Inheritance of Resistance to Pyriproxyfen in *Bemisia tabaci* (Hemiptera: Aleyrodidae) Males and Females (B Biotype)

DAVID W. CROWDER,[1] CHRISTA ELLERS-KIRK, CHRISTINE M. YAFUSO, TIMOTHY J. DENNEHY, BENJAMIN A. DEGAIN, VIRGINIA S. HARPOLD, BRUCE E. TABASHNIK, AND YVES CARRIÈRE

Department of Entomology, University of Arizona, Tucson, AZ 85721–0036

J. Econ. Entomol. 101(3): 927–932 (2008)

ABSTRACT We evaluated effects of the insect growth regulator pyriproxyfen on *Bemisia tabaci* (Gennadius) (B biotype) (Hemiptera: Aleyrodidae) males and females in laboratory bioassays. Insects were treated with pyriproxyfen as either eggs or nymphs. In all tests, the LC_{50} for a laboratory-selected resistant strain was at least 620 times greater than for an unselected susceptible strain. When insects were treated as eggs, survival did not differ between males and females of either strain. When insects were treated as nymphs, survival did not differ between susceptible males and susceptible females, but resistant males had higher mortality than resistant females. The dominance of resistance decreased as pyriproxyfen concentration increased. Resistance was partially or completely dominant at the lowest concentration tested and completely recessive at the highest concentration tested. Hybrid female progeny from reciprocal crosses between the susceptible and resistant strains responded alike in bioassays; thus, maternal effects were not evident. Rapid evolution of resistance to pyriproxyfen could occur if individuals in field populations had resistance with traits similar to those of the laboratory-selected strain examined here.

KEY WORDS *Bemisia tabaci*, pyriproxyfen, insecticide resistance, haplodiploid, insect growth regulator

Pyriproxyfen has been used since 1996 in Arizona cotton (*Gossypium* spp.) to manage the B biotype of the sweetpotato whitefly, *Bemisia tabaci* (Gennadius) (Hemiptera: Aleyrodidae) (Dennehy et al. 1996, Palumbo et al. 2001). It is a juvenile hormone mimic that inhibits embryogenesis and adult emergence (Ishaaya and Horowitz 1992, Horowitz and Ishaaya 1994). Pyriproxyfen use in Arizona has reduced applications of broad-spectrum insecticides, and it has helped restore the profitability of cotton production (Dennehy and Williams 1997, Ellsworth and Martinez-Carillo 2001). Pyriproxyfen use has also aided in the conservation of natural enemies that were negatively affected by broad-spectrum insecticides (Naranjo et al. 2004).

Resistance to pyriproxyfen was detected in the Q biotype of *B. tabaci* after 5 yr of use in cotton fields in Israel, despite limits of one application per season (Denholm et al. 1998, Horowitz et al. 1999). Although no pyriproxyfen failures have been documented in Arizona fields, laboratory bioassays over 11 yr show that resistance levels are increasing in certain regions (Li et al. 2003; Dennehy et al. 2004; unpublished data). Consistent with trends seen in Arizona and Israel, simulation models show that resistance to pyriproxyfen by *B. tabaci* in Arizona may arise in 10–20 yr (Crowder et al. 2006).

B. tabaci is haplodiploid, with males produced from unfertilized haploid eggs and females produced from fertilized diploid eggs (Byrne and Devonshire 1996). In haplodiploid insects, if males and females are equally susceptible to pesticides, the frequency of resistance alleles can build up more rapidly in males than females (Denholm et al. 1998, Carrière 2003, Crowder et al. 2006). Under these conditions, resistance evolves faster than in diploid pests (Denholm et al. 1998, Carrière 2003, Crowder et al. 2006). Resistance can be slowed when resistant males have higher mortality than resistant females (Carrière 2003, Crowder et al. 2006). Several studies have examined resistance to pyriproxyfen by the B and Q biotypes of *B. tabaci* (Horowitz and Ishaaya 1994; Horowitz et al. 1999, 2002, 2003, 2005; Li et al. 2003; Dennehy et al. 2004), yet none have directly compared the response of males and females.

Here, we evaluated effects of pyriproxyfen on survival by males and females of *B. tabaci* (B biotype) from a susceptible strain and a laboratory-selected resistant strain, as well as hybrid female progeny from reciprocal crosses between the strains. Insects were exposed to pyriproxyfen as either eggs or nymphs. Dominance of resistance was measured at each of several concentrations tested.

