Quantification of Larval Resistance to Cypermethrin in Tobacco Budworm (Lepidoptera: Noctuidae) and the Effects of Larval Weight

MICHAEL J. FIRKO[1] AND JANE LESLIE HAYES[2]

Southern Field Crop Insect Management Laboratory, USDA–ARS, Stoneville, Mississippi 38776

J. Econ. Entomol. 83(4): 1222–1228 (1990)

ABSTRACT We examined relationships between larval weight and degree of resistance to cypermethrin in tobacco budworm, *Heliothis virescens* (F.). Laboratory-reared larvae (9.0–175.4 mg) were treated with either 0.1 or 1.0 μg cypermethrin in acetone. Degree of debilitation of each larva was assessed at intervals from 0.5 h to 5 d after treatment; cumulative scores incorporating degree of debilitation and survival time were used as a measure of tolerance (i.e., a continuous measure of resistance). At larval weights <100 mg, relationships between tolerance and weight were linear for both doses. At the rate of 0.1 μg per larva, tolerance reached an asymptote near 100 mg. Proportion of tolerance variation explained by weight decreased with size range, but we obtained significant relationships over weight ranges frequently used in *Heliothis* studies. Weight and dose interacted; slopes of tolerance–weight regressions were steeper at the lower dose (large larvae were disproportionately more tolerant than small larvae). These data indicate that the expression of tolerance and tolerance changes within populations are mediated by even small-scale weight variation, that relationships between tolerance and weight depend on dose, and that it may be possible to refine resistance predictions by accounting for weight variation in treated populations.

KEY WORDS Insecta, *Heliothis virescens*, insecticide resistance, continuous measure

VARIOUS MODELS have been proposed to predict change in populations following insecticide application (Plapp et al. 1979, Taylor 1983, Georghiou & Taylor 1986, Uyenoyama 1986, Via 1986, Roush & McKenzie 1987). Resistance to any particular insecticide depends not only on resistance alleles that an individual possesses but also on a variety of nonheritable genetic and environmental factors that affect the expression of resistance (Taylor 1983). Thus, the accuracy of models designed to predict genetic change depends not only on how accurately genetic factors have been defined but also on the extent to which epigenetic factors affecting genetic expression of resistance are considered.

Because expression of resistance is affected by multiple genetic and epigenetic factors, individuals within populations do not fall into discrete categories. Instead they show continuous tolerance variation (Finney 1971). The term *tolerance* is sometimes associated with low-level resistance, but we use *tolerance* in its original sense to refer to a continuous measure (Finney 1971) of less-than-complete resistance (Georghiou 1972). For convenience, resistance to insecticides has been typically treated as a categorical trait (i.e., susceptible versus resistant), but continuous measures exist (Finney 1971, Cohan & Graf 1985, Holloway 1986, Tabashnik & Cushing 1989). With a continuous measure of resistance (i.e., tolerance), relationships between tolerance and other continuous traits (e.g., weight, fecundity, age) can be analyzed directly with parametric statistical procedures (e.g., regression, analysis of variance).

It is commonly accepted that an individual's weight affects expression of tolerance. For tobacco budworm, *Heliothis virescens* (F.), Roush & Wolfenbarger (1985) reported that small weight differences affect percentage of larval mortality after treatment with methomyl. Mullins & Pieters (1982) reported significantly different LD_{50}'s (methyl parathion and permethrin) among discrete weight classes. However, the functional relationship between tolerance and weight is unknown for *H. virescens* and most insect–insecticide systems. The relationship between tolerance and weight is pertinent because weight variation confounds genetic analyses and predictions of genetic change that are based solely on genotype; small individuals possessing genotypes for superior tolerance are more easily killed than expected, and large individuals with genotypes for inferior tolerance may survive because of their size alone.

Weight effects are partially controlled by testing larvae within particular weight ranges (i.e., 10 mg is a typical range for *Heliothis* studies). However, when larvae weigh 18 ± 5 mg (Luttrell et al. 1987),

This paper reports the results of research only. Mention of a proprietary product or pesticide does not constitute an endorsement or a recommendation for its use by USDA nor does it imply registration under FIFRA as amended.

[1] Current address: USDA-ARS-SABL, Weslaco, Tex. 78596.

[2] Current address: USDA-FS, SFES, Pineville, La. 71360.

the largest larva is 77% heavier than the smallest. Although weight ranges of this magnitude may be typical of variation in field populations, laboratory studies that examine particular factors affecting expression of resistance (e.g., genetic factors) are most accurate when factors such as larval weight are either tightly controlled or precisely considered. Therefore, laboratory-derived relationships between tolerance and weight can be coupled with data on size variation in wild populations to improve the resolution of resistance predictions. Toward this end, we quantified larval tolerance to cypermethrin in laboratory-reared *H. virescens* and examined relationships between tolerance and larval weight.

