Field Evaluation of Resistance to Pyriproxyfen in *Bemisia tabaci* (B Biotype)

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ABSTRACT We determined effects of aerial sprays of the insect growth regulator pyriproxyfen on sweetpotato whitefly, *Bemisia tabaci* (Gennadius) (B biotype), in Arizona cotton (*Gossypium* spp.) fields. We measured survival for males and females from a susceptible strain and a laboratory-selected resistant strain, as well as for hybrid female progeny from crosses between the strains. Insects were exposed directly to pyriproxyfen sprays in the field or indirectly in the laboratory by rearing them on sprayed leaves collected from the field. In all tests, survival was higher for the resistant strain than the susceptible strain, but did not differ between sexes in each strain. Survival to the adult stage did not differ between eggs and nymphs directly exposed to sprays. For susceptible and hybrid individuals, survival was lower on leaves collected the day of spraying than on leaves collected 2 wk after spraying. In contrast, survival of resistant individuals did not differ based on the timing of exposure. Dominance of resistance to pyriproxyfen depended on the type of exposure. Resistance was partially or completely dominant in direct exposure bioassays and on leaves collected 2 wk after spraying ($h > 0.6$). Resistance was partially recessive on leaves collected the day of spraying (mean $h = 0.34$). Rapid evolution of resistance to pyriproxyfen could occur if individuals in field populations with traits similar to those of the laboratory-selected strain examined here were treated intensively with this insecticide.

KEY WORDS *Bemisia tabaci*, pyriproxyfen, insecticide resistance, haplodiploid, genetics

The sweetpotato whitefly, *Bemisia tabaci* (Gennadius), is a serious pest in subtropical regions throughout the world (Byrne and Bellows 1991, Brown et al. 1995, Oliveira et al. 2001). The B biotype of this pest was introduced to the United States at the end of the 1980s (Brown et al. 1995; Ellsworth and Martinez-Carillo 2001), and it is a key pest of cotton (*Gossypium* spp.) and other crops. Chemical control is often required to manage *B. tabaci* (Horowitz and Ishaaya 1996; Palumbo et al. 2001).

Pyriproxyfen is a biorational insecticide that has been used for over a decade in Arizona cotton for management of *B. tabaci* (Dennehy and Williams 1997, Ellsworth et al. 2006). It is an insect growth regulator (IGR) that inhibits egg hatch and adult eclosion (Ishaaya and Horowitz 1992). Pyriproxyfen use has reduced dependence on broad-spectrum insecticides, preserved beneficial species, and increased farmer profits (Ellsworth and Martinez-Carillo 2001; Naranjo et al. 2004). Although no pyriproxyfen failures have been documented in Arizona fields, laboratory bioassays over 11 yr reveal an areawide decline in susceptibility to pyriproxyfen in the B biotype of *B. tabaci* (Li et al. 2003; Dennehy et al. 2004; unpublished data).

B. tabaci reproduces by arrhenotoky with males produced from unfertilized haploid eggs and females from fertilized diploid eggs (Byrne and Devonshire 1996). Modeling results suggest that, as in diploid insects, resistance evolution in haplodiploid insects such as *B. tabaci* depends on the survival of resistant individuals relative to susceptible individuals and the dominance of resistance (Carrière 2003, Crowder et al. 2006). Modeling results also suggest that, in haplodiploid insects, differences in susceptibility between males and females could affect the rate of resistance evolution (Carrière 2003; Crowder et al. 2006). Although the level and dominance of resistance to pyriproxyfen have been tested with laboratory bioassays of the Q and B biotypes of *B. tabaci* (Horowitz et al. 2003; unpublished data), field evaluations of pyriproxyfen resistance in *B. tabaci* have not been reported previously.

