C. Terriere.** 1979. Host plant simulations of detoxifying enzymes in a polyphagous insect. Pestic. Biochem. Physiol. 12: 380-384.
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