Materials and Methods

We collected *H. virescens* eggs during September 1987 from a 950-acre (≈380 ha) cotton field (three cotton varieties) immediately southeast of Leland, Washington County, Mississippi. Eggs were randomly collected from 5 plots (8 ha) within the field. Larvae were reared in plastic cups (22.5 ml) containing about 15 ml of artificial diet (King & Hartley 1985) containing a mold inhibitor (Powell & Hartley 1987). Of the field-collected eggs, 219 survived to the pupal stage and founded the laboratory colony. Initially, pupae emerged and mated in 4-liter cardboard cartons (25 males and 25 females in each at 25 ± 2°C, 60 ± 10% RH, and a photoperiod of 14:10 [L:D]). Rearing techniques, which were designed specifically to maintain genetic diversity in the colony, resulted in overlap among generations. Multiple breeding chambers were used at all times. Eggs were collected from each container 3–4 times each week until all adults died, and larvae from each collection were included among the breeding population. All individuals surviving to the adult stage (regardless of development time) were provided with potential mates and given the opportunity to contribute to subsequent generations. Colony size was increased over 3–5 generations with approximate cohort sizes of 200, 543, 1,185, and 1,699. Beginning in April 1988, virgin adults were collected daily, their sexes were determined, and they were mated as single pairs in 0.5-liter cardboard cartons to produce the experimental cohort. Relatedness between parents was minimized by pairing males and females from different subcolonies. Not all pairings produced larvae, and not all larvae were used; 158 females and 54 males produced the 416 larvae used in this experiment.

Each larva was weighed to the nearest 0.1 mg no more than 1 h before topical treatment (1.0 μl) with either 0.1 or 1.0 μg cypermethrin (technical-grade; FMC Corporation, Princeton, N.J.) in acetone (analytical grade). Larvae were treated in their original diet cups where they remained until they died or pupated. Large larvae were not available for 1.0-μg treatments. Although we did not discriminate against larvae in specific weight classes, larvae showing evidence of an impending or recent molt were not used.

Table 1. Scoring criteria

Score	Ability to right itself	Activity and capabilities
0	No	None
1	No	Minor activity after persistent probing
2	No	Minor activity, immediate response
3	No	Slight independent activity, responsive
4	No	Active, slow writhing, no control
5	No	Active, vigorous writhing, no attempts to right itself
6	No	Active, attempts to right itself
7	Yes	Active, rights itself with difficulty
8	Yes	Active, rights itself easily, but adverse effects
9	Yes	Active, rights itself easily, minor effects only
10	Yes	Active, no visible effects

Each individual larva was assigned a score from 0 to 10 at each observation (see text).

Beginning 0.5 h after treatment, we observed each larva when it was agitated gently with a blunt probe; upright larvae were rolled onto their backs. Responsiveness, activity level, type of activity, and ability of the larva to right itself were the criteria for scoring tolerance as a continuous trait. Eleven levels of adverse response (0–10) were distinguished (Table 1). In other studies, individuals that were scored from 0 to 6 on our scale would have been considered dead (Roush & Wolfenbarger 1985, Luttrell et al. 1987, Hoy et al. 1988). We observed and scored each individual at 0.5, 1, 2, 4, 8, 24, 48, 72, 96, and 120 h after treatment. Data from consecutive observations were summed to provide variables for analysis that represented cumulative scores through times (T) 24, 48, 72, 96, and 120 h (variables T24h, T48h, T72h, T96h, and T120h). Individual observations were not independent (e.g., once an individual died all subsequent scores were 0), but this continuous measure of tolerance incorporated degree of debilitation at numerous intervals and total survival time. SAS (SAS Institute 1985) was used for analysis of variance and regression procedures.

Results

Larval tolerance to cypermethrin in *H. virescens* was expressed as a continuous trait at both doses (Fig. 1 and 2). The shapes of the frequency histograms of tolerance phenotypes were similar to those described by Finney (1971) for tolerance phenotypes. Histogram shape depended partly on the underlying distribution of larval weights that was approximately uniform. Many larvae (9.0–175.4 mg) treated with 0.1 μg cypermethrin survived. Some individuals that could have been classified as dead after 24 h subsequently recovered, pupated, emerged as adults, and reproduced. Some individ-

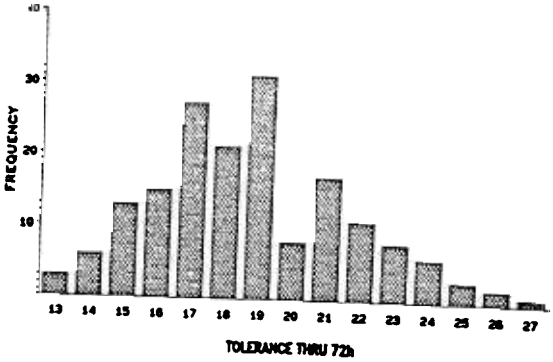

Fig. 1. Frequency histogram of cumulative tolerance scores of *H. virescens* larvae, dose = 1.0 μg cypermethrin per larva. Larvae weighed 9.4–36.1 mg. Variable, T72h (sum of tolerance scores through 72 h, see text); $n = 173$.

