Inheritance and Fitness Consequences of Resistance to Fenvalerate in *Spodoptera exigua* (Lepidoptera: Noctuidae)

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ABSTRACT Inheritance and fitness consequences of resistance to fenvalerate in *Spodoptera exigua* (Hübner), the beet armyworm, were evaluated with inbred lines of fenvalerate-resistant and susceptible populations. Ancestors of the fenvalerate-resistant population were exposed to a selection regime of multiple insecticides in the wild and were subsequently selected intensely with fenvalerate in the laboratory. Tolerance distributions of six generations of known relationship, derived from crosses of the inbred lines and their offspring, were estimated using susceptibility data. Because the tolerance distributions of the parental and F_1 generations overlapped, discrimination between a polygenic and monogenic model of fenvalerate resistance inheritance was difficult. By fitting mean tolerance of these and the F_2 and two backcross generations to additive-dominance models, variation of mean tolerance among generations was adequately explained by a reduced polygenic model of five parameters that included epistatic effects of additive-by-additive and additive-by-dominance digenic interactions. Therefore, a polygenic model of inheritance seemed most plausible to explain fenvalerate resistance expression. Measurements of fecundity and development time differed among generations. Although an additive fitness cost explained a significant amount of the observed variation, resistance may be acting on these traits in a complex manner. Polygenic inheritance of resistance and fluctuating fitness effects because of changing patterns of fenvalerate use may contribute to the spatial and temporal variation in resistance seen in wild populations.

KEY WORDS Insecta, *Spodoptera exigua*, insecticide resistance, generation means analysis

Spodoptera exigua (Hübner), the beet armyworm, is a highly polyphagous lepidopteran that feeds on tomato, cotton, celery, lettuce, and many other cultivated and wild hosts (Metcalf & Flint 1962). As a consequence of pyrethroid use on its cultivated hosts, *S. exigua* has evolved resistance to several pyrethroids. For example, Chaufaux & Ferron (1986) reported a resistance ratio of 168 at the LC_{50} of deltamethrin for second instars of a Guatemalan strain compared with a susceptible French strain. Brewer et al. (1990) reported a resistance ratio of 22.5 for fenvalerate at the LC_{50} for adults of a Mexican strain compared with a susceptible Californian strain. In the same study, considerable variation was found in resistance in field populations sampled in nine regions from Monterey County, Calif., to the Guasave and Del Fuerte valleys of Mexico. This variation was associated with differences in fenvalerate use across these regions and the ability to control populations with insecticide. Fenvalerate resistance was a heritable trait, was variable in wild populations, and appeared to affect control efficacy.

A more complete understanding of the variation in resistance that occurs in wild populations requires analyses of inheritance and fitness consequences of resistance. Whitten & McKensie (1981) proposed that pesticide resistance evolving in an agricultural setting usually is controlled by one or few genes. Many studies have indicated that resistance of practical importance is under such control (Roush & Croft 1986 and citations therein). The expectation of single or multiple gene involvement is important, because mode of inheritance can affect the rate of change of the trait in a population. Supported by computer simulation, Plapp et al. (1979) showed that resistance is more gradual in polygenic systems than in monogenic systems. Firko & Hayes (1990) reviewed the application of quantitative genetic methods in explaining resistance variation attributable to single or multiple gene action. Pleiotropic effects of resistance on fitness traits are among the factors that can affect the stability of resistance in a population of mixed genotypes (Georghiou 1983).

In our study, we evaluated inheritance and fitness consequences of *S. exigua* resistance to fenvalerate as an aid in understanding why variation of resistance in wild populations was occurring. With this information, we may better assess the likelihood of fenvalerate resistance evolving to a severity associated with lack of agricultural control.

