Modeling Evolution of Resistance to Pyriproxyfen by the Sweetpotato Whitefly (Homoptera: Aleyrodidae)

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ABSTRACT We used computer simulations to examine evolution of resistance to the insect growth regulator (IGR) pyriproxyfen by the sweetpotato whitefly, *Bemisia tabaci* (Gennadius), biotype B [=*Bemisia argentifolii* (Bellows & Perring)]. Consistent with trends seen in cotton (*Gossipyium* spp.) fields in Arizona and Israel, results suggest that evolution of resistance to pyriproxyfen may occur rapidly in this haplodiploid insect. Similar to results from models of diploid insects, resistance evolved faster with increases in toxin concentration, dominance of resistance in females, the initial frequency of the resistance allele, and the proportion of the region treated with pyriproxyfen. Resistance was delayed by fitness costs associated with resistance. Movement between treated and untreated cotton fields had little effect, probably because untreated cotton leaves provided internal refuges in treated fields and whiteflies were controlled with other insecticides in external refuges. Resistance evolved faster when susceptibility to pyriproxyfen was greater in susceptible males than susceptible females. In contrast, resistance evolved slower when susceptibility to pyriproxyfen was greater in resistant males than resistant females. Results suggest that growers may be able to prolong the usefulness of pyriproxyfen by applying lower toxin concentrations and promoting susceptible populations in refuges.

KEY WORDS *Bemisia tabaci*, modeling, pyriproxyfen, resistance management, haplodiploid

The sweetpotato whitefly, *Bemisia tabaci* (Gennadius), is one of the world's most destructive crop pests (Byrne and Bellows 1991, Perring et al. 1993, Bedford et al. 1994, Brown et al. 1995, Viscaret et al. 2003). *B. tabaci* comprises biotypes that attack field and horticultural crops across subtropical and tropical regions (Brown et al. 1995, Perring 2001). The B biotype [=*Bemisia argentifolii* (Bellows & Perring)], was introduced to the United States at the end of the 1980s (Brown et al. 1995, Ellsworth and Martinez-Carillo 2001) and is a key pest of cotton (*Gossypium* spp.) and other crops in the U.S. Southwest.

Controlling pests such as *B. tabaci* with specific, environmentally friendly methods rather than broad-spectrum insecticides is a cornerstone of integrated pest management (IPM) (Lewis et al. 1997, Matson et al. 1997, Carrière et al. 2004). Use of two insect growth regulators (IGRs), pyriproxyfen and buprofezin, to control *B. tabaci* in Arizona and Israel exemplifies integration of selective IGRs into IPM systems (Dennehy and Williams 1997, Denholm et al. 1998, Ellsworth and Martinez-Carillo 2001). These IGRs have been rotated with other insecticides in Arizona cotton since 1996. A single, timely application of either of these IGRs has dramatically reduced broad-spectrum insecticide use, helped to conserve natural enemies, and restored farmer's profits (Dennehy and Williams 1997, Ellsworth and Martinez-Carillo 2001, Naranjo et al. 2004).

Whitefly resistance to pyriproxyfen has occurred in Israel (Horowitz et al. 2002). Although no field failure of pyriproxyfen has been yet documented in Arizona, laboratory bioassays over nine years reveal an area-wide decline in whitefly susceptibility to pyriproxyfen (Li et al. 2003, Dennehy et al. 2004).

Resistance management has focused almost exclusively on diploid pests, whereas haplodiploid pests have received little attention (Denholm et al. 1998, Carrière 2003). Yet, the differences between diploidy and haplodipoloidy may have important implications. *B. tabaci* is haplodiploid, with males produced from unfertilized haploid eggs and females produced from fertilized diploid eggs. Thus, this article analyzed insect resistance management (IRM) strategies in a haplodiploid pest.

We simulated the population dynamics and genetics of *B. tabaci* to analyze its potential for evolution of resistance to pyriproxyfen. We used sensitivity analysis to examine effects of pyriproxyfen concentration, dominance, and other key factors on the evolution of resistance.

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Materials and Methods

Model Structure. We used a temperature-dependent, deterministic model written in FORTRAN (Intel 2003). We modeled a region with two habitat types: 1) treated cotton fields (treated with pyriproxyfen and other insecticides) and 2) refuge cotton fields (treated with insecticides other than pyriproxyfen). In treated fields, pyriproxyfen was applied once per year when populations exceeded an action threshold of three adults per leaf (Ellsworth et al. 1996). The refuge was not treated with pyriproxyfen, but other insecticides were used if adult density exceeded an action threshold of five adults per leaf (Ellsworth et al. 1996). Based on pyriproxyfen use in Arizona cotton from 1999 to 2004 (Arizona Agricultural Statistics Service 1999, 2000, 2001, 2002, 2003, 2004), standard use of pyriproxyfen in a given year was 20% of cotton fields treated with pyriproxyfen and 80% not treated. We also performed simulations without insecticides to compare population dynamics predicted by the model with field observations on untreated cotton fields (Watson et al. 1992, Naranjo and Ellsworth 2005).

The model included three life stages: eggs, larvae, and adults. Whitefly mortality, development, movement, mating, and oviposition were simulated independently in each field with a daily time step.