Here, we used field experiments to measure some important factors influencing evolution of resistance to pyriproxyfen in the B biotype of *B. tabaci.* Arizona cotton fields were sprayed aerially with pyriproxyfen

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to assess survival of males and females from a susceptible strain and a laboratory-selected resistant strain as well as survival of hybrid female progeny from crosses between the two strains. Insects were exposed directly to pyriproxyfen sprays in the field or exposed indirectly by rearing them on sprayed leaves collected from the field. The results show nonrecessive inheritance of resistance to pyriproxyfen that could favor rapid evolution of resistance in the field.

Materials and Methods

Strains. We used a susceptible strain of *B. tabaci* collected from a cotton field in Yuma, AZ, in August 2004 (Yuma 04-S). Since August 2004, the Yuma 04-S strain has been reared in the laboratory on cotton plants without exposure to pesticides. We also used a laboratory-selected resistant strain collected 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 has been reared on cotton plants sprayed with 1.0 μg/ml pyriproxyfen.

Insect Types. We tested five types of *B. tabaci:* susceptible females, susceptible males, resistant females, resistant males, and hybrid females produced by crosses between the two strains. We did not test progeny from the reciprocal cross in this field evaluation, because results from laboratory bioassays showed no differences between progeny of reciprocal crosses for the B biotype (unpublished data) or the Q biotype of *B. tabaci* (Horowitz et al. 2003). Virgin females and males of both strains were sexed and isolated as pupae (Horowitz et al. 2003). After sexing, pupae were removed with a small piece of surrounding leaf. Each pupa was individually placed in a 10-ml scintillation vial containing agar and a leaf disk. After eclosion, the sex of adults was confirmed under a microscope. For the bioassays, the progeny of virgin females were all males, whereas mated females produced male and female progeny. We also tested the progeny from a cross between susceptible females and resistant males. For this cross, five newly emerged susceptible females were placed in 20-ml scintillation vials and allowed to mate with 8–10 resistant males for 2 d. The progeny from this cross were susceptible males and hybrid females.

Field Sites. Two cotton fields in Marana, AZ, were used for the bioassays. Cotton plants were ≈1 m high when sprays occurred. Fields were sprayed by a commercial applicator attached to a fixed wing airplane between 0500 and 0600 hours. Field 1 was sprayed on 18 July 2006. Field 2 was sprayed on 25 July 2006. For both fields, the area sprayed was 18 by 403 m, along an edge of the field, representing 441 rows. Fields were sprayed using formulated pyriproxyfen (Knack 0.86 EC, Valent USA, Walnut Creek, CA). The concentration applied was 0.58 liters per ha in a volume of water of 28 liters per ha (per label instructions).

Direct Exposure Bioassays. Concurrent assays were conducted to test the response of eggs and nymphs to direct exposure with pyriproxyfen. For the egg exposure assay, 3 d before a field was sprayed, five virgin females (susceptible or resistant) or five mated pairs (susceptible, resistant, or susceptible × resistant) were aspirated into petri dishes containing an excised untreated cotton seedling (15–25 cm in height) with one true leaf to lay eggs for 48 h in growth chambers (27°C, 50% RH, and a photoperiod of 16:8 [L:D] h). After 48 h, adults were removed, eggs were counted, and seedlings were inserted individually into waterpiks containing deionized water. The following day, between 0100 and 0400 hours, the waterpiks containing seedlings were attached to wooden stakes and placed 1–5 cm below the uppermost leaves of the cotton canopy. Within 1 h after fields were sprayed, seedlings were brought to the lab and placed in 20-ml scintillation vials. To assess mortality, live nymphs were counted 7 d after seedlings were collected, and adults were counted 20 and 25 d after seedlings were collected. Adults were frozen and sexed the day they were counted.

The methods for the nymphal exposure assay were the same, except for the following modifications: the 48-h oviposition period in growth chambers began 13 d before fields were sprayed. After oviposition, eggs were counted and seedlings were held in growth chambers for 11 d, at which point all individuals surviving on seedlings were second or third instars. Seedlings were placed in the field and collected as described above. Adults were counted and sexed as described above.