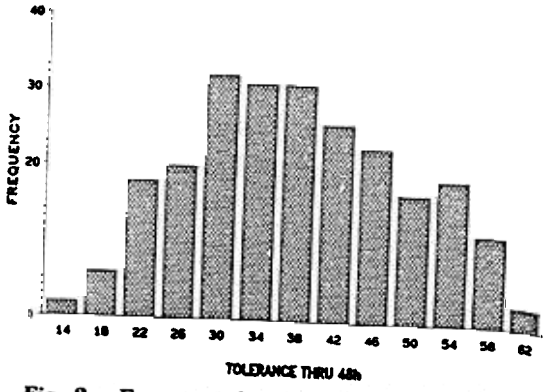

Fig. 2. Frequency histogram of cumulative tolerance scores of *H. virescens* larvae, dose = 0.1 μg cypermethrin per larva. Larvae weighed 9.0–175.4 mg. Variable, T48h (sum of tolerance scores through 48 h, see text); $n = 241$.

uals probably survived because of their size alone. At larval weights <100 mg, the relationship between tolerance and weight was linear, with weight variation accounting for 55% of observed tolerance variation (Fig. 3). Tolerance values reached an asymptote near 100-mg larval weight. The 100-mg cutoff is probably dose-specific; based on our results, we expect the asymptote to occur at heavier weights for higher doses. Tolerance scores of some large larvae approached the maximum possible value. Only 5 of 31 larvae >100 mg died before reaching pupation (86% survival).

Most larvae (9.4–36.1 mg) treated with 1.0 μg cypermethrin were totally debilitated within 24 h, none survived to pupation, and none showed evidence of weight gain following treatment. The weight range of these larvae is greater than that usually used for *Heliothis* studies, but the results can be put into familiar perspective. No larvae scored higher than 4 at the 48-h observation, indicating that 1.0 μg per larva corresponds with at least a 48-h LD_{99} for this population. Table 2 shows frequency distributions of tolerance scores at particular observation times (24-, 48-, and 72-h observations, noncumulative). Tables 1 and 2 can be used to generate LD_x values for a variety of fatal symptoms.

Among larvae treated with 1.0 μg cypermethrin, weight variation accounted for 11% of the observed tolerance variation (Fig. 4). Less tolerance variation was explained by weight variation at 1.0 μg relative to 0.1 μg (Fig. 3) largely because a smaller weight range was examined (26.7 versus 91.0 mg). Genetic variation among individuals explained much of the observed tolerance variation and will be reported separately (M.J.F. & J.L.H., unpublished data); consequently, the amount of variation explained by weight depended on the size of weight range examined. Over very narrow weight ranges, weight variation explained only a small portion of observed tolerance variation and other factors predominated. However, even over a 10-mg weight range typically used for pyrethroid toxicity studies in *Heliothis*, weight variation was a significant factor and explained 9% ($P < 0.005$) of observed tolerance variation among larvae treated with 1.0 μg cypermethrin (Fig. 5). Over wider weight ranges, weight accounted for a larger portion of observed tolerance variation (Fig. 3 and 4).

To examine how dose affected relationships between tolerance and weight, tolerance scores of individuals treated with 1.0 μg (9.4–36.1 mg, $n = 173$) were compared with those of larvae treated with 0.1 μg (9.0–36.3 mg, $n = 69$; Fig. 6). Regression lines for T24h differed in height because larvae were more tolerant to 0.1 μg. The interaction between dose and weight was significant (Table 3), demonstrating that the effect of weight on tolerance depended on dose. Over identical weight ranges, large larvae were relatively more tolerant than small larvae at 0.1 μg than they were at 1.0 μg. Differences in slope were greater than T24h for T48h and T72h (data not shown); however, neither interaction was significant. The lack of significance at later time periods may indicate that size advantages disappear over time, or it may indicate overall greater variability among individuals 48 or 72 h after treatment (Table 2) as individuals either died or continued to accumulate high scores.

Table 2. Frequency distribution of tolerance scores for *H. virescens* larvae treated with cypermethrin

Score	No. larvae per observation period		
	24 h	48 h	72 h
0			
1			
2			
3			
4			
5			
6			

Scores are for particular observation periods, not cumulative scores. Dose = 1.0 μg per larva, $n = 173$. Larvae weighed 9.4–36.1 mg. See Table 1 for description of scores.