Accumulated degree-days (DD) affected cotton dynamics, and whitefly movement and oviposition. The average daily temperature in °C (T) affected immature development, adult survival, and oviposition. Daily temperature and DD were based on the 30-yr average for each date in Maricopa, AZ (WRCC 2003), with DD accumulated using a base of 12.8°C and a maximum of 30°C (Silvertooth 2001a,b).

Genetics of Resistance. We assumed that resistance to pyriproxyfen is controlled by one gene with two alleles (Horowitz et al. 2003; S for susceptibility; R for resistance). Haploid males were S or R. Diploid females were SS, RS, or RR. We assumed that mutations did not occur after the start of the simulations.

Crop Phenology. Crop phenology was the same in the pyriproxyfen-treated and refuge fields. Fields were planted on 20 April (400 DD) (Silvertooth and Brown 2001), at 98,800 plants per ha. The first leaves occurred on 1 May, ≈175 DD after planting (UCDANR 1996). The number of leaves increased linearly to a maximum of 200 per plant by 11 August, ≈3000 DD after planting (Silvertooth 2001b), and remained there throughout the season. Increasing leaf age affected oviposition and movement after 11 August until 1 October, ≈4500 DD after planting, coinciding with a typical date of defoliation of cotton in Arizona (Naranjo et al. 1998), at which point we assumed leaves were unsuitable for oviposition.

Immature Development and Survival. We used data from three studies (Butler et al. 1983, Powell and Bellows 1992a, Wagner 1995) to determine the developmental rate of eggs and larvae, with the four larval instars modeled as a single larval stage. We used nonlinear regression (PROC NLIN, SAS Institute 2002) to fit the data to an equation from Logan et al. (1976):

$$R = a \times [\exp\{b \times (T - THR)\} - \exp\{(b \times [TM - THR]) - (\{(TM - THR) - (T - THR)\}/c)\}] \quad [1]$$

where R is the developmental rate (1/d); THR is the minimum temperature threshold for development; TM is the maximum temperature threshold; and a, b, and c are fitted parameters. We used 10.8°C for THR and 36°C for TM (Butler et al. 1983, Von Arx et al. 1983). When fit to the observed data, the estimates for a, b, and c were 0.048, 0.079, 1.436, respectively, for the egg stage ($n = 35$, $r^2 = 0.98$) and 0.040, 0.141, and 6.071, respectively, for the larval stage ($n = 35$, $r^2 = 0.98$). Eggs completing development became larvae. Larvae completing development became 1-d-old adults.

Based on field data from insecticide-free plots in Maricopa, AZ (Naranjo and Ellsworth 2005), we assumed that 63.5% of eggs hatched and 15.7% of larvae became adults. Thus, survival from the egg to adult stage was 10%.

Adult Longevity. We used linear regression (PROC GLM, SAS Institute 2002) to determine the longevity of adult *B. tabaci* as a function of average daily temperature (T) based on data from three studies (Butler et al. 1983, Von Arx et al. 1983, Powell and Bellows 1992b). Female longevity, $L_{F,T}$, ($n = 6$, $r^2 = 0.57$, $P = 0.057$) was as follows:

$$L_{F,T} = -1.86 \times T + 69.56 \quad [2]$$

Male longevity, $L_{M,T}$, ($n = 6$, $r^2 = 0.55$, $P = 0.057$) was as follows:

$$L_{M,T} = -1.49 \times T + 56.64 \quad [3]$$

For the temperature range modeled (14–32°C), longevity was 10–44 d for females and 9–36 d for males.

Adult Movement. We modeled adult movement based on observations of *B. tabaci* in the field and flight chambers (Horowitz 1986, Horowitz and Gerling 1992; Watson et al. 1992; Blackmer and Byrne 1993a,b; Blackmer et al. 1995a,b; Isaacs and Byrne 1998; Naranjo and Ellsworth 2005). Adults moved only once at 5 d old.

Each day, under standard conditions, 6% of 5-d-old adults went into a "cotton movement pool" that was distributed between the two field types in proportion to their relative abundance. Thus, under standard conditions with 20% pyriproxyfen-treated cotton and 80% refuge cotton, 4.8% of 5-d-old adults in treated cotton moved to refuge cotton (6% in movement pool × 0.80 refuge cotton) and 1.2% of 5-d-old adults in refuge cotton moved to treated cotton (6% in movement pool × 0.20 treated cotton).

We also modeled movement into and out of the region. From 1 wk after the first leaf occurred (8 May) until 7 June (≈1,000 DD after planting), immigration of 5-d-old adults into the region was 0.1 adults per plant per day. The number of adults per plant per day moving into the region increased linearly from 0.1 to 50 from 7 June to 23 June (≈1,500 DD after planting), was 50 from 23 June until 9 July (peak bloom) and then dropped to 0 for the rest of the year. The R allele

frequency of immigrants in year 1 was 0.001. For all other years, the R allele frequency of immigrants was equal to the R allele frequency of adults that moved out of the region the previous year (Note: This statement applies to all simulations without overwintering fitness costs, a special case explained later). Movement out of region was 0% before 11 August, increased linearly from 0 to 18% from 11 August until 1 October, and remained at 18% for the rest of the year.

Mating and Oviposition. One-day-old females mated randomly with males age 2 d old or greater in the field of emergence (Li et al. 1989). All females mated if males were present in the field. Females age 2 d or greater laid viable eggs (Wagner 1994, Tsai and Wang 1996, Wang and Tsai 1996).