Both direct and indirect exposure assays were conducted in treated fields 1 and 2, described above. For field 1, a portion of an adjacent cotton field 200 m outside of the sprayed area was used as the control. For field 2, an unsprayed portion of the field 150 m outside of the sprayed area was used as the control. For both assays, eight replicates of each insect type were placed in each treated field (five insect types × replicates per type = 40 seedlings total) and four replicates of each type were placed in each unsprayed (control) field. In each treated field, seedlings were randomly distributed in 10 evenly spaced rows, with four seedlings containing eggs or nymphs in each row (Fig. 1). For the controls, seedlings were randomly placed in two rows (Fig. 1). In both treated and control fields, seedlings were spaced 3 m from each other and at least 4.5 m from field edges.

Indirect Exposure Bioassays. For indirect exposure bioassays, cotton leaves were sampled from the field at two time intervals: the day fields were sprayed and 2 wk after fields were sprayed. Leaves were removed at the fourth terminal node from plants in treated and control fields (Ellsworth et al. 1999). Leaves were removed from plants next to seedlings for the direct exposure bioassays. Leaves were taken to the lab within 1 h, and they were trimmed to an area of ≈12 cm^2 (Ellsworth et al. 1999). The trimmed area was examined under a microscope and all visible organisms were removed. These clean leaves were placed in petri

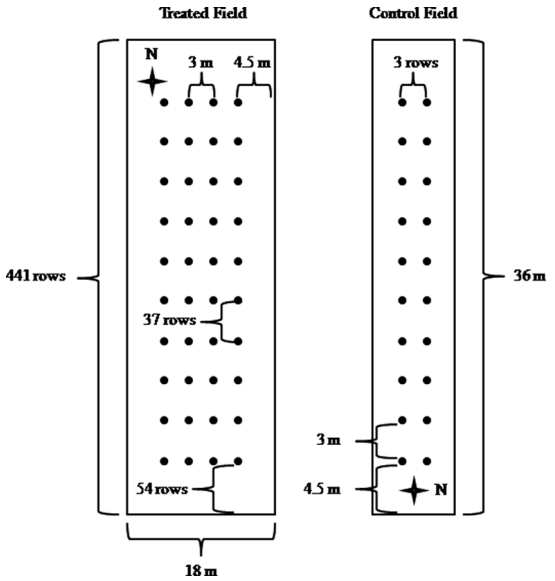

Fig. 1. Diagram of the experimental design (not drawn to scale) in treated and control fields. Circles represent the location of bioassay seedlings and field-collected leaves.

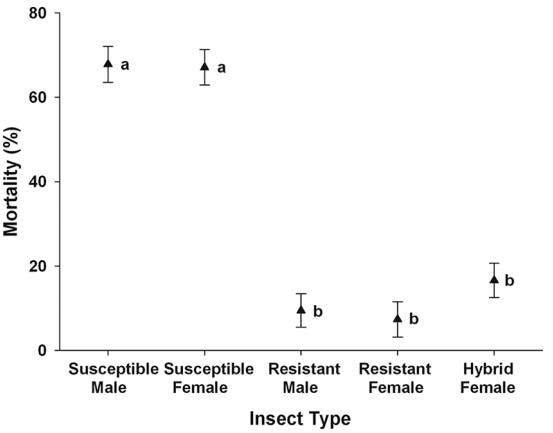

Fig. 2. Mortality caused by pyriproxyfen (% ± SE) from egg to adult for five insect types (pooled across fields and treatment stage) in direct exposure bioassays. Values that do not share the same letter were significantly different (α = 0.05; Tukey HSD test).

dishes with five virgin females or five mated pairs. After 48 h, adults were removed and eggs were counted. Seven days later, nymphs were counted, and egg mortality was assessed. Mortality from egg to adult was not measured as leaves taken from the field typically remained in good condition for only 2 wk. For leaves collected the day of spraying, this assay was done in both treated fields. For leaves collected 2 wk after spraying, this assay was performed only in field 2, because field 1 was accidentally sprayed a second time before 2 wk elapsed.

