

**Pyrethroid resistance and esterase activity in selected laboratory populations of  
sweetpotato whiteflies *Bemisia tabaci* (Homoptera: Aleyrodidae)**

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**Toxicology, *Bemisia tabaci*, whiteflies, insecticide resistance, esterase activity**

**Abstract.** Selection for resistance to fenpropathrin (a pyrethroid insecticide) was performed by exposing adults of two field-collected populations of the whitefly, *Bemisia tabaci*, to increasing doses of fenpropathrin for four generations. Concurrently, samples of adults were used for colorimetric measurements of esterase (EST) activity.

The two populations differed in their response to selection. EST activity increased as a correlated response to pyrethroid selection in one population but not in the other. No changes in either resistance or esterase activity were detected in the unselected control lines. The data suggest that both resistance and EST activity have a genetic basis, and that the frequency of high EST-activity alleles may have been different in the two populations.

INTRODUCTION

Many recent studies on several important pest insect species describe correlations between total non-specific esterase (EST) activity and insecticide resistance (e.g. Mouches et al., 1986; Field et al., 1988; Byrne & Devonshire, 1991; Ferrari & Georgiou, 1991). Esterases are known to be involved in the detoxification of a number of organophosphates, carbamates (Devonshire & Moores, 1982) and pyrethroids (Ishaaya et al., 1987). However, Soderlund & Bloomquist (1990) in their review of molecular mechanisms of pesticide resistance, state that “despite the central role of esterases in pyrethroid metabolism in arthropods, very few instances are known in which resistance is linked clearly with enhanced esterase activity” (p. 69).

The association of resistance with activity of non-specific EST cannot by itself be proof that esterases are involved in the degradation of the pesticide. Also, not all EST isozymes are involved necessarily in pesticide metabolism: for example, 4 major EST isozymes are revealed by electrophoresis in the aphid *Myzus persicae* Sulzer (Homoptera: Aphididae) (Wool et al., 1978) but only one is known to be involved in pesticide degradation (Devonshire & Moores, 1982). Nevertheless, if a strong correlation between total EST activity and resistance can be demonstrated, the former may be used as a marker for resistant individuals in the field (e.g. Pasteur & Georgiou, 1981). The importance of such a marker increases when the frequency of resistant individuals is low, because the conventional dose-mortality tests for resistance then require large sample sizes and are very inefficient (Roush & Miller, 1986).

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The whitefly *Bemisia tabaci* (Gennadius) emerged as a primary cotton pest in many countries in the last 30 years (see reviews by Dittrich et al., 1990a; Byrne et al., 1990). Heavy use of pesticides is often blamed for whitefly outbreaks (Dittrich, 1987; Ahmed et al., 1987).

EST activity was correlated positively with pyrethroid and organophosphate resistance in *B. tabaci* populations from Sudan, Guatemala and Nicaragua (Dittrich et al., 1990b). Mean EST activity in samples from field populations in Israel was considerably lower than in samples from control populations, unexposed to pesticides (Wool & Greenberg, 1990). However, low mean and skewed distribution of EST activity need not be proof that a genetic change in frequencies of genotypes has occurred in the field population, and the effect of other causes cannot be ruled out (Byrne & Devonshire, 1991; Bloch & Wool, 1992).

To evaluate the correlation of EST activity and resistance, it is desirable to understand the mode of inheritance of the two characters. Insecticide resistance is often assumed to be inherited monofactorially (Roush & McKenzie, 1987), but some studies published recently criticise the conventional method of testing inheritance models and suggest that inheritance of insecticide resistance may often be polygenic (Tabashnik, 1991; Firko, 1991; Firko & Hayes, 1991).

The present work was designed to study the response of *B. tabaci* to selection for pyrethroid resistance to the synthetic pyrethroid fenpropathrin and the association between resistance levels and EST activity, when both variables are considered to be quantitative characters. The study was performed in parallel to Bloch & Wool's (1992) investigation of resistance to the organophosphorous insecticide methidathion, using the same source populations and similar (but not identical) methodology.