Fecundity was affected by age of the female, temperature, and leaf age (Gameel 1974, Von Arx et al. 1983, Horowitz and Gerling 1992, Powell and Bellows 1992b).

We used the following formula from Von Arx et al. (1983) to calculate the fecundity rate (eggs per day) based on age (A), R_A:

$$R_A = \{8.9413 \times (A - 0.4344)\}/\{1.2264^{(A - 0.4344)}\} \quad [4]$$

We fit the following function for oviposition rate based on temperature (T), R_T, (Von Arx et al. 1983), to data from two studies (Powell and Bellows 1992b, De Barro and Hart 2000) (PROC NLIN, SAS Institute 2002) ($n = 5$, $r^2 = 0.94$):

$$R_T = 0.00384 \times T^3 - 0.3029 \times T^2 + 7.8049 \times T - 65.0074 \quad [5]$$

When equation 5 produced negative R_T values, R_T was set to 0.

The oviposition rate as a function of leaf age, R_{LA}, was 1.0 until 8 August. R_{LA} decreased linearly from 1.0 to 0.0 from 11 August until 1 October, corresponding with increasing leaf age (Gameel 1974, Von Arx et al. 1983).

The standard value for proportion of male eggs, PM, was 0.4 (De Barro and Hart 2000). The number of male eggs per day, EM, of genotype i in field p as a function of the number of females of age A and genotype j in field p, $F_{A,j,p}$ was as follows:

$$EM_{i,p} = \left(\sum_{A=2}^{L} F_{A,j,p} \times R_A \times R_T \times R_{LA} \times 0.4 \right) \times \sum_{j=1}^{3} Q_j \quad [6]$$

Q is the frequency of eggs of genotype i (S or R) produced by females of genotype j (SS, SR, or RR) and L is the longevity of females at temperature T.

The number of female eggs per day, EF, of genotype i in field p was as follows:

$$EF_{i,p} = \left(\sum_{A=2}^{L} F_{A,j,p} \times R_A \times R_T \times R_{LA} \times 0.6 \right) \times \sum_{j=1}^{3} \sum_{k=1}^{2} Q_{j,k} \quad [7]$$

Q is the frequency of eggs of genotype i (SS, SR, or RR) produced by females of genotype j (SS, SR, or RR) mated by males of genotype k (S or R).

Pyriproxyfen. Pyriproxyfen inhibits egg hatching and adult eclosion (Ishaaya and Horowitz 1992, Horowitz and Ishaaya 1994, Horowitz et al. 1999). Mortality from pyriproxyfen depended on the genotype of the insect and the pyriproxyfen concentration (low, medium, or high). Pyriproxyfen killed eggs and larvae for 28 d (P.C.E. et al., unpublished data). Leaves that developed after the application were not affected, because pyriproxyfen is not systemic (Ellsworth and Martinez-Carrillo 2001). Thus, as plants in treated fields grew after a pyriproxyfen application, the new growth served as an untreated refuge within the treated fields. Pyriproxyfen did not kill resistant (R or RR) eggs and larvae. Mortality of susceptible (S or SS) eggs due to pyriproxyfen was 40, 60, and 90% with a low, medium, and high concentration, respectively. Mortality of susceptible larvae due to pyriproxyfen was 70, 95, and 99% with a low, medium, and high concentration, respectively. The three mortality classes are associated with an assumed range of variation that might be expected from variable rates used by growers or deposition of concentrations through the canopy vertically. Mortality due to pyriproxyfen for eggs and larvae occurred at egg hatching or adult eclosion, respectively. For females, the survival of RS eggs and larvae was affected by dominance (h) and insecticide concentration, where h is as follows:

$$h = (\text{survival RS} - \text{survival SS})/(1 - \text{survival SS}) \quad [8]$$

We simulated values for h of 0, 0.1, 0.5, 0.9, and 1 for each concentration.

Insecticides Other than Pyriproxyfen. Based on IPM recommendations, insecticides other than pyriproxyfen were applied to both field types depending on adult densities (Ellsworth et al. 1996). When adult densities first exceeded five adults per leaf in the refuge, an insecticide other than pyriproxyfen was applied that killed 90% of all immatures for 28 d after application. In the pyriproxyfen-treated field, an insecticide was applied after the first treatment if adult densities again exceeded five adults per leaf. If densities in the refuge or treated field exceeded five adults per leaf after the application of an insecticide, another insecticide was applied.