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[1] Corresponding author, e-mail: dcrowder@ag.arizona.edu.

0022-0493/08/0927–0932$04.00/0

Materials and Methods

Strains. We used a susceptible strain of the B biotype of *B. tabaci* derived from a cotton field in Yuma, AZ, in August 2004 (Yuma 04-S). Since August 2004, the Yuma 04-S strain had been reared in the laboratory on cotton plants without exposure to pesticides. We also used a laboratory-selected resistant strain of the B biotype derived from cotton fields in Queen Creek, AZ, in October 2002 (QC-02). Two selections with 0.1 μg/ml pyriproxyfen in the subsequent 6 mo resulted in a 1,000-fold increase in resistance to pyriproxyfen in this strain (Dennehy et al. 2004). Since April 2003, the QC-02 strain had been reared on cotton plants sprayed with 1.0 μg/ml pyriproxyfen.

Insect Types. We tested five types of *B. tabaci*: susceptible (Yuma 04-S) males and females, resistant (QC-02) males and females, and hybrid females from reciprocal crosses between the strains. Virgin females and males of both strains were sexed and isolated as pupae (Horowitz et al. 2003) with a small piece of surrounding leaf. Each pupa was placed individually in a 10-ml scintillation vial containing a leaf disk on agar. The sex of emergent adults was confirmed under a microscope. Unmated females from both strains produced only male progeny, whereas mated females produced male and female progeny. For the reciprocal crosses, 10 virgin females (susceptible or resistant) were placed in 20-ml scintillation vials containing agar and a leaf disk to mate with 15 males of the other strain for 2 d. These crosses produced males (susceptible or resistant) and hybrid females.

Bioassays. Two types of bioassays were conducted with pyriproxyfen: egg bioassays and nymph bioassays. For the egg bioassays, excised cotton seedlings (15–25 cm in height) with one true leaf were dipped for 20 s in various concentrations of formulated pyriproxyfen (Knack 0.86 EC; Valent USA, Walnut Creek, CA) (0–320 μg/ml). Ten virgin females from both strains or 10 mated pairs (Yuma 04-S [susceptible] and QC-02 [resistant]), Yuma 04-S ♀ × QC-02 ♂ and QC-02 ♀ × Yuma 04-S ♂) were aspirated into modified petri dishes containing a seedling. The stems of the seedlings extended out of a small hole in the petri dish, and the roots were held in water. Seedlings thus arranged were held in growth chambers (27°C, 50% RH, and a photoperiod of 16:8 [L:D] h) for females to lay eggs for 48 h. After 48 h, adults were removed, eggs were counted, and seedlings were inserted individually into 20-ml scintillation vials containing tap water. To assess mortality, live nymphs were counted 7 d after oviposition, and eclosed adults were collected 20 and 25 d after oviposition. Adults were frozen and sexed the day they were counted.

For the nymph bioassays, 10 virgin females from either strain or 10 mated pairs (see below) laid eggs for 48 h on untreated excised seedlings with one true leaf, after which they were removed and eggs counted. Insects were reared in growth chambers for 11 d (until all individuals surviving on seedlings were second or third instar). Then, seedlings were dipped for 20 s in formulated pyriproxyfen (0–100 μg/ml). To assess mortality, pupae were counted and sexed 15 and 20 d after oviposition, and adults were collected and sexed 20 and 25 d after oviposition as described above.

Egg bioassays were conducted in October 2005 and March 2007; nymph bioassays were conducted in January 2006 and April 2007. The Yuma 04-S ♀ × QC-02 ♂ cross was done only in October 2005 and January 2006. The QC-02 ♀ × Yuma 04-S ♂ cross was done only in March and April 2007. For each bioassay, three to six replicates were performed with each pyriproxyfen concentration.

Data Analysis. When progeny included both sexes, the number of eggs of each sex was estimated by correcting for the sex ratio of adult progeny from controls (Horowitz et al. 2003). The number of male nymphs was estimated based on the estimated number of male eggs and their average survival under the appropriate treatment. The estimated number of male nymphs was subtracted from the total number of nymphs to estimate the number of female nymphs.

We used two-way analysis of variance (ANOVA) to compare mortality across dates for the four insect types that were tested in two separate months (susceptible males, susceptible females, resistant males, and resistant females). Month, pyriproxyfen concentration, and the interaction of month and concentration were factors in the ANOVA. With any insect type or bioassay, neither month nor the interaction of month and concentration was significant ($\alpha = 0.05$) (data not shown). Thus, for all subsequent analysis, data were pooled across dates.

