A MODEL OF INSECTICIDAL CONTROL FAILURE: THE EXAMPLE OF *HELIOTHIS VIRESCENS*[1] ON COTTON

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ABSTRACT

We examine a complex model of insecticidal control failure that simulates population density as well as frequency of a resistance gene. While the model is only semi-realistic, it is representative of a broad class of multi-parameter models that include the actual situation in the field. The model includes over 40 potentially measurable parameters relating to insecticide decay rates, probability that insects in different larval stages will contact an insecticide deposit, age- and genotype-related tolerance, and rates of increase. The purpose of this paper is to show how this diversity of parameters can be simplified to just four parameters important in control failures (selection pressure, s, dominance, h, and rates of increase of susceptible and resistant populations in the presence of insecticides, R_{SS} and R_{RR}). Standard population genetic models (using s and h alone) often describe the behavior of such systems adequately. Dosage-mortality response curves are expected to be much flatter in the field than in laboratory topical bioassays. This means that curves of different genotypes will overlap considerably, so that making resistance effectively recessive in a pest like *Heliothis virescens* will be hard. Many techniques of resistance management advocated in the literature will have little effect, both because of the shape of field dose-mortality curves, and because of tight financial constraints on altering field dosage rates. However, there is one very useful way to slow the evolution of resistance: reduce the number of insecticide applications as much as possible while maintaining farm profits. The problems with this "low input" approach currently seem to be more social than economic.

INTRODUCTION

A number of workers (e.g. Tabashnik 1986, 1989, Sawicki and Denholm 1987) have questioned the relevance of simple genetic models for the evolution of resistance because they ignore many complexities of real systems, particularly population dynamics. In this paper we estimate underlying parameters from a complex "semi-realistic" model, and assess the importance of population dynamics in control failures. We show how the use of a simple genetic model with few parameters can

[1] Lepidoptera: Noctuidae

usually be justified. However, in order to estimate the few important parameters in the simple model, we need to know something about the complexities in the field. Although the type of insect control system examined is general, we focus on a specific example: the tobacco budworm, *Heliothis virescens* (F.), in cotton.

In *H. virescens*, mortality due to insecticides will be equal to the product of (1) the probability of contact of each stage with sites of insecticide deposition on the plant; (2) the fraction of the initial insecticide residue remaining on these sites, and (3) the stage- and genotype-specific mortality expected with a given dose of insecticide, integrated over the whole life cycle. For (1), recent evidence shows that the probability a late instar larva will contact an insecticide is lower than for earlier instars, because late instars usually tunnel into squares or bolls away from insecticidal deposits (Luttrell et al. 1990a,b, Micinski et al. 1990). For (2) it has been found that, while many pyrethroids are relatively persistent, plant growth will reduce residues on the upper parts of the plant where most eggs and larvae are placed (Luttrell et al. 1990a,b, Goodenough et al. 1990). For (3) we know that early instars of even resistant larvae can be killed by insecticides, and conversely, later instars of susceptible larvae are relatively tolerant to insecticides (Wolfenbarger et al. 1984, Luttrell et al. 1990a,b, Micinski et al. 1990, Goodenough et al. 1990); results on the related *Helicoverpa armigera* are similar (Daly et al. 1988).

MATERIALS AND METHODS

One of us (R.G.L.) formulated a "semi-realistic" model of control failure in *Heliothis*, diagrammed in Fig. 1. This model has been used by R.G. Luttrell to explore how different control parameters would affect the speed of control failure in *H. virescens* on cotton when resistance is becoming a problem. The model was run on a spreadsheet, and the output has been used by R.G.L. in presentations to growers and industry representatives. Although some assumptions of the model are unrealistic, the model is a good example of a class of possible realistic models which could incorporate survival rates due to a variety of different factors. The factors are listed below, together with the parameter values used in actual simulations.