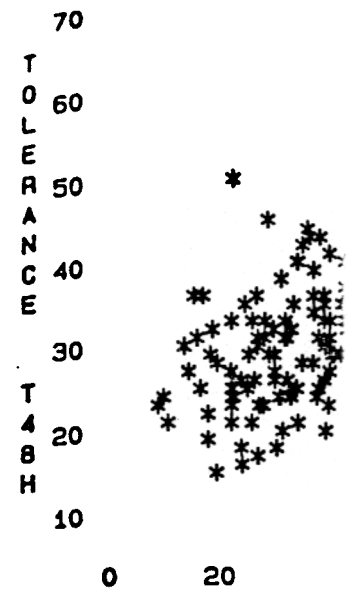

Fig. 3. Plot of tolerance versu larvae weighing 9.0–175.4 mg, n weight was linear. Least-squares

Discussion

With topical application of that individuals in particular t ceive and the actual amount o ing detoxification vary slightly

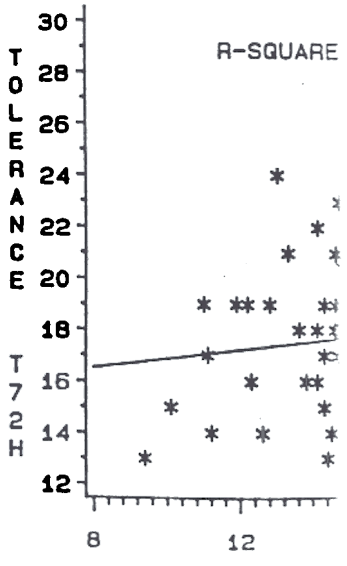

Fig. 4. Plot of tolerance ver larvae weighing 9.4–36.1 mg, n

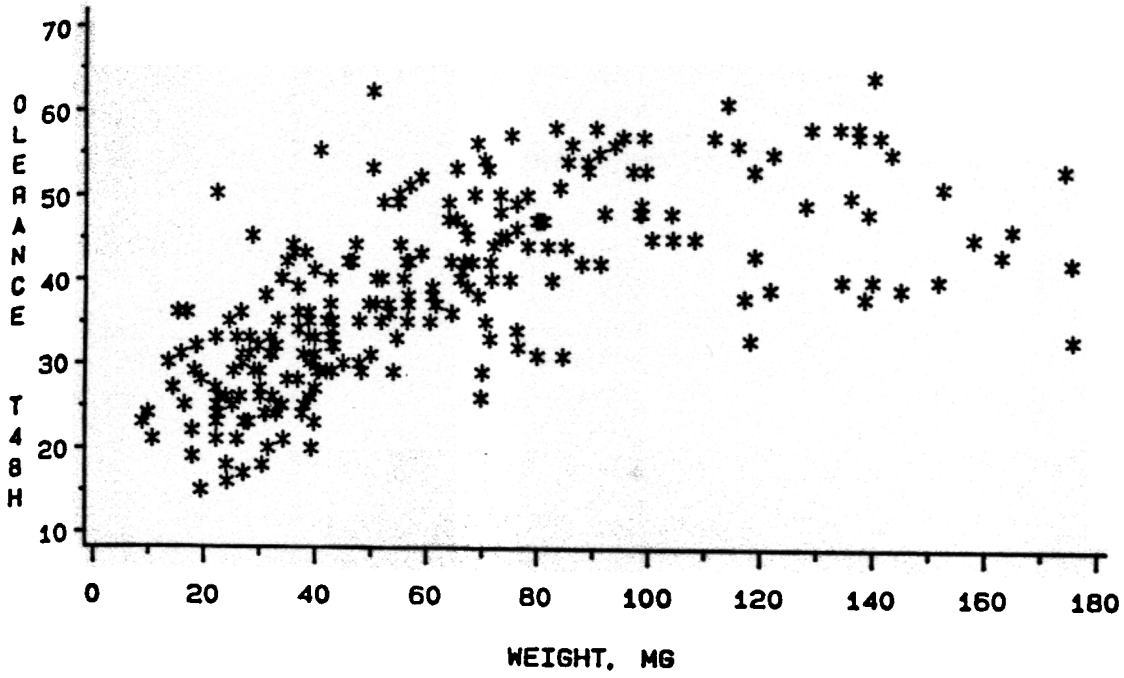

Fig. 3. Plot of tolerance versus weight for *H. virescens* larvae treated with 0.1 μg cypermethrin per larva for larvae weighing 9.0–175.4 mg, $n = 241$. For larvae <100 mg ($n = 205$) the relationship between tolerance and weight was linear. Least-squares regression equation, tolerance = 18.03 + 0.25(weight), $r^2 = 55\%$; $P < 0.0001$.

Discussion

With topical application of insecticides, doses that individuals in particular treatment groups receive and the actual amount of insecticide requiring detoxification vary slightly. Each individual in our tests was treated with as precise a dose as possible with microapplication techniques. Resistance to insecticides has a complex biological and genetic basis (Georghiou & Taylor 1986). Absorption rates, transport through cell membranes, and various