Simulation Conditions. For each set of conditions (Table 1), we ran the model for 100 yr. The time to resistance was the number of years for the R allele frequency to reach 0.5. When the R allele frequency did not reach 0.5 in 100 yr, we report the R allele frequency after 100 yr. For each year, the R allele

Table 1. Parameter values used in the model

Parameters		Values used[a]	
Percentage of landscape treated with pyriproxyfen		10, 20*, 40	
Concentration of pyriproxyfen		High, medium, low	
Dominance of resistance in females (h)		0, 0.1, 0.5, 0.9, 1.0	
Initial R allele frequency		0.001*, 0.01, 0.05	
Percentage of adults that move between cotton fields		6*, 0–100	
Proportion of males laid by mated females		0.4*, 0.2–0.8	
Mortality of susceptible (S males and SS females) eggs as a function of pyriproxyfen concentration[b]	High	Medium	Low
1) Standard	90%*	60%*	40%*
2) S males 1/2 survival of SS females	95%	80%	70%
3) S males 1/4 survival of SS females	97.5%	90%	85%
Mortality of susceptible (S males and SS females) larvae as a function of pyriproxyfen concentration[b]	High	Medium	Low
1) Standard	99%*	95%*	60%*
2) S males 1/2 survival of SS females	99.5%	97.5%	80%
3) S males 1/4 survival of SS females	99.75%	98.75%	90%
Mortality of resistant eggs and larvae (R males and RR females) with any pyriproxyfen concentration[c]		0%*, 10%, 25%	
Reduction in survival (%) for immatures in areas not treated with pyriproxyfen (fitness cost)[d]	R	RR	RS
1) Standard	0*	0*	0*
2) Case 1	10	10	0
3) Case 2	10	10	5
4) Case 3	20	10	0
5) Case 4	20	10	5

[a] Default values are indicated by an asterisk.
[b] Values used in sensitivity analyses were varied only for S males (not SS females) to represent increased susceptibility of males compared with females.
[c] Values used in sensitivity analyses were varied only for R males (not RR females) to represent increased susceptibility of males compared with females.
[d] R denotes males of genotype R. RR and RS denote females of genotypes RR and RS.

frequency was calculated at the end of the cotton-growing season based on individuals that moved out of the region, because individuals remaining in the region died when cotton was harvested and thus did not influence future generations.

Sensitivity Analysis. We analyzed the influence of variation in eight factors: dominance, concentration, initial R allele frequency, refuge size, adult movement, proportion of male eggs laid by mated females, susceptibility of males relative to females, and fitness costs (Table 1). For each sensitivity analysis, all parameters except the parameter being varied were set to the standard values. For each parameter value for the latter six factors listed above, we ran simulations with 15 combinations of toxin concentration (low, medium, and high) and dominance ($h = 0, 0.1, 0.5, 0.9$, and 1).

Based on bioassay data for *B. tabaci* susceptibility to several insecticides (for review, see Carrière 2003), we tested effects of greater susceptibility to pyriproxyfen in males than females as follows: survival of susceptible eggs and larvae for males relative to females was 1.0 (standard), 0.5, or 0.25; mortality of resistant male eggs and larvae due to pyriproxyfen at any concentration was 0 (standard), 10, or 25%; and both of the above (susceptible and resistant males more susceptible than their female counterparts).

Based on data from Horowitz et al. (2002), we tested the effects of fitness costs. We simulated four cases in which the survival of resistant individuals was reduced compared with S males and SS females: 1) 10% reduction in survival for R males and RR females; 2) 10% reduction in survival for R males and RR females, 5% reduction in survival for RS females; 3) 20% reduction in survival for R males, 10% reduction in survival for RR females; and 4) 20% reduction in survival for R males, 10% reduction in survival for RR females, 5% reduction in survival for RS females (Table 1). These costs occurred during the immature stage for individuals in the refuge and treated fields when pyriproxyfen was not present. In addition, because fitness costs could affect the survival of *B. tabaci* over the winter (not modeled in standard simulations), we simulated these costs over four generations during the winter. For these generations, the change in the R allele frequency was calculated assuming that reproduction and survival were based solely on relative fitness.

Results

Simulations without Insecticides. Without insecticide use, adult and immature populations remained at low densities early in the season and grew exponentially later in the season when temperature and plant conditions were more favorable (Fig. 1A and B). These trends are similar to field observations of *B. tabaci* population growth in untreated cotton fields (Watson et al. 1992, Naranjo and Ellsworth 2005).

Concentration, Dominance, Initial Allele Frequency, and Refuge Size. Resistance evolved faster with increases in toxin concentration and dominance (Fig. 2). The effect of pyriproxyfen concentration on

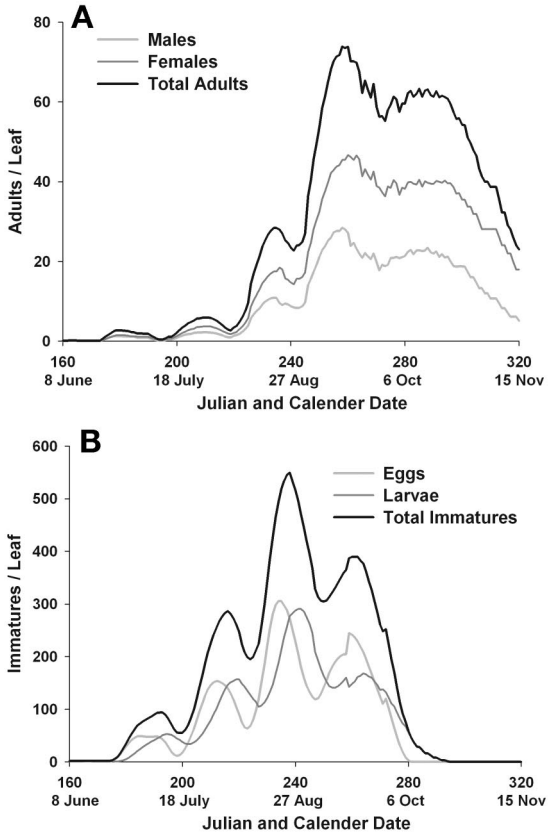

Fig. 1. Simulated densities of (A) adults and (B) immatures over one season without insecticides.

the evolution of resistance decreased as dominance increased (Fig. 2). Resistance evolved faster as initial frequency of the R allele or proportion of the region treated with pyriproxyfen increased (data not shown).

