SUSCEPTIBILITY MANAGEMENT OF TARNISHED PLANT BUG IN THE SOUTH

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Abstract

Development of insecticide resistance in the tarnished plant bug ,*Lygus lineolaris* (Palisot de Beauvois) has become a major concern for cotton growers in the southern U.S. Resistance seems to be associated with pyrethroid use, and resistance management recommendations encourage limited exposure of tarnished plant bug populations to the pyrethoids. Resistance levels are highest in the delta regions of Arkansas, Louisiana, and Mississippi where populations may be concentrated on cotton for one to two generations in June and July. The highly polyphagous nature of the insect has encouraged scientists to recommend the development of cultural management practices based on vegetation management and manipulation of preferred hosts. The wide host range of the insect also raises questions about the evolution of resistance in such a highly polyphagous pest with large population densities (or refuges) associated with non-crop hosts not treated with insecticide. A simple population model was developed to examine the possible role of population structure (portion of population on crop being sprayed versus that on non-sprayed, non-crop hosts) on resistance evolution in the tarnished plant bug. The model was designed to mimic typical population growth patterns of the tarnished plant bug in the Mississippi Delta and included simulated growth of eight generations per year. Two of the eight generations (the third and fourth) were "bottlenecked" on cotton and exposed to selection from an insecticide. Under these simulated conditions resistance evolved in 101 generations over a 12.6 year period. Pyrethroid insecticides have been used extensively in the Mississippi Delta for nearly 20 years.

Introduction

The tarnished plant bug, *Lygus lineolaris* (Palisot de Beauvois) is a classical key pest of cotton in the southern U.S. With the commercialization of cotton cultivars expressing insecticidal proteins for control of tobacco budworm, *Heliothis virescens* (F.), and bollworm, *Helicoverpa zea* (Boddie), and the anticipated successful eradication of the boll weevil, *Anthonomous grandis grandis* Boheman, tarnished plant bug may become the most important arthropod pest of cotton. Development of insecticide resistant populations of tarnished plant bug over the past decade in delta regions of Arkansas, Louisiana, and Mississippi (Hollingsworth et al. 1997, Pankey et al. 1996, Snodgrass and Elzen 1995, Snodgrass and Scott 1988, 1996, Snodgrass 1994, 1996a, 1996b) are of major concern because of the increasing pest status of the insect and the unavailability of alternative control measures. Resistance is not limited to the pyrethroid class of insecticides. Resistance to organophosphorous and cyclodiene chemistries have also been reported (Hollingworth et al. 1997, Snodgrass 1996a). Snodgrass and Elzen (1995) found multiple resistance in tarnished plant bug to different insecticide classes and cross resistance to all pyrethroids they tested. However, pyrethroids have been implicated as the most important insecticides selecting for resistance (Snodgrass 1996a), and resistance management strategies have focused on reducing exposure of tarnished plant bug populations to the pyrethroids.

Tarnished plant bug is one of the most polyphagous insects with hundreds of hosts (Young 1986). The seasonal history of tarnished plant bug in cotton growing regions of the southern U.S. has been followed by numerous researchers (Anderson et al. 1983, Cleveland 1982, Fleischer and Gaylor 1987, Snodgrass and Stadelbacher 1994, Snodgrass et al. 1984, Stadelbacher 1987, Tugwell et al. 1976, Womack and Schuster 1987). The insect overwinters as an adult, and two or more generations may be produced on non-crop hosts prior to the initiation of squaring in cotton. The abundance and distribution of suitable hosts during the spring are important to the population growth of the insect within a growing season. The adults move from patch to patch of different host plants as they become more or less attractive. Young parous females are generally the first colonizers (Stewart and Gaylor 1991, 1994). These early season hosts tend to senesce and decline in abundance during early summer when cotton begins to fruit. It is this decline of suitable non-crop host plants that encourages movement of adults into cotton (Cleveland 1982, Fleisher and Gaylor 1987, Snodgrass et al. 1984, Tugwell et al. 1976). Concentration of tarnished plant bug densities on cotton seems to be more common in the large-scale agriculture systems of the Delta than in the more diversified Hill regions of the southern U.S. Insecticide resistance is also more common in the Delta regions (Hollingsworth et al. 1997, Pankey et al. 1996, Snodgrass 1996a). Plant bugs have been found on other crops in the Delta regions, including soybean (Freeman and Mueller 1989, Lambert and Snodgrass 1979), but cotton is the most important crop affected by tarnished plant bug and probably the most important crop in population development of the insect. Two or more generations may be produced on cotton. Over

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the past few years there is a growing concern caused by increased plant bug densities on cotton during later periods of the growing season.

Researchers, particularly those at the USDA Southern Insect Management Laboratory in Stoneville, MS and those with the Department of Entomology at Auburn University, have intensely studied the ecology and seasonal phenology of tarnished plant bug (Fleischer and Gaylor 1987, 1988, Snodgrass et al. 1984). Results of their studies and earlier observations by numerous researchers suggest that tarnished plant bug populations could be managed by manipulating the availability or abundance of attractive hosts. We have initiated studies on the potential value of a trap crop system for tarnished plant bug control (Craig et al. 1997). Tarnished plant bug, like all insects, is regulated by natural control (Snodgrass and Fayad 1991, Snodgrass and Stadelbacher 1994, Young 1989a, 1989b), and some reductions in plant bug numbers have been observed when nectariless cottons were grown (Scott et al. 1988). However, the most important factors regulating population growth are those associated with abundance of non-crop hosts, particularly early-season non-crop hosts. It is this wide spread utilization of many host plants that creates a question about the value of refuges for insecticide resistance management and the factors important in the evolution of insecticide resistance in such a highly polyphagous insect. We examine this and other questions related to the management of insecticide resistance in tarnished plant bug in this paper. A chronological list of the development of resistance management strategies and recommendations for control of tarnished plant bug in Mississippi are summarized in Table 1.