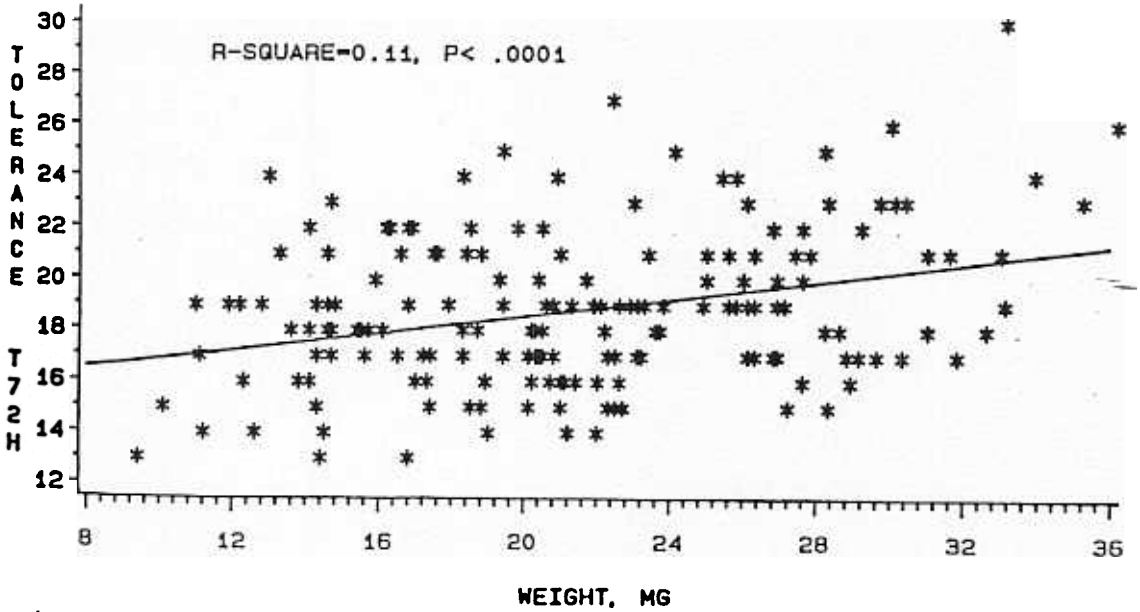

Fig. 4. Plot of tolerance versus weight for *H. virescens* larvae treated with 1.0 μg cypermethrin per larva for larvae weighing 9.4–36.1 mg, $n = 173$. Least-squares regression equation, tolerance = 15.16 + 0.17(weight).

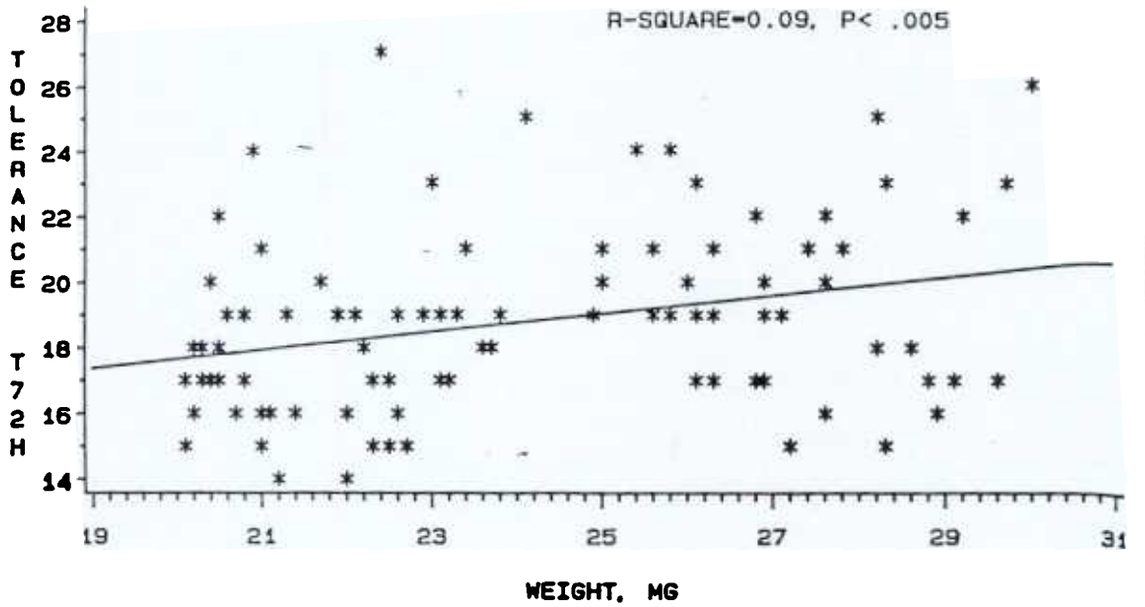

Fig. 5. Plot of tolerance versus weight for *H. virescens* larvae treated with 1.0 μg cypermethrin per larva for larvae weighing 25 ± 5 mg, $n = 91$. Least-squares regression equation, tolerance = 12.06 + 0.28(weight).