Materials and Methods

Strains. A susceptible strain of *S. exigua* was maintained without insecticide pressure on artifi-

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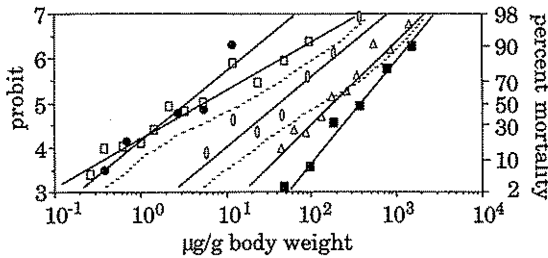

Fig. 1. Susceptibility responses of the parental and hybrid generations to fenvalerate. Generations are P_1 (●, susceptible), P_2 (■, resistant), F_1 (0, progeny from combined $P_1 \times P_2$ reciprocal crosses), B_1 (□, F_1 female × P_1 male), and B_2 (△, F_1 female × P_2 male). Solid lines are probit regressions. Dotted lines are the expectations for the B_1 and B_2, assuming a monogenic model of inheritance.

cial diet since establishment from collections made at Orange County, Calif., in 1982. Rearing conditions were described by Brewer & Trumble (1989). The susceptibility of this colony corresponded closely with that of a laboratory colony that had been continuously reared for 25 yr (Brewer & Trumble 1989). The Orange County strain was used as the parental susceptible generation (P_1) in crossing experiments.

A fenvalerate-resistant strain originated from 30 egg masses collected from a wild population on tomato in Colonia Guerrero, Baja California Norte, Mexico, in September 1987. The wild population had been exposed to a mixed insecticide selection regime of fenvalerate, permethrin, methomyl, and methamidophos. Because adults are highly mobile, it is likely that the population was exposed to other insecticides as well. For the wild population, a resistance ratio of 12 measured at the LC_{50} was documented using an adult susceptibility test (Brewer et al. 1990). The resistant strain and all generations derived from this strain were maintained in the laboratory under the same rearing conditions as the susceptible strain.

Homozygosity of the resistant strain was increased by selection of larvae. Beginning with the first laboratory generation, the strain was exposed to selection pressure through 19 generations by exposing third instars to topically applied fenvalerate (94%; E.I. du Pont de Nemours & Company, Wilmington, Del.) diluted in acetone. An Arnold (Type LV 65) microapplicator (Burkard, Rickmansworth, Herts., England) was used to deliver 0.5 μl to the thoracic dorsum of each of at least 660 larvae per generation. Selection pressure was increased in increments from 0.10 μg per larva at the first generation to 3.20 μg per larva at the 19th generation. Mortality ranged from 45 to 85%; survivors were used to establish subsequent generations. Results from this selection experiment were reported by Brewer et al. (1990). Briefly, resistance in larvae had increased 8.3-fold by generation 20, with the greatest increase in resistance occurring within the first six generations. Selections were not conducted in generations 20 and 21. At generation 22, resistance was not different than resistance in the 20th generation. Generation 22 also was selected (3.20 μg per larvae) after resistance status was determined. Overlap of concentration–response curves of generation 23 and the P_1 did not occur at 5 and 98% mortality, respectively (Fig. 1). Based on these data, we assumed the resistance strain was homozygous for resistance. Generation 23 was used as the parental resistant generation (P_2) in crossing experiments.

Hybrids were obtained by crossing the inbred parental generations. Two generations were obtained from reciprocal crosses of the P_1 and P_2 (two F_1s: P_2 female × P_1 male and P_1 female × P_2 male). One generation (F_2) was obtained by random inbreeding of the F_1. Two backcross generations were obtained from backcrosses of F_1 females to each parental generation (B_1: F_1 females × P_1 males, B_2: F_1 females × P_2 males). Maternal effects, if present, were detectable by comparing susceptibility of the two F_1s; therefore, reciprocal backcrosses were not done. For these crosses, pupae were separated by sex. Mating pairs of newly emerged moths were isolated in containers (0.48 liters) lined with green construction paper to serve as an oviposition site and provisioned with a source of liquid (10 ml of a 20% honey solution). For each cross, 15–20 pairs were randomly selected from the appropriate generation.

All life stages were reared at 27°C (with accuracy of ± 1°C) and a photoperiod of 16:8 (L:D) in the same environmental chamber to minimize complications from genotype-by-environment interaction. Eggs were collected every 24 h from each mating pair until death of the female. Egg masses were surface-sterilized and placed in 210-ml ice cream cups filled with 65 ml of artificial diet (Brewer & Trumble 1989). Each cup contained age-specific (daily) progeny of a female.