#### MATERIAL AND METHODS

**INSECTS.** Approximately 6000 field collected adult whiteflies were used to establish each of two laboratory base populations. Population AM was collected in greenhouses of commercial cucumbers at Achituv, approx. 50 km north-east of Tel Aviv. These vegetables had been sprayed heavily with pyrethroids (including fenpropathrin) and other insecticides. Population GZ was sampled from cotton in a greenhouse at Tel-Aviv University. No insecticides were used in this greenhouse, but it was colonised in the past with adults that had been collected in commercial, insecticide-treated cotton fields. Populations AM and GZ were maintained in a rearing room in plastic cages (36 × 27 × 32.5 cm) with glass cover and 4 screened ventilation holes in the sides. Each cage contained 6 cotton seedlings in small pots, which were watered twice weekly. Plants were replaced every 3–4 weeks. Temperature in the room in winter was  $27 \pm 2^\circ\text{C}$ , and in summer  $30 \pm 2^\circ\text{C}$ , and humidity was  $70 \pm 15\%$  RH.

**INSECTICIDE.** We used the pyrethroid fenpropathrin (technical grade; SUMITOMO Japan) which is used currently for whitefly control in Israel.

**RESISTANCE MEASUREMENTS.**  $LC_{50}$  of the two populations before selection was calculated from log dose – probit mortality regression lines. Samples of 50 adults were exposed in glass 50 mm diam. Petri dishes to *Hibiscus* sp. leaves dipped in acetone solutions of the insecticide at different concentrations (Bloch & Wool, 1992). There were 4 samples per insecticide concentration and 7 concentrations per regression line. Mortality was determined after an exposure period of 24 hours and corrected for control mortality (on leaves dipped in the solvent only) by Abbott's formula (as cited in Brewer & Trumble, 1991).

**ARTIFICIAL SELECTION FOR RESISTANCE TO FENPROPATHRIN.** Three replicate selection lines and three controls were established from each base population. About 1500 adults were used to initiate each line. In every generation of selection, the selected lines were exposed to fenpropathrin treatment at a single discriminating dose, and the surviving adults were transferred to untreated cotton plants for oviposition. The control lines were transferred without insecticide treatment. The discriminating doses were chosen according to

mortality in the preceding generation and had to be increased when mortality became very low. In population GZ, the selective dose was increased from 0.03% fenpropathrin in generations 0, 1 and 2 to 0.1% in gen. 3–4. In population AM the dose was increased from 0.075% fenpropathrin in gen. 0 to 0.5% in gen. 1 and 1% in gen. 3.

It was not possible to collect sufficient numbers of adults to estimate  $LC_{50}$  during the selection experiment (see results). It was undesirable to allow the adults to reproduce more than one generation between treatments (to increase population size) since relaxing selection would have slowed down the development of resistance. Therefore the response of the selected lines to selection was measured as percentage mortality at the single, selective dose.

The selection procedure was not the same in all lines. Selection in the first generation in all 4 selected lines was performed by treating the cotton plants with the insecticide. This procedure was continued in GZ lines for further 2 generations (gen. 2 and 3). Thereby both adults and progeny were exposed to the pesticide. The reason for adopting this procedure was to intensify the selection pressure and to mimic field treatment more closely. Live adults were counted 1 week after the treatment on the plants. Since the number of introduced adults was known, mortality was estimated as one minus the proportion of adults still alive after 7 days on treated plants. This mortality rate was also corrected for mortality on control plants. In AM lines, from gen. 2 onwards – and in GZ from gen. 4 – selection was performed in Petri dishes as described above.

Each generation, 10 samples of 50 adults from the control lines were exposed to 0.015% fenpropathrin (half the selective dose at gen. 0). Mortality was determined 24 hours later. Control adults surviving treatment were not used in further work.