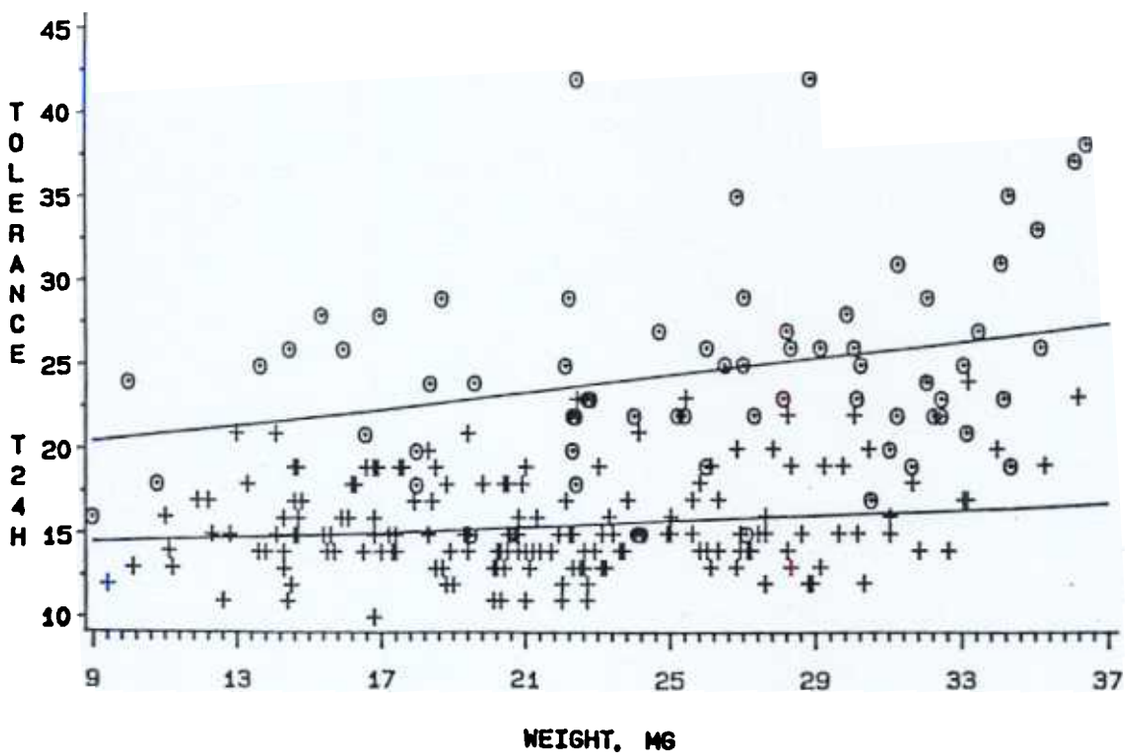

Fig. 6. Plot of tolerance versus weight for *H. virescens* larvae showing interaction between dose and weight. Slopes differ by a factor of three and are significantly different (ANOVA, $P < 0.05$, see Table 3). Circles and upper regression line, larvae treated with 0.1 μg cypermethrin per larva; larval weights 9.0–36.3 mg, $n = 69$, tolerance = 18.22 + 0.25(weight), $r^2 = 0.08$, $P < 0.02$. Pluses and lower regression line, larvae treated with 1.0 μg cypermethrin per larva; larval weights 9.4–36.1 mg, $n = 173$, tolerance = 13.8 + 0.08(weight), $r^2 = 0.03$, $P < 0.03$.

methods of detoxification
mechanisms that affect an
Despite these factors, relati
ance and larval weight were
quantified in our study. Smal
significantly affected expres

Previous reports of suscep
in certain size classes of *H.*
based on one observation per
ters 1982, Roush & Wolfen
peated observations of the s
cated that they responded d
cypermethrin. Some larvae t
ter 24 h later recovered cor
mortality based on a single c
duce bias against larvae in pa
because larvae in different
differently over time. By de
a molt (see Mullins & Pieters
observing each larva, we d
mortality or tolerance diffe
classes beyond reported lir
tween tolerance and weight.

Tolerance variation amor
plained by weight variation n
any number of genetically l
anisms. Because insect growt
particular stages are genetic
(Zirkle & Riddle 1983; Via 1
seau 1987), genetically base
affects tolerance variation. I
variation in field populations
asynchronous hatch, food av
Although weight variation
fecting expression of toleran
relationships between tolera
mediate expression of tolera
used to improve the accurac
to predict the direction and e
in populations following in
For example, linear relatio
ance and weight can be inc
gene (Taylor 1983), multi-ge
or quantitative genetic (Via
unpublished data) models.

Expression of tolerance
weight to an even greater
cations than in laboratory st
plications of insecticides, th
ceives depends partly on a
length (its surface area), w
cubed function of length (S
Small larvae receive a larger
gram of body weight. Becaus
ical that an individual can d
on the extent of its metabol
lationship between tolerance
be linear in field applicatio
have an even greater adva
small larvae than that predic
Techniques that apply insec
body size (e.g., spray table a

methods of detoxification are only some of the mechanisms that affect an individual's tolerance. Despite these factors, relationships between tolerance and larval weight were readily observed and quantified in our study. Small differences in weight significantly affected expression of tolerance.