Data Analysis. When progeny included males and females, the number of eggs of each sex was estimated by correcting for the sex ratios of adult progeny emerging 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 relevant treatment. The estimated number of male nymphs was used to estimate the number of female nymphs.

Mortality on treated seedlings was corrected for control mortality (Abbott 1925). For the direct exposure bioassays, a three-way analysis of variance (ANOVA) on transformed mortality data (arcsine square root of proportion mortality) was used to compare adjusted mortality from egg to adult for all treatments. Insect type (susceptible male, susceptible female, resistant male, resistant female, or hybrid female), field (1 or 2), treatment stage (egg or nymph), and all interactions were explanatory variables. For the indirect exposure bioassays, two separate ANOVAs were performed. The first compared adjusted mortality on leaves taken from the field on the day of spraying based on the explanatory variables insect type, field, and the interaction between these terms. The second compared adjusted mortality in the egg stage in field two based on the explanatory variables insect type, time after spraying (0 d or 2 wk), and the interaction term. These analyses were performed separately, because data on mortality 2 wk after the spray were not collected in field 1. Adjusted mortality in the egg stage was compared between the direct and indirect exposure bioassays (0 d after a spray) by using a three-way ANOVA with insect type, field, type of exposure (direct or indirect), and all interactions as explanatory variables. When significant differences were detected, Tukey honestly significant difference (HSD) tests (SAS Institute 2002) were used to test for differences between treatment levels.

Dominance of resistance (h) 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, in the presence of the insecticide (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 ($0.5 < h < 1$). Values of $h > 1$ occur if W_h is greater than W_r. If W_h is significantly greater than W_r, this would indicate overdominance. However, as seen here in some cases, this difference was not significant, and thus we reported resistance as completely dominant in these cases.

Results

Direct Exposure to Eggs and Nymphs. In direct exposure bioassays, insect type was the only factor that significantly influenced mortality caused by pyriproxyfen ($F_{4,97} = 45.7$; $P < 0.0001$) (Fig. 2). Survival did not differ based on the stage treated, the field, or any interaction term ($P > 0.3$ for each factor). None of these factors became significant when nonsignificant variables were sequentially removed from the ANOVA model (one-way ANOVA: $F_{4,112} = 48.8$; $P < 0.0001$; $r^2 = 0.64$).

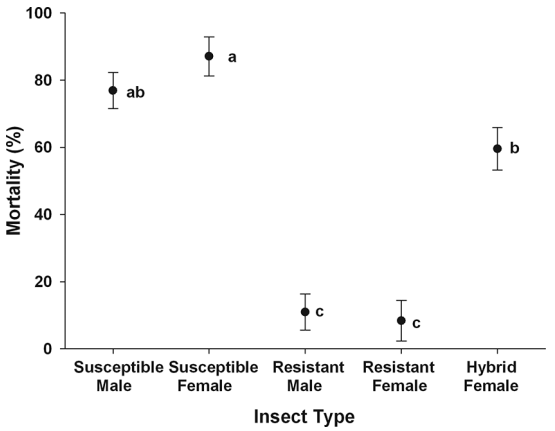

Fig. 3. Mortality caused by pyriproxyfen (% ± SE) in the egg stage, for five insect types (pooled across fields) in indirect exposure bioassays. Values that do not share the same letter were significantly different ($\alpha = 0.05$; Tukey HSD test).

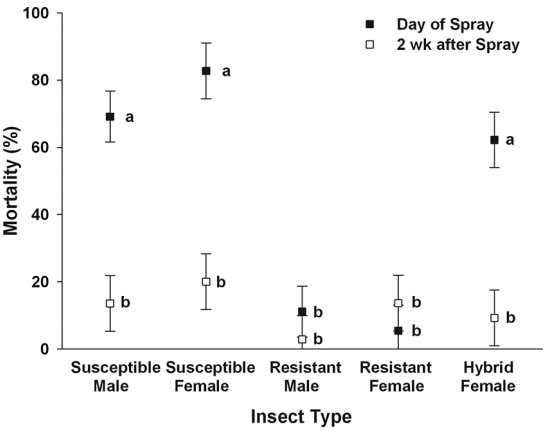

Fig. 4. Mortality caused by pyriproxyfen (% ± SE) in the egg stage, for five insect types on leaves collected from the field the day pyriproxyfen was sprayed or 2 wk after a spray in indirect exposure bioassays. Values that do not share the same letter were significantly different ($\alpha = 0.05$; Tukey HSD test).