We also measured EST activity (see below) in 50–100 females from the control and selected lines every generation to find out if EST activity and fenpropathrin resistance were correlated.

**HERITABILITY OF RESISTANCE.** Our original plan was to calculate heritability by the method suggested by Tabashnik (1992). [We were not aware of this paper at the time of writing, but had used the same method in another paper now in press (Bloch & Wool, J.E.E.)]. The low population sizes precluded its use here. Thus,  $h^2$  was estimated by the assumption that resistance to fenpropathrin is a quantitative genetic character with an (unobservable) normal frequency distribution [referred to as “incidence” (Falconer, 1981) or “liability” (Hartl & Clark, 1989, pp. 481–483)], which is expressed as a threshold character with a binomial phenotype in individuals (“live” or “dead” when the insecticide is applied). If this assumption is true, then realised heritability may be estimated from comparisons of mortality at a selective dose, in the parental population and in their offspring (Hartl & Clark, 1989, pp. 481–483). This requires the additional assumption (which may not be justified, see Discussion) that the variances of liability in parental and offspring generations are equal (i.e., only the mean is changed by selection).

**EST ACTIVITY ASSAY.** EST activity was measured colorimetrically as in Wool & Greenberg (1990). Sample sizes were 50–100 adults in each test. Each adult whitefly was homogenised in 50  $\mu$ l of 40 mM phosphate buffer, pH 7.0 containing 0.01% (W/V) of Triton X-100. The reaction mixture contained 10  $\mu$ l homogenate, 20  $\mu$ l of 0.1 mM  $\beta$ -naphthyl butyrate solution as substrate and 470  $\mu$ l phosphate buffer. The mixture was incubated for 30 min at 37°C and the reaction was terminated by adding 100  $\mu$ l, 1% O-dianisidine solution containing 5% SLS. Absorbance of the red colour was read at 545 nm with a Brinkmann PC-800 colorimeter. The absorbance values were converted to microgram  $\beta$ -naphthol per individual, using a calibration curve based on several concentrations of  $\beta$ -naphthol.

**HERITABILITY OF EST ACTIVITY LEVELS.** Four inbred lines originating from 4 pairs of virgin adults from population GZ were reared on cotton plants in cages until F4. The adults of F4 were allowed to oviposit for 2 weeks on fresh cotton seedlings. Then all adults were collected and frozen for EST activity assay. When offspring emerged, they were collected and frozen similarly. EST activity was measured in 8 or 9 families (females and their offspring) from each line. (Most of the male parents were dead when the insects were collected, so that regression on mid-parent, although desirable, was not possible). Heritability was estimated from daughter – mother regression (Falconer, 1981). The genetic component in the total variance of esterase activity in female and male offspring was estimated from nested ANOVA (Sokal & Rohlf, 1981).

**INHIBITORY EFFECT OF FENOPROPATHRIN ON EST ACTIVITY.** Samples of 50 adult whiteflies were exposed to fenpropathrin-treated *Hibiscus* leaves in glass Petri dishes (as described above). Samples from population

AM were exposed to two concentrations, 0.015% and 0.1% fenpropathrin. Adults from population GZ – to 0.015% only. Mortality was determined after exposure periods of 3, 5, 8, 17 and 24 hours, 4 replicates per period. Adults surviving the treatment were collected and frozen for EST activity assay.

## RESULTS

**SENSITIVITY TO FENPROPATHRIN BEFORE SELECTION.** Mortality from exposure to fenpropathrin in population AM and GZ before selection was very similar ( $LC_{50}$  values in Table 1). A test for homogeneity of regression slopes (Sokal & Rohlf, 1981) indicated, however, that the slopes of the AM lines were lower significantly than the GZ lines (Table 1). Replicate slopes within populations were not different significantly from each other. (Test for combined probabilities of all pairwise tests of AM versus GZ lines (Sokal & Rohlf, 1981)  $X^2 = 23.0$ , 8 df. ( $P < 0.005$ )). The lower slope in AM than GZ indicates that the former population was more heterogeneous genetically in resistance than the latter.