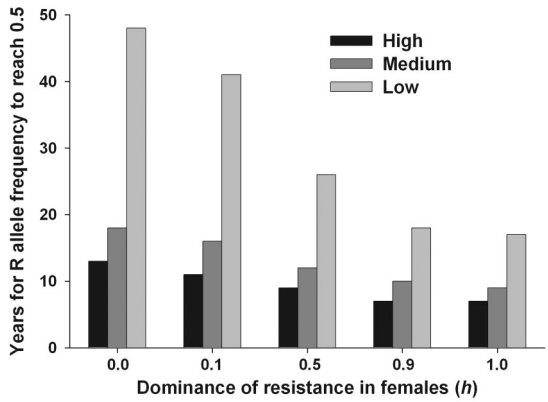

Fig. 2. Number of years for the frequency of the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations of the standard model with five levels of dominance (h) and three pyriproxyfen concentrations.

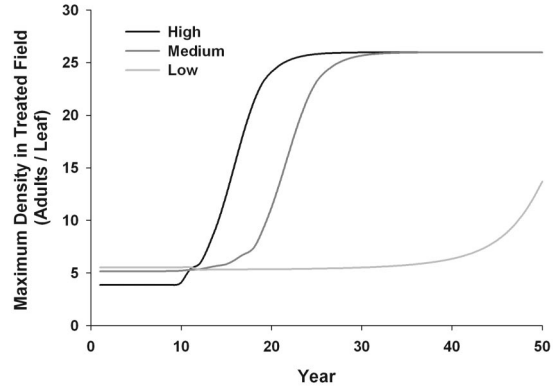

Fig. 3. Maximum population density (adults per leaf) each year in treated fields for the first 50 simulated years with three pyriproxyfen concentrations and dominance (h) = 0.

Population density reached a maximum of four to six adults per leaf in treated fields with low R allele frequency (<0.10) (Fig. 3). As resistance evolved and pyriproxyfen became less effective, the maximum population density in treated fields increased (Fig. 3). With a high concentration and low R allele frequency, a single spray of pyriproxyfen reduced densities below three adults per leaf for the remainder of the year, whereas two insecticide applications per year were needed with a medium or low concentration. With any concentration, as the R allele frequency approached 0.5, three insecticide applications per year were needed in treated fields. Two insecticide applications per year were sufficient to maintain populations in the refuge below five adults per leaf.

Movement. Variation in adult movement among cotton fields had little effect on the rate of resistance evolution, except at extreme values (Table 2). Effects

Table 2. Number of years for the frequency of the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations with varying levels of movement, with five levels of dominance (h) and three pyriproxyfen concentrations

% individuals capable of movement	Dominance of resistance in females (h)				
	0.0	0.1	0.5	0.9	1.0
	High concentration				
0%	12	11	8	7	7
6%	13	11	9	7	7
50%	15	13	9	7	7
100%	16	14	10	7	7
	Medium concentration				
0	18	16	12	10	9
6	18	16	12	10	9
50	17	16	11	9	8
100	16	15	11	8	8
	Low concentration				
0	52	44	27	19	18
6	48	41	26	18	17
50	31	28	19	15	14
100	22	20	15	12	11

Movement is expressed as the percentage of individuals capable of moving between fields. The standard value for movement was 6%.

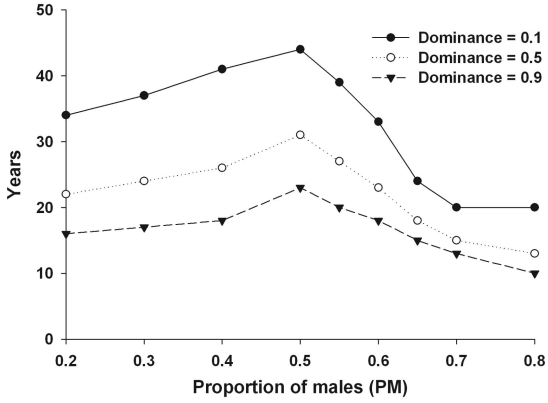

Fig. 4. Number of years for the frequency of the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations with a low concentration, three levels of dominance (h), and varying values for the proportion of males produced by mated females, PM.

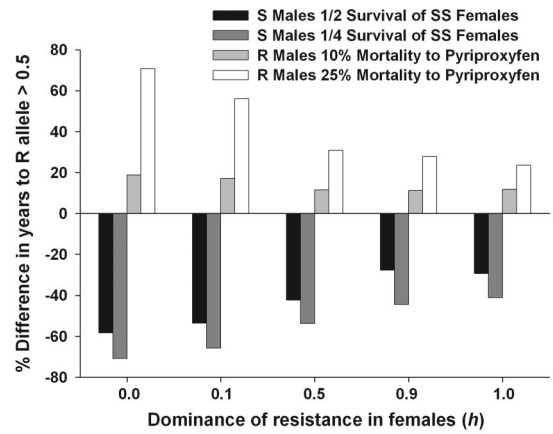

Fig. 5. Percentage (%) of difference in the number of years for the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations with increased susceptibility of males compared with the standard simulations. Results shown with a low concentration, five levels of dominance (h), and four cases of increases susceptibility of either S or R males compared with SS or RR females, respectively.

of movement were greater with a low pyriproxyfen concentration than with a high or medium concentration (Table 2). With a low concentration, resistance evolved slower when movement among cotton fields was low (maximum time = 52 yr, 0% in cotton movement pool (see Materials and Methods) than when it was high (minimum time 22 yr, 100% in cotton movement pool).