Inheritance of Resistance. Randomly selected third instars, weighing 3–5 mg, of each generation were tested for susceptibility to fenvalerate as described previously. Six test concentrations of fenvalerate and a control concentration (acetone alone) were used to test the P_1, P_2, and F_1. At least 10 concentrations were used to test the B_1, B_2, and F_2. Mortality after 24 h was recorded. Bioassays were replicated three times, and each replicate was completed on a different day. No fewer than 15 larvae per concentration were used for each bioassay. Bioassays of the P_1, P_2, and F_1 were done concurrently, as were bioassays of the B_1, B_2, and F_2. Each concentration was converted to a body weight basis (micrograms per gram body weight) using an average weight among replicates for each generation. Mortality data from each generation were pooled for probit analysis (POLO-PC; LeOra Software 1987). The χ^2 goodness-of-fit test was used to test the hypothesis that the probit model adequately

described the data. Probit analysis is a good model of dose–mortality data from homozygous populations (Finney 1971). It may also be an adequate model of data from heterozygous populations in which resistance is polygenic (Via 1986). Differences in susceptibility among generations were compared with likelihood ratio tests (POLO-PC). We use "LR" to report the likelihood ratio statistic, which follows a χ^2 distribution (POLO-PC), to avoid confusion with the χ^2 goodness-of-fit test.

The fit of the mortality data to polygenic and monogenic models of inheritance were compared. If probit analysis adequately described the data from the backcross generations and the F_2, polygenic inheritance of resistance may have contributed to this pattern of susceptibility. Under a monogenic model, a different pattern of susceptibility was expected. In this model, the resistant trait was assumed to be governed by one gene with two alleles, one wild (S) and one resistant (R). The genotypic expectations of the P_1, P_2, and F_1 were SS, RR, and RS, respectively, assuming homozygosity of the P_1 and P_2 for this trait. Susceptibility to fenvalerate in these generations would be adequately described by the probit model (whether resistance was monogenic or polygenic). However, in a monogenic model, the genotypic expectations of the B_1, B_2, and F_2 were composed of a mixture of genotypes: SS/RS in a 1:1 ratio for the B_1, RS/RR in a 1:1 ratio for the B_2, and SS/RS/RR in a 1:2:1 ratio for the F_2. Therefore, the B_1, B_2, and F_2 mortality data would be better described by Georghiou's (1969) model that corrects for heterogeneity of these generations. Departure of the data from this model was tested with the χ^2 goodness-of-fit test (Georghiou 1969).

If variation was polygenically controlled, a complementary approach to analyze mortality data among these generations was with generation means analysis. For this test, homozygosity of the P_1 and P_2 for all relevant loci was assumed. This test estimated genetic values of additive-dominance models using means of a quantitative trait obtained from generations of known relationship (Bulmer 1985, chapter 5). These estimates could be compared with the expected genetic values given by Bulmer (1985; Table 5.1). Bulmer (1985) showed the derivation of these expected genetic values for a six-parameter model, which included mean, additive, and dominance effects, and effects of digenic interactions (epistasis) between loci. Observed means weighted by their variances from six generations of known relationship could be used to estimate six or fewer parameters. Beaver & Mosjidis (1988) clarified the application of weighted least squares for this analysis. The assumption of normality was necessary for this analysis; therefore, the logarithms of mean tolerance (log[LD_{50}]) and the variances of these values were used as the means and variances in the analysis. These values were obtained from the probit procedure (SAS Institute 1985).

Reduced models, formed by deletion of parameters from the full model, were considered. The expected genetic values (parameters) included in the reduced models were selected from a hierarchy of genetic effects. The three-parameter genetic model was a simple additive-dominance model consisting of the mean (m), additive (a), and dominance (d) effects summed across loci. Four- and five-parameter models added digenic interactions (epistasis) of additive-by-additive effects (aa), additive-by-dominance effects (ad), and dominance-by-dominance effects (dd) in all appropriate combinations to the three-parameter model. Because no hierarchy of the digenic interactions was assumed, there were three models with four parameters and three models with five parameters. The full six-parameter model consisted of all effects. The minimum number of parameters necessary to explain the variation among generation means adequately was determined by testing the fit of the data to the reduced models using the χ^2 goodness-of-fit test (Beaver & Mosjidis 1988). The computer program GENEFF (Mosjidis et al. 1989) was used to calculate the estimates of the genetic values and the statistics used to compare the estimates with the expected values.