TABLE 1. Resistance of *B. tabaci* populations to fenpropathrin before selection (7 doses tested per line, 8 samples of 50 adults per dose).

Populations	Number of regression lines	Range of doses	$LC_{50}$ (% fenpropathrin)	Slope $\pm$ SE
Tel-Aviv Univ. (GZ)	2	0.005 – 0.020	0.020	$1.155 \pm 0.224$
			0.039	$1.290 \pm 0.366$
Moshav Achituv (AM)	2	0.001 – 0.500	0.032	$0.400 \pm 0.114$
			0.041	$0.466 \pm 0.325$

**SELECTION FOR RESISTANCE TO FENPROPATHRIN.** Selection by treating the cotton plants was much more effective than exposure of adults to dipped *Hibiscus* leaves. For example, mortality on plants treated with 0.03% fenpropathrin was 99.5% in selected GZ lines (5 surviving adults of 1088 treated) but only 44.9% in the Petri dishes on leaves treated with the same concentration. The narrow bottlenecks did not cause extinction: two reproductive generations after the bottleneck the populations recovered their former size.

TABLE 2. Range of mortality on 0.015% fenpropathrin in the control populations at the beginning and end of the selection experiment (10 replicates per test).

Population	Generations 0–1	Generations 4–5
GZ	$42.63 \pm 5.24$	$41.20 \pm 2.03$
AM	$36.21 \pm 6.28$	$36.62 \pm 4.79$

Figure 1 shows the progress of selection in GZ and AM lines. It was necessary to almost triple the selective dose for GZ lines in generation 2 (Fig. 1a) because mortality became very low. The selective dose in selected AM lines was increased in generation 2 and 3 for the same reason (Fig. 1b).

Response to selection is demonstrated also by comparing mortality of adults after one generation of selection ( $F_1$ ) to mortality of their parents ( $F_0$ ) when exposed to the same concentration of fenpropathrin (Table 3). In all cases, offspring mortality was lower than parental mortality on the same dose.