Proportion of Males. With a medium or high pyriproxyfen concentration, the proportion of male eggs laid by mated females (PM) had little or no effect on resistance evolution (data not shown). At a low pyriproxyfen concentration, resistance evolved slowest with PM = 0.5 and fastest at the extreme value of 0.8 (Fig. 4).

With a low concentration, changing PM affected population dynamics in both field types, which affected the evolution of resistance. With PM = 0.5, when resistance evolved slowest, the average density of adults in the treated field was 0.3–0.4 times the density in the refuge during the period when adults moved out of cotton. With PM = 0.4, the average density of adults in the treated field was 1.3–1.6 times greater than in the refuge. Finally, with PM = 0.8, the average density of adults in the treated field was 2.3–2.4 times greater than in the refuge. The frequency of resistance to pyriproxyfen was greater in individuals surviving a pyriproxyfen treatment than in individuals surviving other insecticides in the refuge. Thus, as the capacity of whitefly populations to buildup after a pyriproxyfen treatment increased compared with populations in refuges, the strength of selection for resistance to pyriproxyfen increased and the efficacy of refuges decreased. Accordingly, PM modulated the evolution of resistance through its effects on population dynamics, which affected selection intensity and refuge efficacy.

Increased Susceptibility of Males. Increased susceptibility of males compared with females affected the evolution of resistance to pyriproxyfen, especially with a medium or low concentration. With a high concentration, increased susceptibility of S males compared with SS females, increased susceptibility of R males compared with RR females, or both had little or no effect on the evolution of resistance. In most cases, the time to resistance changed <1 yr compared with the standard simulations.

With a low concentration, resistance evolved faster with susceptibility to pyriproxyfen greater for S males than SS females (Fig. 5). In general, increased susceptibility of males had a smaller effect on the evolution of resistance as dominance increased. When one-half or one-quarter as many S males as SS females survived the effects of pyriproxyfen, the time to resistance decreased by 29–58 or 41–77%, respectively, compared with the standard simulations (Fig. 5). Results were similar with a medium concentration, although the change was smaller than with a low concentration.

Resistance evolved slower with increased susceptibility of R males compared with RR females (Fig. 5). With any dominance, when 10 or 25% of R male eggs and larvae incurred mortality due to pyriproxyfen, the time to resistance increased by 12–19 or 24–71%, respectively, compared with the standard simulations (Fig. 5). Again, results were similar with a medium concentration, although the change was smaller than with a low concentration.

Resistance generally evolved faster with a medium or low concentration and increased susceptibility of both S and R males compared with SS and RR females. Thus, in most cases, increased susceptibility of S males compared with SS females affected the evolution of resistance more than increased susceptibility of R males compared with RR females. However, in several cases, the effects of increased susceptibility of both S males and R males effectively canceled each other.

Table 3. Number of years for the frequency of the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations with fitness costs applied for R males, RR females, and RS females, with five levels of dominance (*h*) and three pyriproxyfen concentrations

Reduction in survival for resistant individuals	Dominance of resistance in females (*h*)				
	0.0	0.1	0.5	0.9	1.0
			High concentration		
No fitness costs	13	11	9	7	7
R 10%, RR 10%	14	13	12	8	8
R 10%, RR 10%, RS 5%	16	17	12	9	8
R 20%, RR 10%	>100 (0.39)	>100 (0.39)	16	10	9
R 20%, RR 10%, RS 5%	>100 (0.36)	>100 (0.36)	21	11	10
			Medium concentration		
Fitness costs	18	16	12	10	9
R 10%, RR 10%	25	25	24	15	14
R 10%, RR 10%, RS 5%	31	28	18	13	12
R 20%, RR 10%	>100 (0.30)	>100 (0.37)	28	15	14
R 20%, RR 10%, RS 5%	>100 (0.30)	>100 (0.38)	34	17	14
			Low concentration		
No fitness costs	48	41	26	18	17
R 10%, RR 10%	>100 (0.076)	100	42	26	23
R 10%, RR 10%, RS 5%	>100 (0.0001)	>100 (0.001)	73	34	29
R 20%, RR 10%	>100 (0.0002)	>100 (0.004)	98	39	34
R 20%, RR 10%, RS 5%	>100 (0.00)	>100 (0.00)	>100 (0.002)	58	47

These fitness costs were only applied for individuals on cotton plants not treated with pyriproxyfen (i.e., not in the overwintering generations). If the R allele did not exceed 0.5 within 100 yr, the R allele frequency in year 100 is shown in parentheses. Values of 0.00 indicate that the R allele frequency decreased below 10^{-8} after 100 yr.