Mortality caused by pyriproxyfen from egg to adult was highest in susceptible males (67.8%) and susceptible females (67.1%), and it did not differ significantly between these two types (Fig. 2). Mortality was lowest in resistant males (9.5%), resistant females (7.3%), and hybrid females (16.6%) and did not vary significantly among these three types (Fig. 2).

Indirect Exposure to Eggs. Mortality of eggs laid on leaves collected from fields on the day of spraying depended on insect type ($F_{4,51} = 32.3$; $P < 0.0001$). Mortality did not differ between fields, and the interaction of insect type and field was not significant ($P > 0.6$ for each factor). Neither of these factors became significant when nonsignificant variables were sequentially removed from the ANOVA model (one-way ANOVA: $F_{4,56} = 35.9$; $P < 0.0001$; $r^2 = 0.72$).

Mortality did not differ significantly between susceptible males (76.9%) and susceptible females (87.0%) or between susceptible males and hybrid females (62.2%) (Fig. 3). Mortality was higher for susceptible females than hybrid females. Resistant males (10.9%) and resistant females (8.4%) had the lowest mortality (Fig. 3).

Mortality was higher on leaves collected the day pyriproxyfen was sprayed than on leaves collected 2 wk after the spray ($F_{1,45} = 37.0$, $P < 0.0001$) and varied based on insect type ($F_{4,45} = 10.1$; $P < 0.0001$) (Fig. 4). A significant interaction occurred between insect type and time after spraying ($F_{4,45} = 5.1$; $P = 0.0018$).

Mortality of eggs was highest for susceptible males (69.1%), susceptible females (82.7%), and hybrid females (62.2%) and lowest for resistant males (11.1%) and resistant females (5.4%) on leaves collected the day pyriproxyfen was sprayed (Fig. 4). Egg mortality was low for all insect types on leaves collected 2 wk after the spray: susceptible males (13.5%), susceptible females (20.0%), resistant males (2.9%), resistant females (13.6%), and hybrid females (9.2%) (Fig. 4).

Comparison of Direct and Indirect Exposure. Mortality was affected by insect type ($F_{4,95} = 41.3$; $P < 0.0001$), the type of exposure ($F_{1,95} = 27.1$; $P < 0.0001$), and the interaction between these terms ($F_{4,95} = 5.0$, $P = 0.001$). Mortality was not affected by field or interaction terms other than insect type and type of exposure ($P > 0.3$ for each term). None of these factors became significant when nonsignificant variables were sequentially removed from the ANOVA model (two-way ANOVA: $F_{9,105} = 26.0$; $P < 0.0001$; $r^2 = 0.69$).

Egg mortality was lower with direct than indirect exposure bioassays ($F_{1,105} = 29.6$; $P < 0.0001$). The interaction between insect type and type of exposure affected mortality (Table 1). Mortality was highest in susceptible males and susceptible females exposed indirectly. Mortality was lowest in resistant males and resistant females, regardless of the exposure. Mortality for all other combinations of insect type and type of exposure were intermediate between these extremes (Table 1).