Previous reports of susceptibility increases within certain size classes of *H. virescens* larvae were based on one observation per larva (Mullins & Pieters 1982, Roush & Wolfenbarger 1985). Our repeated observations of the same individuals indicated that they responded differently over time to cypermethrin. Some larvae that appeared dead after 24 h later recovered completely. Estimates of mortality based on a single observation may introduce bias against larvae in particular weight classes because larvae in different weight classes respond differently over time. By deleting larvae nearing a molt (see Mullins & Pieters 1982) and repeatedly observing each larva, we detected no consistent mortality or tolerance differences among weight classes beyond reported linear relationships between tolerance and weight.

Tolerance variation among individuals not explained by weight variation may have resulted from any number of genetically based tolerance mechanisms. Because insect growth rates and weights at particular stages are genetically determined traits (Zirkle & Riddle 1983; Via 1984a,b; Roff & Mousseau 1987), genetically based variation in weight affects tolerance variation. However, most weight variation in field populations probably results from asynchronous hatch, food availability, or nutrition. Although weight variation is only one factor affecting expression of tolerance, laboratory-derived relationships between tolerance and factors that mediate expression of tolerance in the field can be used to improve the accuracy of models designed to predict the direction and extent of genetic change in populations following insecticide applications. For example, linear relationships between tolerance and weight can be incorporated into single-gene (Taylor 1983), multi-gene (Plapp et al. 1979), or quantitative genetic (Via 1986; M.J.F. & J.L.H., unpublished data) models.

Expression of tolerance may be mediated by weight to an even greater degree in field applications than in laboratory studies. With aerial applications of insecticides, the dose that a larva receives depends partly on a squared function of length (its surface area), whereas its weight is a cubed function of length (Schmidt-Nielsen 1972). Small larvae receive a larger dose of insecticide per gram of body weight. Because the amount of chemical that an individual can detoxify depends partly on the extent of its metabolic capabilities, the relationship between tolerance and weight may not be linear in field applications. Large larvae may have an even greater advantage compared with small larvae than that predicted by a linear model. Techniques that apply insecticide in proportion to body size (e.g., spray table application techniques) may provide relationships between tolerance and weight that more accurately reflect field patterns.

Table 3. Results of analysis of variance testing for interaction between dose of cypermethrin and weight of *H. virescens* larvae

Effect	df	MS	F ratio
Dose			
Weight			
Dose × weight			
Error			

Analyzed variable: cumulative tolerance score through 24 h (T24h). Larvae treated with 0.1 μg per larva weighed 9.0–36.3 mg, $n = 69$; larvae treated with 1.0 μg per larva weighed 9.4–36.1 mg, $n = 173$. Mean squares (MS) are based on Type III sums of squares. *, Significant at $P = 0.05$; ***, significant at $P = 0.001$.

If large larvae are more tolerant than small larvae because they have more resources to fight the insecticide, the significant interaction between dose and weight may result for purely physical reasons. For example, at 1.0 μg per larva, 20 g and 100 g larvae have 2 g and 10 g of body weight, respectively, for each 0.1 μg of insecticide, a difference of 8 g. At 0.1 μg per larva, 20 g and 100 g larvae have 20 g and 100 g of body weight, respectively, for each 0.1 μg of insecticide, a difference of 80 g. At lower doses, large larvae have relatively more resources to fight a given amount of insecticide than they do at high doses.

In *H. virescens*, as in other pest species, resistance has developed more slowly in the field than in laboratory selection experiments (Brown & Payne 1988). Any epigenetic factor affecting expression of tolerance can potentially facilitate or delay development of resistance in the field. Most epigenetic factors probably delay resistance development in the field because of increased environmental variability in field populations relative to laboratory strains. Delayed resistance in the field can result from effects mediated by weight if enough larvae survive field application because of their large size alone. Differences between laboratory and field in the length of time required for the development of resistance may be reduced by using empirically derived relationships to correct for the relatively high variability in weight and other epigenetic factors in field populations.

Acknowledgment

We thank Randy Bell for the loan of his microapplication equipment and P. O'Brien for helpful discussions on *Heliothis* toxicity tests. We thank G. Snodgrass, W. Kitten, D. Wolfenbarger, R. Luttrell, J. Schneider, and J. Mallet for their helpful comments on the manuscript.

References Cited

Brown, T. M. & G. T. Payne. 1988. Experimental selection for insecticide resistance. J. Econ. Entomol. 81: 49–56.

Cohan, F. M. & J. D. Graf. 1985. Latitudinal cline in *Drosophila melanogaster* for knockdown resistance to ethanol fumes and for rates of response to selection for further resistance. Evolution 39: 278–293.

Finney, D. J. 1971. Probit analysis, 3rd ed. Cambridge, London.

Georghiou, G. P. 1972. The evolution of resistance to pesticides. Annu. Rev. Ecol. Syst. 3: 133–168.

Georghiou, G. P. & C. E. Taylor. 1986. Factors influencing the evolution of resistance, pp. 157–169. *In* Pesticide resistance: strategies and tactics for management. National Academy, Washington, D.C.

Holloway, G. J. 1986. A theoretical examination of the classical theory of inheritance of insecticide resistance and the genetics of time to knockdown and dry body weight in *Sitophilus oryzae* (L.) (Coleoptera: Curculionidae). Bull. Entomol. Res. 76: 661–670.