Fitness Costs. Fitness costs associated with pyriproxyfen slowed the evolution of resistance, especially if they were greater in males than females (Tables 3 and 4). When fitness costs were simulated for four generations over the winter each year, resistance to pyriproxyfen was further delayed (Table 4).

When fitness costs were only expressed in individuals on cotton plants not treated with pyriproxyfen, the impact of fitness costs increased as dominance of resistance and toxin concentration decreased (Table 3). The evolution of resistance was least affected with a 10% reduction in survival for both R males and RR females and most affected with a 20, 10, and 5% reduction in survival for R males, RR females, and RS females, respectively. Greater costs in males than females slowed resistance more than costs in heterozygous females (Table 3).

When fitness costs were expressed in individuals on cotton plants not treated with pyriproxyfen as well as over four generations during the winter, generally the

Table 4. Number of years for the frequency of the allele for resistance to pyriproxyfen, R, to reach 0.5 in simulations with fitness costs applied for R males, RR females, and RS females, with five levels of dominance (*h*) and three pyriproxyfen concentrations

Reduction in survival for resistant individuals	Dominance of resistance in females (*h*)				
	0.0	0.1	0.5	0.9	1.0
			High concentration		
No fitness costs	13 (1.00)	11 (1.00)	9 (1.00)	7 (1.00)	7 (1.00)
R 10%, RR 10%	>100 (0.36)	>100 (0.40)	>100 (0.42)	17 (0.59)	17 (0.59)
R 10%, RR 10%, RS 5%	>100 (0.38)	>100 (0.39)	>100 (0.24)	35 (0.59)	24 (0.60)
R 20%, RR 10%	>100 (0.28)	>100 (0.28)	>100 (0.21)	>100 (0.19)	>100 (0.27)
R 20%, RR 10%, RS 5%	>100 (0.01)	>100 (0.25)	>100 (0.23)	>100 (0.15)	>100 (0.15)
			Medium concentration		
No fitness costs	18 (1.00)	16 (1.00)	12 (1.00)	10 (1.00)	9 (1.00)
R 10%, RR 10%	>100 (0.35)	>100 (0.33)	>100 (0.34)	29 (0.51)	23 (0.53)
R 10%, RR 10%, RS 5%	>100 (0.001)	>100 (0.09)	>100 (0.19)	>100 (0.40)	>100 (0.49)
R 20%, RR 10%	>100 (0.001)	>100 (0.07)	>100 (0.16)	>100 (0.12)	>100 (0.19)
R 20%, RR 10%, RS 5%	>100 (0.00)	>100 (0.00)	>100 (0.09)	>100 (0.10)	>100 (0.10)
			Low concentration		
No fitness costs	48 (1.00)	41 (1.00)	26 (1.00)	18 (1.00)	17 (1.00)
R 10%, RR 10%	>100 (0.00)	>100 (0.00)	>100 (0.065)	>100 (0.36)	>100 (0.39)
R 10%, RR 10%, RS 5%	>100 (0.00)	>100 (0.00)	>100 (0.00)	>100 (0.0002)	>100 (0.0006)
R 20%, RR 10%	>100 (0.00)	>100 (0.00)	>100 (0.00)	>100 (0.0004)	>100 (0.0007)
R 20%, RR 10%, RS 5%	>100 (0.00)	>100 (0.00)	>100 (0.00)	>100 (0.00)	>100 (0.00)

These fitness costs were applied for individuals on cotton plants not treated with pyriproxyfen and during four overwintering generations on noncotton plants. The R allele frequency in year 100 is shown in parentheses. Values of 0.00 indicate that the R allele frequency decreased below 10^{-8} after 100 yr.

R allele frequency did not reach 0.5 within 100 yr (Table 4). With a low concentration, the R allele frequency never reached 0.5 within 100 yr with any fitness cost simulated. Results were similar with a medium and high concentration, although there were some cases where the R allele frequency reached 0.5 within 100 yr. However, even when the R allele frequency reached 0.5 within 100 yr, the R allele frequency reached a limit (Table 4). Thus, if fitness costs impact survival on cotton and overwintering crops, the R allele frequency may reach a limit when increases in R allele frequency due to selection with pyriproxyfen is equal to decreases in R allele frequency due to fitness costs.

Discussion

Model results showing that whitefly resistance to pyriproxyfen can evolve in <20 yr, especially with high concentrations (Fig. 2), are consistent with decreases in susceptibility observed in the field in <10 yr in Arizona (Li et al. 2003, Dennehy et al. 2004) and Israel (Horowitz et al. 1999, Horowitz et al. 2005). In addition, *B. tabaci* strains exhibited reduced susceptibility in <10 yr to other insecticides (Prabhaker et al. 1992, Bloch and Wool 1994, Horowitz and Ishaaya 1994, Cahill et al. 1996).

Resistance evolved in our simulations partly because resistant individuals constantly moved into refuges. In the treated field, the effects of pyriproxyfen lasted 28 d. Surviving resistant individuals reproduced and moved into the refuge for the rest of the season. Thus, although resistance evolved faster in treated fields than refuges, the R allele frequency built up rapidly in refuges. In addition, because refuges were treated with insecticides other than pyriproxyfen, densities in refuges were generally similar to densities in fields treated with pyriproxyfen. This decreased the impact of susceptible individuals moving from refuges into treated fields. Thus, resistance to pyriproxyfen might be delayed by increasing action thresholds in refuges compared with fields treated with pyriproxyfen.