Dominance of Resistance. In the direct exposure bioassays, resistance was partially or completely dom-

Table 1. Insect type, exposure method, and mortality of whiteflies treated with pyriproxyfen

Insect type	Exposure	Mortality (%)[a,b,c]	SE[c]
Susceptible male	Direct	42.8cd	5.6
Susceptible male	Indirect	76.9ab	5.2
Susceptible female	Direct	47.0cd	6.2
Susceptible female	Indirect	87.0a	5.6
Resistant male	Direct	5.2e	5.9
Resistant male	Indirect	10.9e	5.2
Resistant female	Direct	12.9e	5.9
Resistant female	Indirect	8.4e	5.9
Hybrid female	Direct	30.8de	6.2
Hybrid female	Indirect	59.5bc	6.2

[a] Averaged across fields (one or two).
[b] Values followed by different letters were significantly different ($\alpha = 0.05$; Tukey HSD test).
[c] Data represent untransformed values.

Table 2. Effects of type of exposure and bioassay interval on dominance of resistance to pyriproxyfen in field tests

Exposure	Bioassay interval	Field	♀ Mortality (%)			Dominance (*h*)
			Susceptible	Hybrid	Resistant	
Direct exposure of eggs	Egg to adult	1	64.5	15.3	7.0	0.86
		2	78.4	19.3	4.9	0.80
Direct exposure of nymphs	Egg to adult	1	56.8	22.5	3.3	0.64
		2	69.9	7.5	14.1	1.12[a]
Indirect exposure (0 d after spray)	Egg to nymph	1	90.1	56.9	11.9	0.42
		2	82.7	62.2	5.5	0.27
Indirect exposure (2 wk after spray)	Egg to nymph	1	20.1	9.2	13.6	1.67[a]

Dominance (*h*) was calculated based on mortality of susceptible, hybrid, and resistant *B. tabaci* females. Values of *h* usually range from 0 (completely recessive) to 1 (completely dominant)[a]. Data not collected for indirect exposure 2 wk after spraying in field one.

[a] In two cases, mortality was numerically lower for hybrid females than for resistant females, but these differences were not significant and we interpret these as complete dominance rather than overdominance.

inant in *B. tabaci* treated as eggs or nymphs and dominance (*h*) varied from 0.64 to 1.12 (Table 2). Resistance was partially recessive in the indirect exposure bioassay (day after spraying), with $h = 0.42$ in field 1 and $h = 0.27$ in field 2. Values of *h* increased from 0.27 (partially recessive) to 1.67 (completely dominant) over the 2 wk period after pyriproxyfen was sprayed (Table 2).

Discussion

Results showed that survival of direct exposure to pyriproxyfen in the field and to leaves sprayed with pyriproxyfen was higher for the laboratory-selected resistant strain (QC-02) than for a susceptible strain. Resistance varied from partially recessive to completely dominant, depending on the method and timing of exposure. When the observed values for mortality and dominance from both the direct and indirect exposure bioassays were incorporated into a previously published model (Crowder et al. 2006), resistance evolved in <20 yr with 20% of fields treated with pyriproxyfen. Thus, assuming that a similar type of resistance is present in individuals from field populations of *B. tabaci* (B biotype), resistance would be expected to increase despite the currently recommended rotation strategy (Ellsworth and Martinez-Carillo 2001; Palumbo et al. 2001; Li et al. 2003; Dennehy et al. 2004). Moreover, the model predictions indicate that even relatively large refuges not treated with pyriproxyfen would not delay future resistance development.

In Israel, resistance to pyriproxyfen in field populations of the Q biotype of *B. tabaci* was detected after 5 yr of pyriproxyfen use, despite limits of one application per season (Denholm et al. 1998; Horowitz et al. 1999, 2002). Previous experiments performed in field simulation cages have indicated that pyriproxyfen was not effective in controlling these resistant strains of *B. tabaci* (Horowitz et al. 2002). These results, along with those described here, demonstrate the potential future impact of the resistance to pyriproxyfen isolated in Arizona. However, resistance has not been documented in field populations of the B biotype in Arizona (Dennehy et al. 2004; unpublished data). Resistance in strains of the Q biotype seems to be monogenic (Horowitz et al. 2003), but the QC-02 strain may have more than one resistance gene (unpublished data). This distinction and differences in the mechanism(s) and intensity of resistance to pyriproxyfen between these biotypes may partially account for the observed levels of resistance in field populations.