It is assumed that when growers first realize there is a *Heliothis* problem, there are larvae in a variety of stages which are then sprayed three times with a pyrethroid at 7 day intervals. For mathematical convenience, each larval instar lasts 3 days. Mortality is assumed to be solely the result of insecticides: however, the "normal" fourfold maximal rate of increase is considerably lower than the fecundity of females because we ignore all those eggs that give rise to larvae that die because of non-insecticidal causes. No density-dependent mortality is assumed; this is approximately realistic because in cotton *H. virescens* is unlikely to approach the carrying capacity of the environment because of insecticidal treatment. Resistance is assumed to be inherited at a single gene. Daily survival is stage- and genotype-specific and also depends on the amount of insecticide residue present (shown at the top of the figure, where dashed line = low persistence, solid line = normal persistence, dotted line = high persistence; low and high persistence are only shown for one 7-day interval) on the infestable plant parts since the last spray, and the stage-specific probability of contact with the insecticide. Any deleterious pleiotropic effects of resistance genes are of course included in these mortalities. The larvae pupate, eclose as adults, and mate to produce another generation of early stages (the adult numbers resulting from this mortality schedule are multiplied by the

maximal rate of increase and then divided up among the instars according to the conditions set). Control was defined to have failed when over 1000 adults are produced. Resistance is said to have evolved when the allele frequency, q, of the resistance allele is greater than 0.5. The parameters important in the model are as follows:

A) Genetic parameters

1) *Initial frequency of resistance genotypes*

Normal initial frequency: SS=0.99 RS=0.009 RR=0.001
Low initial frequency: SS=0.999 RS=0.001 RR=0
High initial frequency: SS=0.9 RS=0.09 RR=0.01

2) *Proportions of offspring genotypes*

Determined by Hardy-Weinberg Law: SS=p^2 RS=$2pq$ RR=q^2, where $q = 1 - p$ is the frequency of the R allele

B) Population dynamic parameters

1) *Total initial numbers of young stages*: 1000

2) *Relative initial proportions of different young stages:*

E=0.2 L1=0.2 L2=0.2 L3=0.2 L4=0.1 L5=0.1

3) *Maximal rate of increase:*

Low=2 Normal=4 High=8

C) Parameters affecting population dynamics and genetics

1) *Probability of contacting insecticides:*

E=0.64 L1=0.64 L2=0.56 L3=0.4 L4=0.24 L5=0.08

2) *Residue levels of insecticides due to persistence and plant growth*

	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7	Day 8	Day 9 ..
Normal:	1.0	0.9	0.8	0.6	0.4	0.1	0	1.0	&c	&c
Low:	1.0	0.6	0.1	0	0	0	0	1.0	&c	&c
High:	1.0	1.0	0.9	0.8	0.6	0.2	0	1.0	&c	&c

3) *Mortality upon contacting insecticides, depending on stage and genotype*

Mortality:	Normal			Low			High		
Genotype:	SS	RS	RR	SS	RS	RR	SS	RS	RR
Stage									
E	0.4	0.4	0.4	0.2	0.2	0.2	0.6	0.4	0.4
L1	1.0	0.95	0.9	0.8	0.75	0.7	1.0	1.0	1.0
L2	0.95	0.8	0.7	0.75	0.6	0.5	1.0	1.0	0.9
L3	0.9	0.5	0.2	0.7	0.3	0	1.0	0.7	0.4
L4	0.7	0.4	0.1	0.5	0.2	0	0.9	0.6	0.3
L5	0.5	0.2	0	0.3	0	0	0.7	0.4	0.2