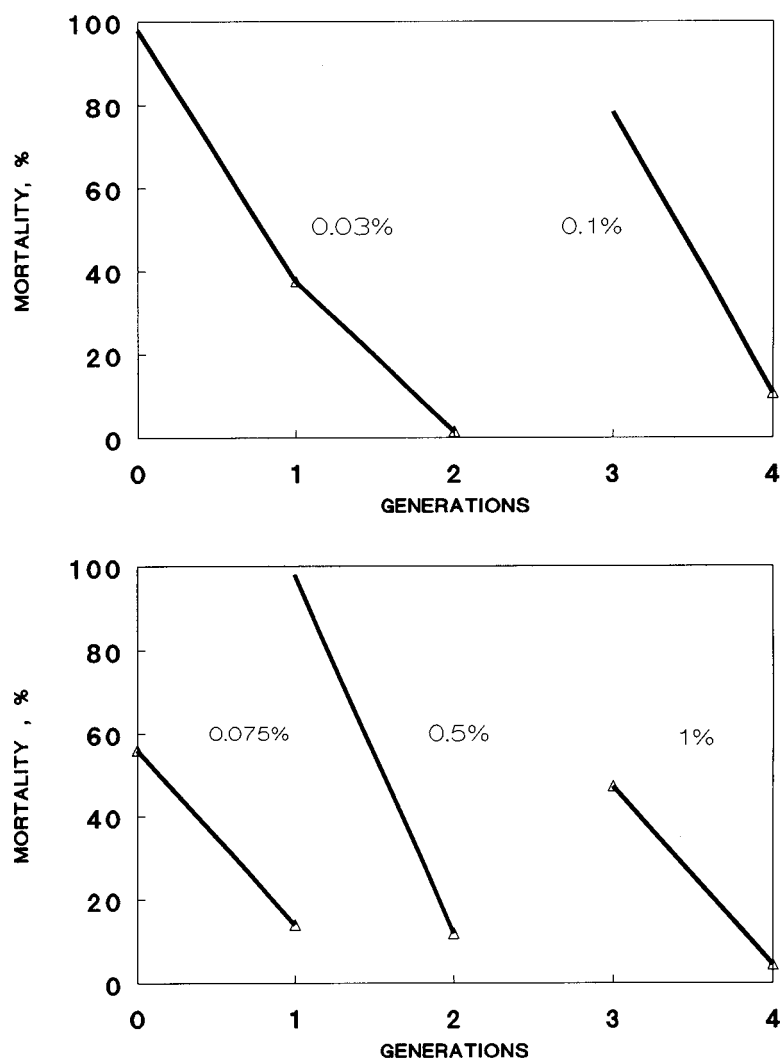


Fig. 1. Percentage mortality of *B. tabaci* adults exposed to the selective dose of fenpropathrin. Upper – population GZ, lower – population AM. The selective dose had to be increased 2–3 times because mortality became very low.

Mortality of adults from unselected lines when exposed to 0.015% fenpropathrin (half the selective dose in gen. 0) did not change greatly between the beginning and the end of the selection experiment (Table 2).

**HERITABILITY OF FENPROPATHRIN RESISTANCE.** Heritability ( $h^2$ ) of a quantitative trait is best estimated in the first generation of selection, because heritability may change as a consequence of selection itself and the inevitable inbreeding associated with the use of small

samples of selected offspring to continue each line. Reliable estimates of  $h^2$  may be useful for prediction of selection response using the relation  $R = h^2S$  where  $S$  is the selection differential (Falconer, 1981).

We calculated  $h^2$  for all parent-offspring pairs in gen. 1 (Table 3). Unfortunately, the values obtained did not appear to be very useful. The values of  $h^2$  for GZ lines were outside the theoretical range for  $h^2$  (0–1.0) and no biological meaning can be attached to them. The values for AM are valid but there were large differences among the 3 replicates (Table 3).

TABLE 3. Mortality of parents ( $F_0$ ) and their offspring ( $F_1$ ) after one generation of selection, when exposed to the same fenpropathrin concentrations. (Sample size in parentheses.)

Population	Selective concentration (in ‰)	Mortality		Heritability $h^2$
		$F_0$	$F_1$	
AM (1)	0.075	0.592 (977)	0.298 (124)	0.53
(2)	0.075	0.379 (1155)	0.259 (340)	0.39
(3)	0.075	0.379 (1155)	0.162 (204)	0.10
GZ (1)	0.005	0.444 (788)	0.321 (467)	> 1
(2)	0.005	0.585 (409)	0.548 (299)	> 1
(3)	0.030	0.995 (1087)	0.430 (193)	negative

Two important assumptions are involved in the calculation of  $h^2$  from parent-offspring mortality data such as ours: (I) that the distribution of “liability” (the genotypic distribution underlying the binomial phenotypic expression) is normal. This assumption cannot be tested, and may be wrong; (II) that the variances of liability in the offspring population are the same as in their parents. We cannot measure the variance in liability directly, but we can measure the variance in its phenotypic expression among replicate samples from each parental and offspring population (Table 4).

TABLE 4. Tests for equality of parental and offspring variances of mortality, among samples of adults of each population exposed to the same concentration of fenpropathrin. (Two-tailed tests. Sokal & Rohlf, 1981)

Population	Parents		Offspring		F
	variance	df	variance	df	
AM 1	169.50	25	87.21	8	1.94 <i>ns</i>
2	169.49	24	126.86	10	1.34 <i>ns</i>
3	169.49	24	294.42	7	1.74 <i>ns</i>
GZ 1	247.50	19	299.17	12	1.21 <i>ns</i>
2	251.35	22	195.97	10	1.18 <i>ns</i>
3 – only 1 replicate					

The equality of offspring and parental variance in mortality was tested by two-tailed F tests (Sokal & Rohlf, 1981, ch. 8). In no case was there a significant difference between the paired variances ( $p > 0.05$ ).