Probit analysis (PROC PROBIT, SAS Institute 2002) was used to estimate slopes of the concentration–mortality lines and their standard errors, as well as the LC_{50} values and their 95% fiducial limits. Mortality observed at each pyriproxyfen concentration was corrected for control mortality (Abbott 1925). Resistance ratios of each insect type were calculated as their LC_{50} divided by the LC_{50} of the susceptible strain for the same sex. LC_{50} values were considered significantly different if their 95% fiducial limits did not overlap.

A two-way ANOVA on transformed mortality data (arcsine square root of mortality) was used to compare adjusted mortality of the sexes of each strain. Sex, pyriproxyfen concentration, and the interaction of sex and concentration were factors in the analysis. A separate ANOVA was performed for each assay and for each strain. For the egg and nymph bioassays, mortality in the treated stage (egg or nymph) was similar to mortality from egg to adult (one-way ANOVA; $P > 0.10$ for all comparisons). Thus, all results reported are lifetime mortality (egg to adult) to facilitate comparisons between the bioassays.

Dominance of resistance (h) at each concentration was calculated as follows: $h = (W_h - W_s)/(W_r - W_s)$, where W_s, W_r, and W_h are the survival of susceptible, resistant, and hybrid females, respectively (Liu and Tabashnik 1997). When $W_s \leq W_h \leq W_r$, h ranges from 0 to 1, with $h = 0$ indicating recessive resistance and $h = 1$ dominant resistance. We use the terms partially recessive ($0 < h < 0.5$) and partially dominant ($0.5 < h < 1$). We used regression (PROC GLM, SAS Insti-

Table 1. Response to pyriproxyfen for five insect types in egg bioassays

Insect type	n^a	Slope (SE)	LC_{50} (95% FL)[b]	RR[c]
Susceptible male	3,348	0.61 (0.060)	0.0059 (0.0029–0.011)	1.0
Susceptible female	3,072	0.65 (0.070)	0.0053 (0.0027–0.0093)	1.0
Resistant male	4,104	1.0 (0.12)	4.0 (2.4–5.9)	670
Resistant female	4,039	1.3 (0.11)	3.3 (2.4–4.4)	620
Hybrid female (a)[d]	1,574	1.2 (0.16)	0.50 (0.27–0.84)	94
Hybrid female (b)[e]	496	0.87 (0.13)	0.44 (0.21–0.85)	83

[a] Number of insects.
[b] Units are micrograms of pyriproxyfen per milliliter of deionized water.
[c] Resistance ratios were calculated by dividing the LC_{50} of an insect type by the LC_{50} of the susceptible strain of the same sex.
[d] Hybrid female (a) were progeny of Yuma 04-S (susceptible) ♀ × QC-02 (resistant) ♂.
[e] Hybrid female (b) were progeny of QC-02 (resistant) ♀ × Yuma 04-S (susceptible) ♂.

tute 2002) to determine whether h varied linearly with the logarithm of concentration.

Results

Egg Bioassays. The laboratory-selected QC-02 strain was resistant to pyriproxyfen, with resistance ratios of 620 for females and 670 for males relative to the susceptible Yuma 04-S strain (Table 1; Fig. 1a). The LC_{50} of hybrid females between reciprocal crosses did not differ significantly (Table 1; Fig. 1a).