4) *Immigration and emigration*: set at 0

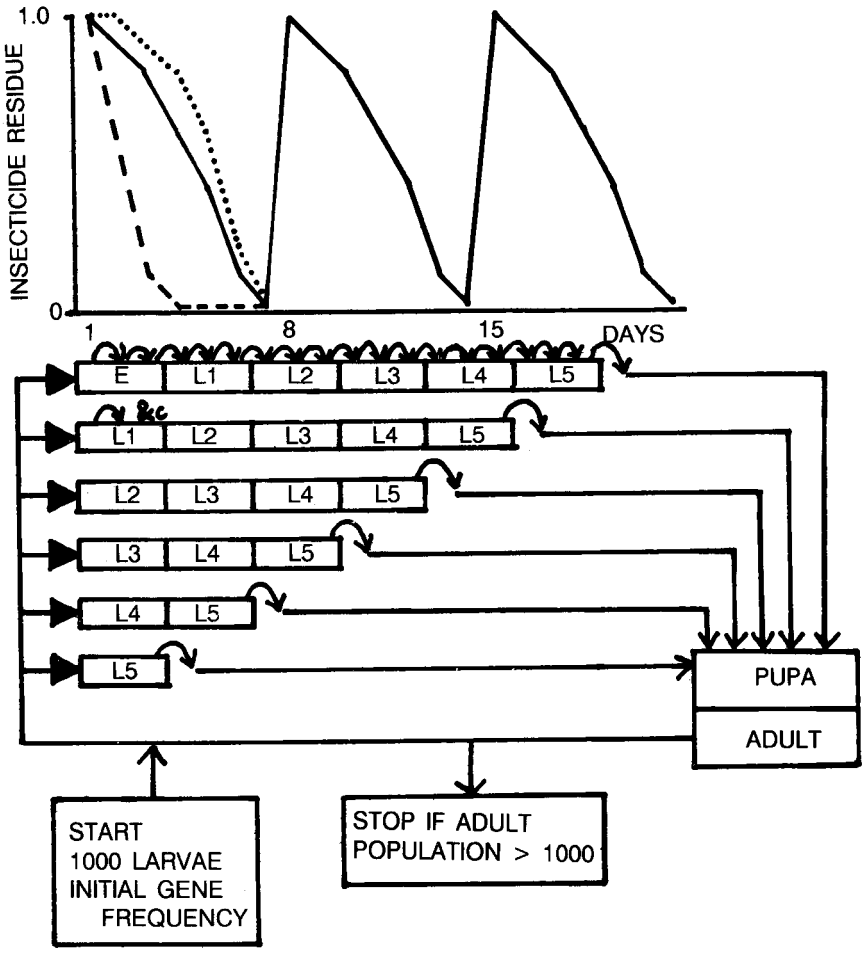

FIG. 1. A "Semi-Realistic" Model of Control Failure

The model is realistic in the sense that it models pesticide decay rates, age- and genotype-specific mortality, and the probability that different life stages contact insecticides. The model is, however, unrealistic in the sense that it does not model overlapping generations very accurately, and that it ignores the destruction of natural enemies of the pests by the insecticide. The latter can be very important in stimulating the evolution of resistance (Comins 1977, Tabashnik and Croft 1982, May and Dobson 1986, Mallet 1989). However, given that pests such as *Heliothis* and their enemies are sprayed with great regularity, the pest can be assumed to be far from its carrying capacity, and will tend to increase exponentially (at least initially) if insecticides are not used. This semi-realism will suffice in this paper because we are concerned chiefly with demonstrating how control failure and evolution of resistance are affected by parameters that are more realistic and complex than those in previous models, and in showing how the underlying coefficients of dominance and selection can be estimated, rather than with providing the last word on realism.

The model of Fig. 1 is complex: even ignoring the parameters that set up the model framework (for example lifespan of juvenile stages, length of time between sprays, &c.) there are still over 40 parameters. The parameters listed above include some values assumed approximately average ("normal") on the basis of field experience and experimental results, and variations on these parameters are used in the simulations. However, because we assume that there is no frequency- or density-dependent selection or density-dependent population regulation, these factors can be combined to give per-generation rates of mortality for each of the three genotypes (SS, RS, and RR). Mating and reproduction will cause interactions between gene frequency and net rates of increase for the genotypes. However, when resistance evolves, it evolves rapidly; most of the time resistance alleles are either rather rare or rather common. Therefore, a convenient marking post, used in this paper, for the evolution of resistance is when the gene frequency of a resistance allele, q, is 0.5. At this value evolution is occurring most rapidly, and q is within a few generations of being relatively low or very high, regardless of dominance.