Pyriproxyfen mortality was higher, and resistance more recessive, as a result of indirect exposure compared with direct exposure. This was surprising and counter to observed trends in the laboratory (unpublished data). Indirect exposure was expected to result in lower mortality because rather than being contacted at time of spraying, eggs may only be affected when pyriproxyfen moves up the pedicel. Additionally, leaves for the indirect exposure assay were taken from the fourth node of cotton plants, whereas seedlings for the direct exposure assay were placed higher in the canopy (≈15 cm) and were expected to receive a higher dose of pyriproxyfen. Because control mortality on field-collected leaves did not differ from control mortality of seedlings (data not shown), it is unlikely that reduced quality and increased handling of field-collected leaves compared with seedlings magnified the effects of pyriproxyfen. The simplest explanation for these results is that eggs exposed directly were 16–64 h old at the time pyriproxyfen was sprayed, whereas eggs in the indirect contact bioassays were exposed to pyriproxyfen as soon as they were laid. As pyriproxyfen inhibits embryogenesis in eggs (Ishaaya and Horowitz 1992), susceptibility to pyriproxyfen may decrease as eggs develop. It is also plausible that unknown factors associated with indirect exposure to pyriproxyfen on leaves from mature plants increased the lethality of pyriproxyfen.

Mortality caused by pyriproxyfen decreased significantly in 2 wk after a spray. This is consistent with findings that the residual time of pyriproxyfen is <14 d (Ellsworth and Martinez-Carillo 2001) or 3.5–16.5 d in cotton fields (Knack IGR Technology Information Bulletin, Valent USA). However, growers often report control for 30 d after a pyriproxyfen application (Ellsworth and Martinez-Carillo 2001). In the field, pest suppression may last longer than 2 wk as a result of increased mortality caused by natural enemies (Ellsworth and Martinez-Carillo 2001), or by sterilization

of adult females feeding on treated leaves. Thus, in contrast to broadly toxic insecticides, the benefits of pyriproxyfen likely last for several weeks after it no longer kills *B. tabaci.*

Laboratory bioassays showed that when treated with pyriproxyfen as nymphs, resistant males had higher mortality than resistant females (unpublished data). Here, no difference in susceptibility to pyriproxyfen was found between susceptible males and susceptible females or resistant males and resistant females. Thus, differential mortality of males and females may not be a factor affecting evolution of resistance in the field. If males and females are equally susceptible to an insecticide, resistance evolution can occur faster in haplodiploid than diploid pests when selection favors resistant hemizygous males (Denholm et al. 1998; Carrière 2003; Crowder et al. 2006).

Laboratory bioassays also indicated that pyriproxyfen was more active against *B. tabaci* treated as nymphs than eggs (Horowitz et al. 1999; unpublished data), although, at least in the B biotype, this result may have been caused by differences in treatment methods rather than variation between the life stages. In contrast, mortality in the field did not differ based on the stage treated. These results suggest that treating *B. tabaci* in either stage with pyriproxyfen may be equally effective.

As described above, results from laboratory and field bioassays were not always similar. Other studies have similarly shown that levels of resistance to insecticides in *B. tabaci* differed in laboratory bioassays and field-simulation trials (Rowland et al. 1991; Cahill et al. 1995). Thus, field trials are a useful complement to laboratory bioassays in understanding the potential impacts of resistance.

This work represents a proactive effort to understand the genetics of resistance to pyriproxyfen in the laboratory-selected QC-02 strain before high levels of resistance occur in the field. Several factors associated with the QC-02 strain could accelerate evolution of resistance in the field by the B biotype of *B. tabaci*, including no differences in survival between resistant males and females (Figs. 2–4), partially to completely dominant resistance (Table 2; Figs. 2–4), and lack of fitness costs associated with resistance (unpublished data). However, the nature and mechanism of resistance in the QC-02 strain may differ from other field populations of *B. tabaci.* It remains to be seen whether the type of resistance in QC-02 will evolve to problematic levels in field populations.

Acknowledgments

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