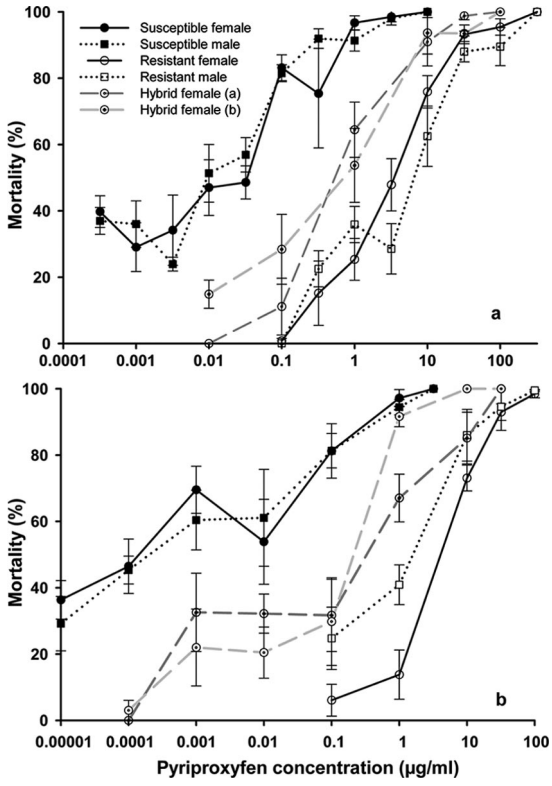

Fig. 1. Mortality caused by pyriproxyfen (±SE) for five insect types in egg (a) and nymph (b) bioassays. Hybrid female (a) were progeny of Yuma 04-S (susceptible) ♀ × QC-02 (resistant) ♂. Hybrid female (b) were progeny of QC-02 ♀ × Yuma 04-S ♂.

Mortality in the susceptible and resistant strains was affected by concentration, but in both strains the interaction of sex and concentration was not significant (Table 2). Mortality caused by pyriproxyfen did not differ significantly between susceptible males and susceptible females or between resistant males and resistant females (Tables 1 and 2).

Nymph Bioassays. The laboratory-selected QC-02 strain was resistant to pyriproxyfen, with resistance ratios of 110,000 for females and 8,100 for males relative to the susceptible Yuma 04-S strain (Table 3; Fig. 1b). The LC_{50} of hybrid females from reciprocal crosses did not differ significantly (Table 3; Fig. 1b). The LC_{50} for susceptible insects and resistant males were lower than for *B. tabaci* treated as eggs.

Mortality caused by pyriproxyfen was affected by concentration in both strains, but the interaction of sex and concentration was not significant (Table 2). Mortality caused by pyriproxyfen did not differ significantly between susceptible males and susceptible females (Tables 2 and 3). Mortality of resistant males was higher than resistant females (Tables 2 and 3).

Dominance of Resistance. In each bioassay, dominance of resistance (h) decreased as pyriproxyfen concentration increased (Fig. 2). In all cases, resistance was partially or completely dominant at the lowest concentration tested and completely recessive at the highest concentration tested (Fig. 2).

Discussion

Using the pyriproxyfen-resistant QC-02 strain of *B. tabaci*, we found here that resistant males treated as nymphs had higher mortality than resistant females across all pyriproxyfen concentrations (Table 2; Fig. 1b), although resistant males were highly resistant to pyriproxyfen compared with susceptible males (Table 3). Differences in susceptibility between haploid males and diploid females are expected if detoxification enzymes play a role in resistance and dosage compensation is imperfect (Carrière 2003). This suggests that metabolic resistance contributes to pyriproxyfen resistance in the QC-02 strain. Other studies have shown that detoxification enzymes contribute to pyriproxyfen resistance in the Q biotype of *B. tabaci* (Devine et al. 1999) and in house flies, *Musca domestica* L. (Zhang et al. 1997; 1998).

Table 2. Two-way ANOVAs to compare adjusted mortality between males and females of the susceptible (Yuma 04-S) or resistant (QC-02) strain in the egg and nymph bioassays

Strain	Bioassay	ANOVA factor								
		Sex			Concn			Sex × concn		
		F	df	*P*	*F*	df	*P*	*F*	df	*P*
Susceptible	Egg	0.03	1,74	0.87	52	1,74	<0.0001	0.10	1,74	0.76
	Nymph	0.20	1,86	0.69	42	1,86	<0.0001	0.0090	1,86	0.93
Resistant	Egg	0.20	1,92	0.64	43	1,92	<0.0001	0.030	1,92	0.86
	Nymph	5.90	1,65	0.018	61	1,65	<0.0001	1.9	1,65	0.20

Sex, pyriproxyfen concentration, and the interaction of sex and concentration were factors in the analyses. A separate ANOVA was conducted for each strain and each bioassay.

Dominance of resistance decreased as pyriproxyfen concentration increased, such that resistance was totally recessive at high concentrations (Fig. 2). This is consistent with other studies that have analyzed the impact of insecticide concentration on dominance (Liu and Tabashnik 1997; Tabashnik et al. 2002, 2004, 2005; Carrière 2003; Alves et al. 2006). In the Q biotype of *B. tabaci*, resistance to pyriproxyfen was estimated to be partially recessive ($h = 0.26$) (Horowitz et al. 2003). Nevertheless, resistance in the QC-02 strain was not recessive in the field (Crowder et al. 2007). This suggests that concentrations expressed in the field are too low, or residue deposition too discontinuous, to cause recessive inheritance.