We shall see that, for most of the time, rates of mortality and of increase affect population growth and the evolution of resistance independently. An understanding of the behavior of the model can then be aided by estimating four parameters: two population dynamic, and two population genetic. These are: the net rate of increase in a population completely fixed for susceptible alleles (R_{SS}), the net rate of increase in a population fixed for resistant alleles (R_{RR}), the dominance of the resistance allele (h), and the selection coefficient against the susceptible allele (s): all parameters are defined given insecticidal treatments. The net rate of increase used here (R) is the per-generation finite growth rate, so that if $R < 1$ the population declines, and if $R > 1$, the population increases. This should be contrasted with the maximal rate of increase, which represents the R that would be observed in the absence of insecticidal mortality. The net rate of increase for a fixed (i.e. all homozygous SS or RR) population is calculated by following a cohort of individuals of a homozygous genotype through a single generation of mortality and reproduction, and dividing the numbers after the generation by the numbers before. Providing there is no heterozygous advantage or disadvantage, the net rate of increase of a polymorphic population will be intermediate between R_{SS} and R_{RR}. The maximal rate of increase (i.e. in the absence of insecticide treatments) is constant across genotypes, so that we need consider only changes in the relative proportions of genotypes within a generation for

the calculation of selection coefficients and dominance. We wish to reduce the complex mortality schedule to the fitnesses (w) of each genotype relative to the RR genotype. We need fitnesses of the form $w_{SS} = 1 - s$, $w_{RS} = 1 - (1-h)s$, $w_{RR} = 1$. Thus we divide the proportion of SS remaining after the larval instar in Fig. 1 by the proportion of RR remaining, and set this equal to $1-s$. The proportion of RS remaining divided by the proportion of RR remaining is similarly equal to $1 - (1-h)s$; these two equations can be solved to give the effective dominance (h) of the resistance allele and the selection coefficient (s) against the susceptible individuals. It will be noticed that the parameter s measures the reduction in R for SS compared with that of RR, and h is a way of comparing R_{RS} with R_{SS}; thus, there are essentially only three parameters that need to be understood, h, s, and R_{RR}.

RESULTS

Results of simulations using approximately "normal" parameter values for *Heliothis* (based on the field experience and experimental information of R.G.L.), together with simulations under possible deviations from normal conditions are shown in Table 1. Table 1 also shows the four essential population dynamic and genetic parameters distilled from the approximately 40 parameters for each case given in Fig. 1. These essential parameters help to explain the behavior of the model.

In the "normal" simulation, the population declines ($R_{SS} < 1$) when resistance is rare, and increases ($R_{RR} > 1$) when resistance is common. Since resistance can evolve under insecticidal pressure, control failure can be said to be caused by evolution of resistance.

Low doses of insecticide might reduce mortality of all young stages, but have relatively little effect on the speed of evolution of resistance. In this case, assuming a rather inflexible spray schedule, a population might build even in the absence of resistance ("low max. mortality" in Table 1). Similarly, a high level of mortality, perhaps caused by high doses of insecticides, could delay control failures, not only because resistance evolves more slowly, but also chiefly because even resistant insects are prone to greater insecticidal mortality which may reduce their net rate of increase.

Low persistence will affect the probability of control failures in a similar way to that of low dose: a reduction of selection may be compensated for by an increase in net reproductive rate. The parameters chosen here cause a rapid population eruption (mainly because $R_{SS} > 1$) long before resistance evolves. The high persistence modeled here causes a slight reduction in the time for evolution of resistance because of slightly higher selection, and a slightly greater time before control failure because of a reduction in the net rate of increase.