EST ACTIVITY LEVELS DURING SELECTION FOR FENPROPATHRIN RESISTANCE. Esterase activity, as measured from the hydrolysis of  $\beta$ -naphthol, was affected directly by exposure of the adults to fenpropathrin. Table 5 summarises the data from five repeated trials, in which

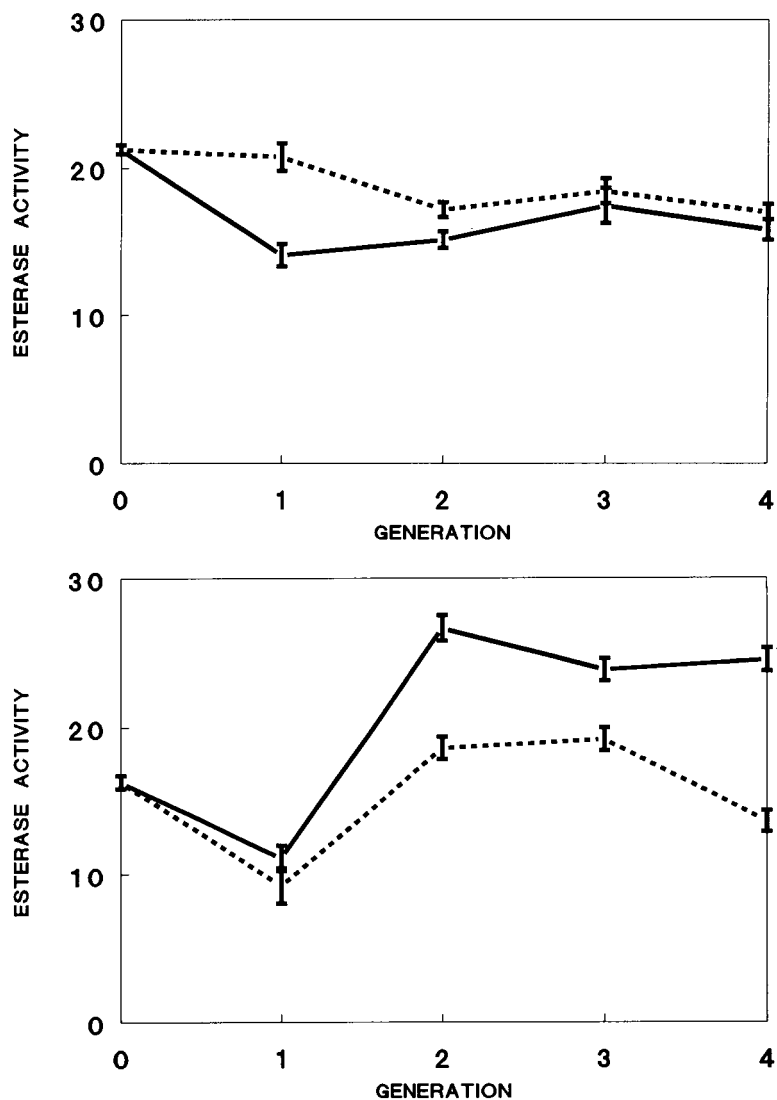


Fig. 2. Mean esterase activity (microgram  $\beta$ -naphthol/individual) during selection for resistance to fenpropathrin. Upper – population GZ, lower – population AM. Solid line – selected populations; broken lines – unselected control (means  $\pm$  SE of three replicate lines in each case).

adults were exposed to treated leaves for periods of 3–24 hours, and the survivors frozen and analysed for EST activity. Although the differences in mean activity within each trial are not very large, and many comparisons among means are not significant statistically (ANOVA followed by a-posteriori tests; Sokal & Rohlf, 1981), most of the means of treated adults are lower than control means (differences significant at  $P < 0.01$ ), and there is a (slight but discernible) trend of decrease in EST activity with the increase in duration

of exposure. Table 5 also indicates that EST activity in AM males was lower than in females. This reflects perhaps the smaller size of males.