Hybrid female progeny from reciprocal crosses responded alike in bioassays (Fig. 1), suggesting that maternal effects were not associated with resistance in the QC-02 strain. Similarity of F_2 male progeny of virgin hybrid females provided further evidence that maternal effects are not associated with resistance in QC-02 (unpublished results). Maternal effects also did not contribute to resistance in the Q biotype of *B. tabaci* (Horowitz et al. 2003). Data for hybrid males were not evaluated because differences in their response are difficult to interpret, as they could be due to variation in male or mother genotype. However, these data suggest that mortality caused by pyriproxyfen for hybrid male progeny of Yuma 04-S ♀ × QC-02 ☼ did not significantly differ from susceptible (Yuma 04-S) males (as expected under haplodiploidy). Similarly, mortality caused by pyriproxyfen for hybrid male progeny of QC-02 ♀ × Yuma 04-S ♂ did not differ significantly from resistant (QC-02) males.

Susceptibility to pyriproxyfen was lower for eggs than nymphs for the B biotype here, as reported previously for the Q biotype of *B. tabaci* (Horowitz et al. 1999). These results may indicate that pyriproxyfen is more effective against *B. tabaci* nymphs than eggs. However, the difference between egg and nymph bioassays may also be attributable to methodology. Eggs were indirectly exposed to pyriproxyfen, as pyriproxyfen had to move from treated leaves via the egg pedicel. In contrast, nymphs were contacted directly by pyriproxyfen because leaves with nymphs were dipped in a pyriproxyfen mixture. Moreover, in field experiments where relatively low concentrations of pyriproxyfen were applied, no differences in susceptibility occurred between individuals treated as eggs or nymphs (Crowder et al. 2007). These data support the hypothesis that methodology contributed to the higher mortality in nymph bioassays seen here.

As described above, results from laboratory and field bioassays were not always similar. When possible, laboratory and field results should be compared with obtain a better understanding of factors affecting resistance evolution (Dennehy and Granett 1984, Farnham et al. 1984, Roush and Miller 1986, Cahill et al. 1996). Levels of mortality and dominance observed here at pyriproxyfen concentrations between 0.1 and 1.0 μg/ml were similar to those in field experiments using the same strains of *B. tabaci* (Crowder et al. 2007). Thus, future laboratory experiments using concentrations in this range may best approximate field conditions.

In egg and nymph bioassays, mortality in the treated stage was similar to mortality from egg to adult. Thus,

Table 3. Response to pyriproxyfen for five insect types in nymph bioassays

Insect type	n^a	Slope (SE)	LC_{50} (95% FL)[b]	RR[c]
Susceptible male	3,128	0.43 (0.060)	1.1×10^{-4} (2.3×10^{-5}–3.3×10^{-4})	1.0
Susceptible female	2,168	0.37 (0.047)	2.9×10^{-5} (7.3×10^{-6}–8.1×10^{-5})	1.0
Resistant male	3,781	0.90 (0.074)	0.89 (0.58–1.3)	8,100
Resistant female	2,122	1.2 (0.12)	3.2 (2.1–4.6)	110,000
Hybrid female (a)[d]	1,173	0.52 (0.094)	0.31 (0.094–1.4)	11,000
Hybrid female (b)[e]	982	0.68 (0.14)	0.090 (0.028–0.41)	3,100

[a] Number of insects.
[b] Units are micrograms of pyriproxyfen per milliliter of deionized water.
[c] Resistance ratios were calculated by dividing the LC_{50} of an insect type by the LC_{50} of the susceptible strain of the same sex.
[d] Hybrid female (a) were progeny of Yuma 04-S (susceptible) ♀ × QC-02 (resistant) ♂.
[e] Hybrid female (b) were progeny of QC-02 (resistant) ♀ × Yuma 04-S (susceptible) ♂.