Hoy, M. A., J. Conley & W. Robinson. 1988. Cyhexatin and Fenbutatinoxide resistance in Pacific spider mite (Acari: Tetranychidae): stability and mode of inheritance. J. Econ. Entomol. 81: 57–64.

King, E. G. & G. G. Hartley. 1985. *Heliothis virescens*, pp. 323–328. *In* P. Singh & R. F. Moore [eds.], Handbook of insect rearing, vol. 2. Elsevier, New York.

Luttrell, R. G., R. T. Roush, A. Ali, J. S. Mink, M. R. Reid & G. L. Snodgrass. 1987. Pyrethroid resistance in field populations of *Heliothis virescens* (Lepidoptera: Noctuidae) in Mississippi in 1986. J. Econ. Entomol. 80: 985–989.

Mullins, W. & E. P. Pieters. 1982. Weight versus toxicity: a need for revision of the standard method of testing for resistance of the tobacco budworm to insecticides. J. Econ. Entomol. 75: 40–42.

Plapp, F. W., Jr., C. R. Browning & P. J. H. Sharpe. 1979. Analysis and rate of development of insecticide resistance based on simulation of a genetic model. Environ. Entomol. 8: 494–500.

Powell, J. E. & G. G. Hartley. 1987. Rearing *Microplitis croceipes* (Hymenoptera: Braconidae) and other parasitoids of Noctuidae with multicellular host-rearing trays. J. Econ. Entomol. 80: 968–971.

Roff, D. A. & T. A. Mousseau. 1987. Quantitative genetics and fitness: lessons from *Drosophila*. Heredity 58: 103–118.

Roush, R. T. & J. A. McKenzie. 1987. Ecological genetics of insecticide and acaricide resistance. Annu. Rev. Entomol. 32: 362–380.

Roush, R. T. & D. A. Wolfenbarger. 1985. Inheritance of methomyl resistance in the tobacco budworm (Lepidoptera: Noctuidae). J. Econ. Entomol. 78: 1020–1022.

SAS Institute. 1985. SAS user's guide: statistics. SAS Institute, Cary, N.C.

Schmidt-Nielsen, K. 1972. How animals work. Cambridge University Press, London.

Tabashnik, B. E. & N. L. Cushing. 1989. Quantitative genetic analysis of insecticide resistance: variation in fenvalerate tolerance in a diamondback moth (Lepidoptera: Plutellidae) population. J. Econ. Entomol. 82: 5–10.

Taylor, C. E. 1983. Evolution of resistance to insecticides: the role of mathematical models and computer simulations, pp. 163–173. *In* G. P. Georghiou & T. Saito [eds.], Pest resistance to pesticides. Plenum, New York.

Uyenoyama, M. K. 1986. Pleiotropy and the evolution of genetic systems conferring resistance to pesticides, pp. 207–221. *In* Pesticide resistance: strategies and tactics for management. National Academy, Washington, D.C.

Via, S. 1984a. The quantitative genetics of polyphagy in an insect herbivore. I. Genotype–environment interaction in larval performance on different host plant species. Evolution 38: 881–895.

1984b. The quantitative genetics of polyphagy in an insect herbivore. II. Genetic correlations in larval performance within and among host plants. Evolution 38: 896–905.

1986. Quantitative genetic models and the evolution of pesticide resistance, pp. 222–235. *In* Pesticide resistance: strategies and tactics for management. National Academy, Washington, D.C.

Zirkle, D. F. & R. A. Riddle. 1983. Quantitative genetic response to environmental heterogeneity in *Tribolium confusum*. Evolution 37: 637–639.

Received for publication 19 May 1989; accepted 3 November 1989.

Survey of North C (Coleopt

D. C. HE

ABSTRACT Laborator *Leptinotarsa decemlinea* icals registered for use extensive in potato beetle levels of resistance also resistance was most prom and carbofuran resistance Results also showed signi counties, among fields w

KEY WORDS Insecta,

COLORADO POTATO BEETLE, *Le lineata* (Say), adults and larvae defoliators of commercially gr *Solanum tuberosum* L., in the ea Their control has involved nearl dency on insecticides and has re severe insecticide resistance prob (Forgash 1981, 1985; Gauthier resistance is extensive, the cost sistant beetles on potato is high existence of the commercial pot 1985). Although many areas of have had severe problems wit tance of the Colorado potato beet it was not until the mid-1980s growers in eastern North Caro perience declining control of Co tle populations with pyrethroid insecticides used frequently on

Managing resistance requires levels and distribution of res throughout the potato-growing r of the spatial patterns of variatio is essential to an understanding resistance (Tabashnik et al. 198 results of a 2-yr survey done to terns of variation that exist in valerate, carbofuran, and azin populations of Colorado potato b growing regions of eastern Nort

Materials and Me

Chemicals. Commercial form erate (Pydrin 2.4 emulsifiable

—Use of trade names does not imply e named nor criticism of similar ones not