Life History. Data on fecundity, development time, pupal weight, and adult emergence were taken for each generation. The number of eggs laid were counted daily for 15–20 mating pairs before the eggs were placed in rearing cups. For each of 10 pairs, 30 larvae were randomly selected 5 d after egg hatch. Each larva was transferred into a 30-ml plastic cup filled with 8 ml of diet; it remained in the cup until pupation. Development time was recorded as number of days from oviposition to pupation. Pupae were collected and weighed individually. Their sex was determined and they were held in rearing containers. For those insects that pupated, development time and pupal weights were averaged for each mating pair. Percentage emergence was recorded for each set of larvae derived from a single mating pair.

A one-factor (generation) analysis of variance (ANOVA) was used to determine if the life history variables differed among generations. Before analysis, development time was transformed by the logarithm of the quantity (days + 1), and the proportion of adults emerging was transformed by the arcsine square root of the proportion. The means of each variable were weighted by the variance of the mean because the genetic expectations of variance among these generations differed (Bulmer 1985, chapter 5).

Variance from the weighted ANOVA was further partitioned by ordering the generation means: P_1, B_1, F_1, F_2, B_2, and P_2. Orthogonal comparisons were used to detect linear and quadratic trends across these ordered means. Equal spacing of means was assumed, except $F_1 = F_2$, according to their genetic expectations (Bulmer 1985). The equality of the F_1 and F_2 means was tested using a com-

Table 1. Probit regression statistics including mean tolerance and variance of the mean

Generation	n^a	Slope ± SE[b]	Tolerance		
			LD_{50} (95% CL)[c]	$\bar{x}^d$	Variance[e]
P_1	335	1.57 ± 0.27	3.36 (2.01–6.45)	0.526	0.00639
B_1	714	1.04 ± 0.10	3.45 (2.42–4.95)	0.538	0.00463
F_1	704	1.52 ± 0.24	44.7 (26.9–85.1)	1.65	0.00877
F_2	704	1.02 ± 0.066	13.0 (10.1–16.7)	1.11	0.00304
B_2	628	1.94 ± 0.14	189 (164–217)	2.28	0.000931
P_2	449	2.21 ± 0.19	388 (329–460)	2.59	0.00134

[a] Number of insects bioassayed.
[b] Slope of the probit line followed by the standard error.
[c] Mean tolerance measured in μg/g body weight followed by 95% fiducial limits.
[d] Log_{10} (LD_{50}).
[e] Variance of mean tolerance.

parison that was orthogonal to the linear and quadratic comparisons. Assuming that nonadditive genetic effects of resistance on these fitness components (e.g., dominance, epistasis) were absent and there was a fitness cost associated with the resistant trait, we expected the ordered means to follow a linear trend. Assuming that nonadditive genetic effects were involved, we expected the ordered means to deviate from a linear trend (tested with a quadratic comparison). A more elaborate generation means analysis (Bulmer 1985) that would give estimates of additive, dominance, and epistatic effects was not considered because the parental resistant and susceptible generations were not bred directly for these fitness components.

Differences in fitness components may be related to resistance factors or other unknown factors. Because the P_1 and P_2 were not derived from the same original population, separation of effects from resistance and unknown factors using data from these generations alone was difficult. As an aid to separate these effects, a control strain (C) was partitioned from the fenvalerate-resistant strain at the first laboratory generation. This strain was not selected with fenvalerate. At generation 20, susceptibility of this strain increased to levels near the P_1 (susceptibility did not differ significantly in two of three bioassays [Brewer et al. 1990]). Fitness components of the P_1, P_2, and C were compared under the same experimental and sampling criteria described previously in this section. Fitness of the C and P_1 were expected to be similar and fitness of the C and P_2 were expected to differ if the primary factor responsible for fitness differences was resistance. A weighted ANOVA was used, as described above, and was followed by comparisons of the C means with the P_1 and P_2 means.

Results

Inheritance. Control mortality was ≤5% in all bioassays. The P_1 and P_2 differed in mean tolerance to fenvalerate by 115-fold at the LD_{50}. Crosses yielded hybrids with tolerance levels between the parental tolerances (Table 1). No maternal effect was detected in the reciprocal F_1's, as indicated by similar dose–mortality lines (LR = 4.58; df = 2; $P > 0.05$); therefore, these data were combined to estimate one probit line (Table 1).