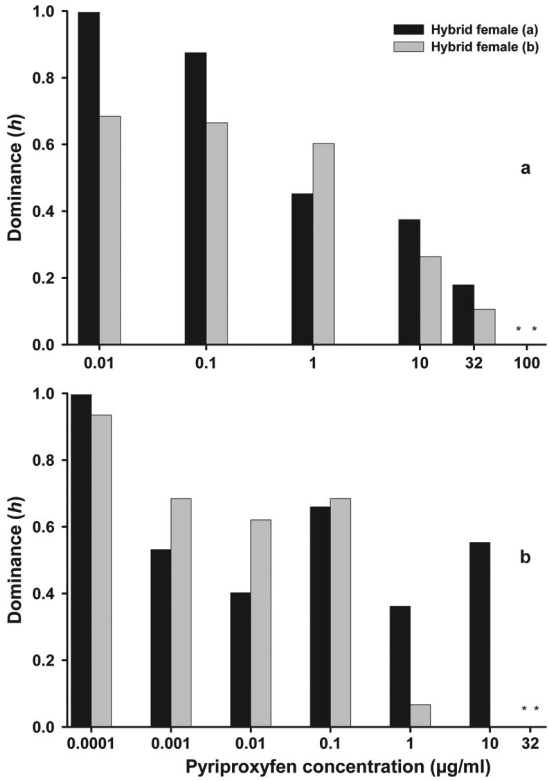

Fig. 2. Dominance of resistance (h) in egg (a) and nymph (b) bioassays. Hybrid female (a) were progeny of Yuma 04-S (susceptible) ♀ × QC-02 (resistant) ♂. Hybrid female (b) were progeny of QC-02 ♀ × Yuma 04-S ♂. For hybrid female (a), dominance varied with concentration as follows: $h = 0.54–0.25 \times \log[\text{concn}]$ in egg bioassays ($t_5 = -10.75$, $P = 0.0004$, $r^2 = 0.97$); $h = 0.38–0.11 \times \log[\text{concn}]$ in nymph bioassays ($t_6 = -2.4$, $P = 0.061$, $r^2 = 0.54$). For hybrid female (b), dominance varied with concentration as follows: $h = 0.43–0.19 \times \log[\text{concn}]$ in egg bioassays ($t_5 = -4.32$, $P = 0.013$, $r^2 = 0.82$); $h = 0.24–0.19 \times \log[\text{concn}]$ in nymph bioassays ($t_6 = -5.9$, $P = 0.0019$, $r^2 = 0.88$). Values of $h = 0$ are indicated with asterisks.

in egg bioassays, measuring lifetime mortality in addition to egg mortality did not provide additional insight on the effects of pyriproxyfen. This suggests that the common practice of measuring only egg mortality in pyriproxyfen bioassays is appropriate (Ishaaya and Horowitz 1992, 1995; Horowitz and Ishaaya 1994; Horowitz et al. 1999, 2002, 2003, 2005; Li et al. 2003; Dennehy et al. 2004). This also suggests that pyriproxyfen did not have any adverse effects on nymphs before the pupal stage, as reported previously (Ishaaya and Horowitz 1992, 1995). However, measuring lifetime mortality was appropriate to better compare mortality between *B. tabaci* treated as eggs and nymphs.

Models suggest that resistance can be greatly delayed in haplodiploid pests if resistant males have higher mortality than resistant females, refuges where insecticides are not used are present, and resistance is nearly recessive (Carrière 2003, Crowder et al. 2006). This study showed that resistance was recessive with high pyriproxyfen concentrations and resistant male nymphs had higher mortality than resistant female nymphs. Nevertheless, pyriproxyfen applications in the field did not produce these favorable conditions (Crowder et al. 2007), suggesting that evolution of pyriproxyfen resistance could be relatively rapid under current whitefly management conditions (Crowder et al. 2006). It is unclear whether application methods could be modified to favorably modify the phenotypic attributes of pyriproxyfen resistance in the field. Experience with the Q biotype in Israel has shown that resistance is a threat to the sustainability of pyriproxyfen (Denholm et al. 1998; Horowitz et al. 1999, 2005). Further analyses of the mechanisms and intensity of resistance in the QC-02 strain and other field populations could help to improve the current Arizona whitefly control program and reduce this threat.

Acknowledgments

We thank Kuanie Julin and Ann Showalter for preparing equipment for the bioassays. We thank to Rami Horowitz and Shai Morin for providing helpful comments on the manuscript. The research was funded in part by the United States Environmental Protection Agency under the Science to Achieve Results Graduate Fellowship Program, grant FP-91648901-0, an Achievement Rewards for College Scientists scholarship, and National Research Initiative Competitive Grants Program grant 2005-00925.

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Received 10 August 2007; accepted 26 January 2008.