As is well known, low initial frequencies of the resistance gene (q) will cause slower evolution of resistance, and higher frequencies will cause more rapid evolution. Here, evolution of resistance again causes control to fail as in the normal case because the relative rates of increase of the genotypes are "normal". As expected, the control failure is delayed when the initial gene frequency is low, and speeded when the initial frequency is high.

Varying the maximal rate of increase from 4 ("normal") can produce dramatic changes in the time to control failure. However, these changes are not due to changes in the time to evolution of resistance because this stays constant at 15 generations: the evolution of resistance depends only on the relative rates of mortality of the three genotypes since the maximal rate of increase is entered as a constant

multiplier across all genotypes and does not affect these relative rates. With a low maximal rate of increase (2), a control failure never occurs, although resistance evolves in 15 generations as in the normal case, because even $R_{RR} < 1$. In contrast, a high maximal rate of increase (8) causes a rapid population explosion, even though resistance remains relatively rare until long after the control failure, because $R_{SS} > 1$.

Table 1. Underlying Population Dynamic and Genetic Parameters and Simulation Results of the "Semi-Realistic" *Heliothis* Model

Simulation Type	Genetic Parameters		Population Dynamic Parameters		Time to $q = 0.5$ (gens.)	Time to Control Failure (gens.)
	h	s	R_{SS}	R_{RR}		
Normal	0.45	0.50	0.85	1.69	15	21
Low max. mortality	0.51	0.50	1.11	2.23	14	10
High max. mortality	0.41	0.43	0.73	1.28	19	47
Low persistence	0.51	0.36	1.41	2.23	21	4
High persistence	0.43	0.52	0.77	1.57	14	24
Low gene frequency	0.45	0.50	0.85	1.69	21	30
High gene frequency	0.45	0.50	0.85	1.69	8	13
Low rate of increase	0.45	0.50	0.43	0.84	15	∞
High rate of increase	0.45	0.50	1.70	3.38	15	4

The model of Fig. 1 was run to produce the results above, using the "normal" parameter values of Fig. 1 except for the parameter changes indicated at the left of this table.

DISCUSSION

Some of the results above could be due to peculiar features of the model or to unrealistic parameter values chosen. In this discussion we will therefore not try to defend this particular semi-realistic model, but will instead concentrate on general conclusions that can be drawn regarding the whole class of realistic models, and hopefully, the field situation itself.

We can broadly categorize pests into three types. The first type has a very high rate of increase relative to the mortality that can reasonably be inflicted using insecticides. These pests will often rapidly return to and exceed pretreatment numbers very soon after insecticide applications. Resistance can evolve rapidly, which will make things worse. Possible examples in cotton are whiteflies, spider mites, and aphids (Bottrell and Adkisson 1977). In citrus, mites and scale insects are known to have the same tendencies (DeBach 1974). Of course, insecticides are known to reduce

the numbers of natural enemies, inflating the pests' net rate of increase as well as causing resistance to evolve. Both factors will increase still further the probability of control failure in such pests. The important point is that species with a very high maximal rate of increase may be hard to control using insecticides. In the Mississippi Delta in 1989 one of us (R.G.L.) observed repeated failures to control cotton aphids, *Aphis gossypii* Glover, using insecticides. The populations were finally brought to very low levels by epizootics of fungal pathogens (principally *Neozygites fresenii*). A similar decline in aphid population density due to fungi, possibly aided by predators, was observed in 1990 (R.B. Head pers. comm.).

Other species may be able to evolve resistance, but because the resistance is relatively ineffective, or the maximal rate of increase is low, the insecticidal mortality is still high enough to prevent outbreaks. Possibly the cotton leafworm, *Alabama argillacea* Hübner, once a major cotton pest in the United States, falls into this category. The boll weevil, *Anthonomus grandis* Boheman, seems to be such a pest with respect to organophosphate insecticides.