The dose–mortality lines of the P_1, P_2, and F_1 significantly differed (LR = 403; df = 4; $P < 0.001$) (Table 1, Fig. 1). Susceptibility of the B_1 and B_2 was within the range of susceptibility bounded by the F_1 response and the respective parent (Table 1, Fig. 1). The data were described adequately by the probit model for the B_1 ($\chi^2 = 18.1$; df = 10; $P > 0.05$) and B_2 ($\chi^2 = 9.04$; df = 9; $P > 0.25$), suggesting polygenic inheritance of resistance (Via 1986). Based on Georghiou's (1969) model, significant departures of the data from expectations of a monogenic model of inheritance were detected for the B_1 ($\chi^2 = 76.9$; df = 10; $P < 0.005$) but not for the B_2 ($\chi^2 = 15.4$; df = 9; $P > 0.05$) (Fig. 1). Departure of B_1 data from a monogenic model was largely due to higher-than-expected susceptibility of this generation across the entire range of doses tested. Data from the F_2 did not aid in discriminating between these two models (probit statistics given in Table 1).

In generation means analysis, a three-parameter model was not sufficient to explain the variation of mean tolerance among the generations ($P < 0.001$, Table 2). Therefore, digenic interactions (epistasis) may be important if variation was under polygenic control. The five-parameter model that added aa and ad interactions did explain the variation among generation means adequately ($0.3 < P < 0.5$; Table 2). The dominance estimate in this model was positive, indicating that the sum of individual dominance effects favors expression of resistance. Fit of the data to all other reduced models was poor ($P < 0.001$; Table 2).

Life History. Life history data of the reciprocal F_1's did not differ ($P > 0.20$), and the data were combined before comparison with other generations. Of the data taken as measures of fitness, fecundity ($F = 6.13$; df = 5, 97; $P < 0.0001$) and development time ($F = 11.41$; df = 5, 61; $P < 0.0001$) differed among the six generations (Fig. 2). Mean fecundity and development time of the F_1 and F_2 did not differ significantly ($P > 0.05$). A linear trend was detected for fecundity ($F = 12.86$;

Table 2. Genetic parameter estimates and standard errors based on mean tolerance (log[LD_{50}]) and variance of the mean for three-, four-, five-, and six-parameter genetic models

Parameter[a]	Estimates: $\bar{x} \pm$ SE for model of ( ) parameters							
	(3)	(4)	(4)	(4)	(5)	(5)	(5)	(6)
m	1.39 ± 0.040	0.675 ± 0.14	1.48 ± 0.041	1.40 ± 0.041	0.592 ± 0.14	1.56 ± 0.044	−0.433 ± 0.25	0.384 ± 0.27
a	−1.27 ± 0.037	−1.24 ± 0.038	−1.08 ± 0.043	−1.27 ± 0.037	−1.03 ± 0.044	−1.03 ± 0.044	−1.21 ± 0.038	−1.03 ± 0.044
d	0.182 ± 0.077	1.23 ± 0.22	−0.179 ± 0.086	0.0317 ± 0.17	1.10 ± 0.22	−1.06 ± 0.20	4.10 ± 0.58	1.65 ± 0.65
aa	—	0.777 ± 0.15	—	—	0.973 ± 0.15	—	1.87 ± 0.25	1.17 ± 0.27
ad	—	—	−1.36 ± 0.15	—	−1.49 ± 0.15	−1.66 ± 0.16	—	−1.41 ± 0.17
dd	—	—	—	0.217 ± 0.22	—	1.15 ± 0.24	−2.02 ± 0.38	−0.388 ± 0.42
	$\chi^2 = 123$	$\chi^2 = 95.6$	$\chi^2 = 42.4$	$\chi^2 = 122$	$\chi^2 = 0.834$	$\chi^2 = 19.4$	$\chi^2 = 66.5$	—
	df = 3	df = 2	df = 2	df = 2	df = 1	df = 1	df = 1	—
	$P < 0.001$	$P < 0.001$	$P < 0.001$	$P < 0.001$	$0.3 < P < 0.5$	$P < 0.001$	$P < 0.001$	—

The minimum number of parameters necessary to adequately explain the variation among generation means was determined by testing the fit of the data to the reduced models (Beaver & Mosjidis 1988). The observed χ^2, degrees of freedom (df), and significance level (*P*) follow the parameter estimates of each reduced model.