TABLE 5. Esterase activity in *B. tabaci* adults surviving exposure to fenpropathrin on treated leaves. (Units: microgram  $\beta$ -naphthol/individual). For brevity, data for 5 and 8, and for 17 and 24 hours exposure were combined. Data are means  $\pm$  standard errors (n = sample size).

Treatment	Time	Esterase activity	
		Males	Females
Population AM			
0 (control)		15.98 ± 0.942 (32)	16.28 ± 0.449 (172)
0.015%	3h	15.18 ± 1.215 (64)	19.61 ± 0.832 (54)
0.015%	5h, 8h	11.87 ± 0.890 (64)	15.20 ± 0.515 (129)
0.015%	17h, 24h		14.60 ± 0.880 (96)
0 (control)		12.54 ± 1.142 (32)	
0.1%	3h	9.67 ± 0.744 (51)	19.81 ± 0.811 (80)
0.1%	5h, 8h	7.76 ± 0.486 (68)	15.36 ± 0.655 (83)
0.1%	17h, 24h		15.09 ± 0.853 (96)
Population GZ			
0 (control)			21.27 ± 0.307 (218)
0.015%	3h		18.62 ± 0.528 (92)
0.015%	5h, 8h		20.65 ± 0.290 (144)
0.015%	17h, 24h		17.35 ± 0.535 (95)

The changes of mean EST activity in the selected populations and the untreated controls are illustrated in Figure 2a,b. Mean EST activity of the selected population GZ was significantly lower than its control at the start ( $P < 0.001$ ) but the difference decreased during selection and was not significant in the end (Fig. 2a). In contrast, mean EST activity in selected population AM was consistently higher than its control, and the difference increased and became statistically significant ( $P < 0.001$ ) in gen. 4 (Fig. 2b).

HERITABILITY OF EST ACTIVITY LEVELS. Daughter – mother regression in the 4 lines produced a mean slope  $b = 0.152 \pm 0.102$ ; therefore, a heritability estimate of  $h^2 = 2b = 0.3$ . However, the predictive value of this estimate is small because the slopes were not different significantly from zero ( $P > 0.05$ ), and the estimates are based on small samples.

The variance component of EST activity among inbred groups was nonsignificant in females when analyzed by ANOVA. In males, 27.3% of the total variance ( $P < 0.01$ ) was due to differences among inbred lines.

#### DISCUSSION

Fenpropathrin is often used against *B. tabaci* in Israel. Hence it was important to discover whether its continuous use could cause the appearance of resistance. Laboratory selection for increased fenpropathrin resistance was effective in populations AM and GZ: mortality at the selective dose was quickly reduced to very low levels after each dose increase (Fig. 1). Therefore, it was puzzling why the level of fenpropathrin resistance (before selection), as measured by  $LC_{50}$  was similar in AM – heavily treated with pesticides in the greenhouse (including fenpropathrin) and GZ, which was not treated. The reason why AM was no more resistant than GZ may be that farmers in Israel are advised to

alternate the chemicals often and not use any one pesticide consistently. This strategy delays the evolution of resistance. Such alternation of chemicals could also account for the greater variance in resistance in AM (Table 1), if different chemicals select for different genotypes of *Bemisia*.