A third, intermediate type of pest species includes those that have a net negative rate of increase when they are susceptible, and a net positive rate of increase when resistance becomes common. In these species, the evolution of resistance causes a population explosion. These species are those for which resistance management could be most effective. The tobacco budworm, *H. virescens*, is in this category and is the species on which the model of Fig. 1 was loosely based. The boll weevil, *A. grandis*, was a member of this third category with respect to organochlorine insecticides: the evolution of resistance to these compounds led to control failures.

What can be done for pests like *Heliothis*? Using a one-insecticide strategy, several approaches are commonly suggested. First, we can try to retard the evolution of resistance by making the resistance recessive. It has been suggested that this can be achieved using a high dose (high maximum mortality in Table 1), thereby increasing the number of heterozygotes that are killed (Curtis 1987). Leaving aside for the moment the problem that resistance rarely seems to be caused by a single gene (see below), we should note that this strategy depends strongly on the dosage-mortality curves for the three genotypes. If the curves overlap little, as in a number of classic laboratory studies of dieldrin resistance, an increase of dose can make the R allele effectively recessive (see Curtis 1987, Roush and McKenzie 1987). However, field studies with *Heliothis* (Luttrell et al. 1987, Luttrell et al. 1990a,b, Micinski et al. 1990) have indicated that whatever the dose, there are always some susceptible genotypes that survive, and some resistant genotypes that die. This occurs because of variation of dose in the field, differences in stage-specific mortality upon contact with a dose, differences in stage-specific probability of contact due to differences in insecticide deposition on different parts of the plant, and changes in mortality as the residue declines (see above). Whatever the field dose, the dominance in the field will alter little. In Table 1, h only varies between 0.41 and 0.51 (perfect codominance is 0.5). For similar reasons, the selection pressure in the field is only liable to vary within relatively narrow limits. In Table 1, s varies only between 0.43 and 0.52. Although these numerical results are based on specific parameter values and the particular model given, the general conclusion that selection is relatively invariant, and that the dominance is liable to remain near 0.5 seem likely to be robust for the field situation in *Heliothis* and many other pests. Even though some types of treatment can increase recessiveness of the gene by killing more RS, they will usually also increase selection by killing more SS genotypes; the second effect will partially cancel the first. A

second approach to resistance management, the "low dose strategy" (Georghiou 1983) (see low maximum mortality in Table 1) is relatively ineffective for the same reasons; increased survival of SS genotypes, which reduces selection, is nearly balanced by an increase in survival of RS genotypes which can increase dominance. In any case, both effects are relatively weak because the field dosage-mortality responses are likely to be shallow and to overlap those of other genotypes.

Reducing or increasing persistence can have a similar effect to changing the dose. While low persistence can be a good management ploy for delaying resistance (Taylor and Georghiou 1982), it has the unfortunate effect of reducing mortality which might lead to control failure even before evolution of resistance. In the example of Table 1, low persistence allows a population eruption of susceptibles, and high persistence causes a slight delay in control failure because of its effect on reducing the net rate of increase. Obviously the disadvantages of low persistence might be overcome by increasing numbers of treatments, but this would also have the effect of increasing selection, thereby canceling the advantages of the management technique.

There are two obvious weaknesses in the assumptions of our "semi-realistic" model which should be discussed. First, we ignore all density-dependent population regulation. In Mississippi, *H. virescens* is probably little affected by density-dependence during the cotton growing season because it is maintained at such a low level, but the first generation of *H. virescens* is found on wild hosts, especially *Geranium* spp. This host could have the important effect of resetting the population size to low levels every year. If this happens, we expect that control failures will rarely be caused by population dynamics alone, as in the low persistence and low dose (low maximum mortality) cases of Table 1. Instead, evolution of resistance will be the most important factor, so that standard population genetic models will be sufficient. Similarly, under density dependent regulation the high dose and high persistence strategies are unlikely to have as much effect in prolonging the time to control failure as suggested in Table 1. We therefore do not recommend these strategies. These examples were merely used to show that control failure can be decoupled from the evolution of resistance if the population is poorly regulated.