[a] *m*, mean; *a*, additive; *d*, dominance; *aa*, additive-by-additive; *ad*, additive-by-dominance; *dd*, dominance-by-dominance effects.

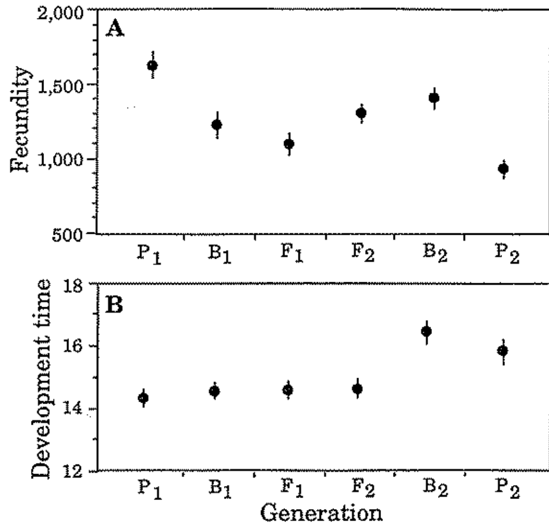

Fig. 2. (A) Differences in fecundity (number of eggs laid) among generations; mean square error is 827,709,013. (B) Differences in development time (days from oviposition to pupation) among generations (data transformed by [logarithm {days + 1}] for analysis); mean square error is 0.00000106. Generations are the P_1 (susceptible), P_2 (resistant), F_1 (combined $P_1 \times P_2$ reciprocal crosses), F_2 ($F_1 \times F_1$), B_1 (F_1 female × P_1 male), and B_2 (F_1 female × P_2 male). Vertical lines are standard errors of the mean.

df = 1, 97; $P = 0.0005$) and development time ($F = 33.97$; df = 1, 61; $P < 0.0001$). These linear responses accounted for 42 and 62% of the treatment variation detected, respectively. A quadratic trend accounted for only 1.2 and 0.2% of the variation detected for fecundity and development time, respectively ($P > 0.20$ for both variables).

Differences in mean fecundity ($F = 14.04$; df = 2, 56; $P < 0.0001$) and development time ($F = 5.00$; df = 2, 26; $P = 0.015$) among the C, P_1, and P_2 were detected (Fig. 3). The C mean did not differ from the P_1 mean in fecundity and development time ($P \geq 0.20$ for both response variables), despite possible differences in genetic background. However, when conditions of the C and P_2 differed only in fenvalerate selection pressure, fecundity of C was greater than that of P_2 ($F = 12.10$; df = 1, 56; $P < 0.001$) and development time of C was less than that of P_2 ($F = 5.13$; df = 1, 26; $P = 0.032$).

Discussion

When tolerance data of the B_1, B_2, and F_2 were fit to monogenic and polygenic expectations from Georghiou's (1969) model and probit analysis, discrimination between monogenic and polygenic inheritance of the resistant trait was not possible. The inability to discern mode of inheritance with these methods was the result of the overlap of tolerance distributions of the parental generations and the F_1 (Tsukamoto [1963] noted this possibility), even though there was a 115-fold difference in suscep-