In our simulations, resistance evolved faster with increases in dominance, initial allele frequency, and proportion of the region treated with pyriproxyfen. These results are similar to results from models of diploid insects (Tabashnik and Croft 1982, Roush 1989, Tabashnik 1990, Denholm and Rowland 1992). Resistance also evolved faster as insecticide concentration increased. However, contrary to the assumption used here, dominance is likely to decrease as concentration increases (Roush and Daly 1990, Carrière 2003, Tabashnik et al. 2004). Therefore, the differences between a high and low concentration may not be as great as results of this model suggest. Bioassays that determine dominance as a function of concentration are needed to address these issues.

Consistent with findings of Carrière (2003) but not with conclusions of Denholm et al. (1998), our results suggest that dominance in females can greatly affect resistance evolution in haplodiploid insects, especially as toxin concentration decreases (Fig. 2). Dominance is an important factor affecting the evolution of resistance in diploids (Onstad et al. 2001, Tabashnik et al. 2004, Crowder and Onstad 2005, Crowder et al. 2005), another similarity between haplodiploid and diploid systems.

The minor effects of movement between the treated and untreated fields seen here differ markedly from previous modeling studies (Peck et al. 1999; Caprio 2001; Ives and Andow 2002; Storer 2003; Sisterson et al. 2004, 2005). In our model, pyriproxyfen applications killed whiteflies only on leaves present at the time of application. As new leaves developed, refuges expanded within treated fields. Thus, movement between treated and untreated leaves within fields treated with pyriproxyfen may have affected resistance more than dispersal between fields.

The proportion of male eggs laid by mated females affected the results with a low concentration. This result may be due to differential effects of sex ratio on population dynamics in treated fields and refuges. Although PM was the same in both treated fields and refuges, the effects of PM varied between the two field types. Accordingly, PM modulated the evolution of resistance through its differential effects on population dynamics in both fields, which affected selection intensity and refuge efficacy. These trends did not occur with a high or medium concentration, because population effects mediated by sex ratio became less important as the efficacy of pyriproxyfen and other insecticides applied in refuges became more comparable. Although we cannot fully explain the effects of PM on resistance, the evolution of resistance may vary across regions with different sex ratios.

Our results and those of Carrière (2003) show that increased susceptibility of R males compared with RR females slows evolution of resistance in haplodiploid insects. In this case, the ratio of resistant to susceptible males surviving exposure to pyriproxyfen was lower than with the standard case, which delays resistance. However, we found here that resistance evolved faster with increased susceptibility of S males compared with SS females. In this case, the ratio of resistant to susceptible males surviving exposure to pyriproxyfen was higher than with the standard case, which accelerates resistance. In haplodiploid insects, both susceptible and resistant males are often more susceptible than their female counterparts (Carrière 2003). The modeling results here suggest that when both resistant and susceptible males have increased susceptibility compared with their female counterparts, the two opposing effects cancel each other.

Similar to previous results from models of diploid and haploid insects (Carrière and Tabashnik 2001, Carrière 2003, Tabashnik et al. 2005), fitness costs delayed resistance. The effects of fitness costs were strongest when they occurred over the winter as well as on cotton not treated with pyriproxyfen (Tables 3 and 4). When fitness costs affected survival over the winter, the evolution of resistance was greatly delayed or even prevented (Table 3). However, observed trends of reduced susceptibility in the field (Li et al.

2003, Dennehy et al. 2004) contradict this prediction, which implies the assumptions in this case are overly optimistic. Consistent with Carrière (2003), if fitness costs were higher in males than in females, resistance was further delayed. In principle, fitness costs can cause the R allele frequency to reach an equilibrium between mutation and selection (Carrière and Tabashnik 2001). In our model, this did not occur, because we assumed no mutation.

We modeled a simplified habitat in which cotton was the dominant crop, and the R allele frequency on other crops had no effect. Because some regions in Arizona and elsewhere have a diverse mixture of crops that support *B. tabaci*, important effects of such crops may be missing from our model. In more diverse agricultural regions where cotton fields receive more insecticide treatments than other crops hosting whiteflies, the other crops may help to delay resistance by increasing the size of external refuges. This effect might be particularly important at the beginning and end of the cotton growing season, when movement between cotton and other crops is most likely. Results suggest that for *B. tabaci*, which has relatively low movement between fields during the middle of the growing season, internal refuges in treated fields may be more important than external refuges.

We also made the simplifying assumption that resistance did not evolve to insecticides other than pyriproxyfen. In the long run, such resistance could evolve, thereby increasing the complexity of the situation. Ultimately, the durability of the scenarios explored here depends on management of resistance to all insecticides used against whitefly on cotton.

For nonsystemic insecticides such as pyriproxyfen, the timing of sprays may be important. Treating fields with pyriproxyfen during a period when cotton is developing fastest will likely delay resistance, because in-field refuges will develop quicker than during other times of the season. Growers may be able to prolong efficacy of pyriproxyfen against *B. tabaci* by varying their IRM practices, such as applying lower toxin concentrations, promoting susceptible populations in refuges, and optimally timing insecticide applications, as long as they maintain an economically beneficial IPM plan.

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