Before selection, the difference in slope of the dose-mortality regression lines between population AM and GZ (Table 1) suggested a greater potential for the development of fenpropathrin resistance in AM. The small numbers of survivors in the present experiment precluded the calculation of  $LC_{50}$  from dose – mortality regression every generation, including the last. To follow the progress of resistance and estimate heritability, we had to use % survival of parents and offspring at a single, selective dose. We could also not compare the progress of selection of AM and GZ because the selection procedure was not the same (see Methods). Still, the greater genetic variance in AM appears to be supported by the measurements of response of the correlated trait, EST activity, to fenpropathrin resistance in the selected lines (see below).

Given the required genetic variation, the response to selection depends on selection pressure. This factor has at least two components: 1) selection intensity, i.e. the proportion of insects killed, and 2) the interval between treatments, which depends, in turn, on the insect reproductive rate (i.e., how fast it recovers and reaches a large enough population size that justifies another treatment). The two components are not independent: an effective treatment which leaves very few survivors may slow down, rather than accelerate, the development of resistance (unless all survivors are homozygous for the relevant resistance alleles) because it may take more than one generation for the population to attain a large enough size for a repeat of the treatment. This delay will introduce another complication because survivors may have a chance to mate with their offspring (i.e., increasing the level of homozygosity, accelerating the evolution of resistance) or with migrants from other fields (i.e., increasing the level of heterozygosity and, perhaps, delaying the evolution of resistance).

Our attempts to estimate the heritability of fenpropathrin resistance, assuming a normal distribution of “liability” (Hartl & Clark, 1989) underlying the binomial phenotype of response, have yielded less than satisfactory results. The  $h^2$  estimates for GZ proved to be unacceptable ( $> 1.0$ ). For AM, replicate lines gave very different estimates ( $h^2 = 0.1-0.53$ ; Table 3). Nevertheless, both the response to selection (Fig. 1), and the fact that offspring mortality after one generation of selection was invariably lower than in the parents (Table 3), indicate that resistance to fenpropathrin is, at least in part, heritable.

**ESTERASE ACTIVITY.** The results of the present study, as well as Bloch & Wool (1992), suggest that there is some association between EST activity and resistance in Israeli *B. tabaci*. The increase in mean EST levels in the selected lines, but not in their parallel control lines, indicates that the increase in EST is not due to the common experimental environment nor to inbreeding. Selection could have produced this result due to some functional relatedness of EST activity and resistance maintaining a linkage disequilibrium between the loci coding for the two traits. It is interesting to note that AM lines responded to fenpropathrin resistance by an increase of EST activity (above the control line) while, in GZ lines, there was no significant change. This seems to reflect the higher genetic variance in AM. In *B. tabaci*, some studies reported positive correlations between EST activity and resistance (Dittrich et al., 1990b; Ishaaya et al., 1987). In contrast, Wool & Greenberg

(1990) reported that resistant field populations had lower levels of mean EST activity than susceptible control, although they did not claim that resistance was due to these esterases.

EST activity variation in *B. tabaci* in Israel is unlikely to be due to variation in EST isozymes. In an intensive study of electrophoretic variation in *B. tabaci* from many localities and host plants in Israel, only one major EST locus was discovered (Wool et al., 1993a), unlike the situation in populations of the same organism, performed simultaneously in Colombia (Wool et al., 1993b). Individuals with high EST activity may be rare in our field populations, although their frequency may be increased by the use of insecticides (see Bloch & Wool, 1992). Variation in EST activity in this organism may be affected strongly by non-genetic factors: that EST activity may be directly reduced in individuals treated with fenpropathrin can be seen from our Table 5 (and see also Byrne & Devonshire, 1991). Differences in EST activity between males and females may also confound the results: activity in males is lower (Table 3; and unpublished data by S. Greenberg and G. Bloch).

The small change in EST activity in AM, and the absence of correlated response to selection in GZ (and the large variance in EST activity among individuals, as reported by Wool & Greenberg, 1990, and Bloch & Wool, 1992) combine to indicate that EST activity does not provide a useful marker for resistance to either insecticide in field populations of *B. tabaci*.

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