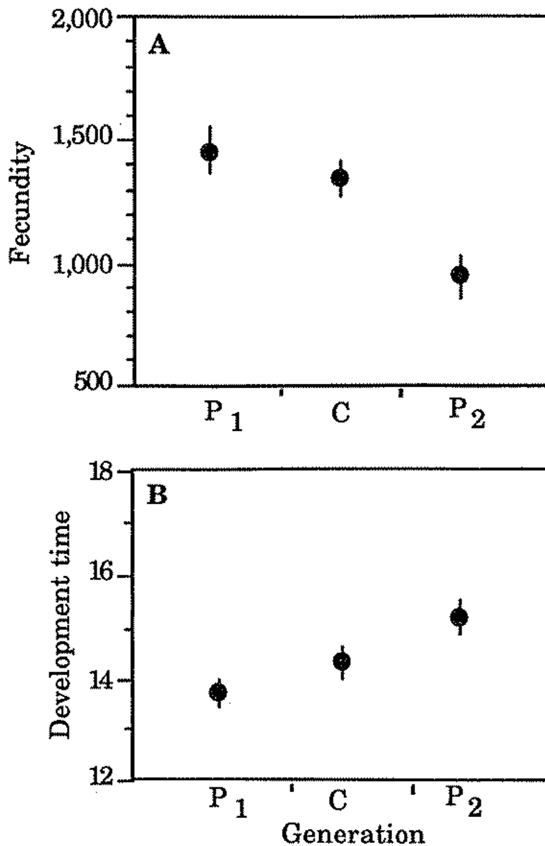

Fig. 3. (A) Differences in fecundity (number of eggs laid) among generations; mean square error is 626,461,403. (B) Differences in development time (days from oviposition to pupation) among generations (data transformed by [logarithm {days + 1}] for analysis); mean square error is 0.00000073. Generations are the P_1 (susceptible), C (derived from the resistant generation but not selected with fenvalerate in the laboratory), and P_2 (resistant). Vertical lines are standard errors of the mean.

tibility between the P_1 and P_2. Using generation means analysis, the best model to explain the variation in mean mortality among generations was a five-parameter model that included epistasis. Therefore, a polygenic model of inheritance to explain the variation of fenvalerate resistance among the generations seemed most plausible. Interaction of loci that govern one or more resistance mechanisms may be responsible for the interactions detected. Esterases have been implicated as a mechanism of resistance to fenvalerate in a Mexican strain (M. J. B., unpublished data) and to deltamethrin (also an α-cyano 3-phenoxybenzyl ester) in a Guatemalan strain (Delorme et al. 1988). In both strains, other mechanisms contributing to resistance were suspected.

An additive cost of fitness (as indicated by the significant linear trend across the ordered generation means) was associated with the resistant trait, but a significant portion of the treatment variation remained unexplained by linear or quadratic trends. Therefore, pleiotropic effects of resistance may be acting on these fitness traits in a complex manner. Other unknown (nonresistant) factors had little effect on fitness differences among these generations, as indicated by comparison of the C with the P_1 and P_2 means.

Resistance in Wild Populations. Whitten & McKensie (1981) stated that resistance is expected to be controlled by one or few genes when the selection pressure is a single insecticide applied within agricultural conditions. Before introduction into the laboratory, ancestors of the parent resistant generation were exposed to fenvalerate and permethrin (pyrethroids), methomyl (carbamate), and methamidophos (organophosphate) (Brewer et al. 1990). Subsequently, the population was selected with fenvalerate in the laboratory. Possibly as a consequence of a mixed insecticide selection regime in the field, resistance to fenvalerate was better described by a polygenic model that included epistasis. Resistance to fenvalerate seen in our study may differ from that resulting from a single insecticide selection regime. Alternatively, fenvalerate resistance in all cases may be polygenic. We note that selection regimes of multiple insecticides are common in the crops where *S. exigua* is found (Trumble 1990).

Results from our study suggest that spatial and temporal variation in resistance seen in wild populations may be partially explained by our observed fitness deficit associated with the resistant trait. Substantial variation in resistance has been reported in wild populations (Brewer et al. 1990, Brewer & Trumble 1991). Using an adult susceptibility test, a survey of 29 populations yielded LC_{50} values that were normally distributed (mean, 647 μg/g; SD, 369). A 9.3-fold difference in susceptibility was detected between the extreme LC_{50} values. Because *S. exigua* is highly mobile and polyphagous, movement of adults between cultivated hosts where insecticides are applied and wild hosts where insecticides are rarely applied may result in continually fluctuating fitness effects and wide variation in resistance. If fenvalerate resistance is polygenic, resistance is less likely to become fixed because evolution of resistance is more gradual in polygenic systems than in monogenic systems (Plapp et al. 1979).

We can attempt to modify the resistance pattern by using factors under man's control, such as intermittent use of fenvalerate on cultivated hosts. Resistance would tend to decrease because of the resistance fitness deficit, although the reduction would be gradual because of the complex mode of inheritance. Managed intermittent use may be partly responsible for continued control of *S. exigua* in many locations in California and Baja California Norte (Mexico), in contrast to sporadic control in Sinaloa (Mexico) where application of pyrethroids has been much greater (Brewer et al. 1990). Managed use may contribute to the sub-

stantial variation in fenvalerate resistance seen in wild populations.

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