A second apparent weakness of our approach could be the assumption of a single locus. It is increasingly being realized that insecticide resistance can be due to more than one mechanism (e.g. in *Heliothis*, Dowd et al. 1987, Little et al. 1988). There may not be very many genes, but even in the extreme, a polygenic model would behave very similarly to the model outlined above, if, as we suspect, the variance in susceptibility is used up relatively quickly during evolution. This is strongly suggested by the existence of genes of major effect in genetically well-studied cases of insecticide resistance (Wood 1981). Under strong per-locus selection, it is well-known that standard quantitative genetic models of evolution do not apply: quantitative models assume that the variance remains constant, and that the phenotypic distribution remains normal, which requires weak selection (Falconer 1981, Hartl and Clark 1989). Thus, we expect that the addition of more genes will make little difference to the general outcome, provided that realistic models with strong per-locus selection are employed.

In the cotton belt today, pyrethroids are the most useful compounds for insecticidal control of *Heliothis*, and we may lose the efficacy of these compounds in the next few years. Pests like *Heliothis*, with negative net rates of increase when resistance is rare and positive net rates of increase when resistance is common, seem to hold the greatest promise for resistance management. But even such pests will

be refractory candidates for standard techniques of resistance management such as low doses, high doses, or low insecticidal persistence. These techniques will have relatively little effect on dominance and selective coefficients in the field because dosage-response curves are likely to be much flatter in the field than in the laboratory, so that curves from different genotypes overlap much more. In addition, dosage can rarely be increased much in the field because of cost. Therefore, the options are extremely limited.

There is one very simple management technique which will always reduce the selection for resistance, and this is to use insecticides less frequently. For example, in the model outlined here, halving the number of generations which are sprayed with insecticides will approximately double the time taken to evolve resistance; treating only once every three generations will increase the time to resistance threefold. Can this be achieved without compromising control of a pest like *Heliothis*? We believe so; with good but not overly expensive scouting, and using our current, admittedly imperfect knowledge, the number of insecticidal applications for *Heliothis* can probably be at least halved on many Mississippi cotton farms (Luttrell and Reed 1986, Varner, D., Head, R.B., Pearson, W., and Owen, D. Cotton insect control demonstration, Tallahatchie County, Mississippi, 1989. Unpublished typescript report, Mississippi Cooperative Extension Service, Mississippi State University). If these techniques were adopted, they would result in a substantial increase in average profits for farmers and would rarely cause any economic loss. In addition, control exerted by natural enemies would be improved because of the reduced insecticide pressure, further enhancing the strategy. Let us remember that *H. virescens* was not a pest before the advent of DDT! Of course, a 50% reduction in sales of chemicals for *Heliothis* control would considerably cut into short-term profits of insecticide companies and retailers. This probably explains why insecticides continue to be marketed aggressively while at the same time the commercial sector ostentatiously expresses great concern about resistance. Some of the blame should also be shouldered by growers, consultants, researchers, and extension. Contemporary cotton production is a capital-intensive enterprise (Phillips et al. 1989), and liberal "insurance" use of insecticides often appears to be a logical management option for growers and consultants. Researchers and extension can also be faulted for continuing to use ineffective marketing techniques for ecologically sound principles of pest management, which were first advocated over 30 years ago by Stern et al. (1959).

The search for economically viable means of reducing insecticide use should be a top priority for continued research funding, as long as crop varieties, pest complexes, cultural practices and insecticides used continue to change: findings in this area will make crop production more profitable as well as delaying insecticide resistance. But researchers, extension specialists and consultants also need to present the current state of knowledge more convincingly, and win back the trust of farmers whose livelihoods depend on making a good crop.

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