Methods

A simple model of population growth of the tarnished plant bug was developed in a Quatro Pro Version 6 environment to study factors influencing insecticide resistance development. The model simulated annual dynamics of plant bug populations over eight generations per year. Natural mortality and fecundity were manipulated so that the standard (comparative) simulation would mimic typical seasonal phenology of the tarnished plant bug in the Mississippi Delta (Cleveland 1982, Snodgrass et al. 1984). Parameters included in the model, values of parameters for a standard simulation, and typical seasonal density patterns are summarized in Tables 2 and 3. Although the model was a simple life table, an effort was made to structure parameters similar to real-world observations reported in the literature (Cleveland 1982, Fleischer and Gaylor 1988, Snodgrass et al. 1984, Snodgrass 1996). Based on unpublished research of G. L. Snodgrass, the model assumed inheritance to be a single, recessive gene. Variations in the influence of this assumption on model results were tested in subsequent simulation experiments. The standard simulation assumed an initial gene frequency of 10^{-6}, an initial population of 1000, selection of the population with insecticide applications on cotton during generations 3 and 4, non-selection of the population during the remaining six generations, no population growth during overwintering between generation 8 and generation 1 of the next season, reduced natural mortality in the sprayed cotton environment, and reduced fecundity of resistant genotypes (Snodgrass 1996).

Experiments were conducted to measure the effects of intensity of insecticide selection (number of generations exposed) (Table 4), effective dominance of the resistant gene (Table 5), manipulation of mortality factors (mortality given exposure and proportion of population exposed) (Table 6), and initial gene frequency (Table 7) on time to resistance development. The population was considered resistant when 50% of the alleles were resistant. In reality, control problems would develop earlier. An examination of the possible impact of manipulating mortality factors and intensity of selection once resistance genes were fairly common (10^{-3} and 10^{-2}) was also made (Table 8).

Results

Selection for resistance during two generations per year and allowing non-selection for six generations per year resulted in resistance development in 101 generations or 12.6 years (Table 4). Selection during all eight generations per year resulted in resistance development in 13 generations (1.6 years). When selection was allowed during three generations each year, resistance developed in 79 or 62 generations depending upon the relative reproductive potentials of the selected generations. When selection was limited to one generation per year, resistance did not develop until 158 generations or 19.8 years.

As illustrated in Table 5, the dominance and inheritance of resistant genes is critical to the rate of resistance development. In the standard simulation, the resistant gene was assumed to be incompletely recessive. Heterozygotes (RS) died at a rate of 0.7. Those homozygous for susceptibility (SS) and resistance (RR) died at rates of 0.9 and 0.3, respectively. When the effective dominance was changed to make the resistant gene more recessive (0.8 mortality of RS), resistance was delayed until 244 generations (30.5 years). When effective dominance was changed to make the gene more dominant (0.5 mortality of RS), resistance developed much faster (78 generations). When the gene was made nearly completely dominant, resistance developed in 53 generations or 6.6 years.

Insecticidal mortality in the model was a product of mortality rate given exposure times the proportion of the population exposed. The standard simulation assumed that 95% of the tarnished plant bug population was bottlenecked on cotton during generations 3 and 4 and that the insecticide caused high mortality of susceptible genotypes (95%).

When the selection was reduced to 90% mortality of susceptibles (or 90% of population exposed), resistance developed in 109 generations, 8 generations longer than the standard simulation (Table 6). When mortality was reduced to 80% and the fraction of the population exposed was reduced to 50%, resistance was delayed until 156 generations.

Initial gene frequency is important in the subsequent development of resistance. Evolution of resistance is a progressive process, and it is important to know where you are in the process if you want to develop effective resistance management strategies. The time to resistance development ranged from 101 to 2 generations depending upon a range of gene frequencies from 10^{-6} to 10^{-1}, respectively.

Given that resistance has been measured in tarnished plant bug populations, it is likely that the current gene frequency is between 10^{-3} and 10^{-1}. Table 8 projects time to resistance (50% of alleles are resistant (R)) when mortality and selection were manipulated under one or two generations of selection and initial gene frequencies were 10^{-3} and 10^{-2}. When resistant genes were as common as 1 in 10 (10^{-1}), manipulating selection or mortality had no effect. Resistance developed in two generations.

Resistance can be delayed for 20 to 40 generations if the gene frequency is one in a thousand (10^{-3}), rather than one in a hundred (10^{-2}). Using the insecticide for only one generation rather than two increased time to resistance from 6 to 23 generations depending upon the mortality levels simulated. Using low mortality levels extends the time to resistance, but it is probably not useful in terms of protecting the crop from pest damage.

Discussion

Development of insecticide resistance in the highly polyphagous tarnished plant bug can be explained by a simple population model based on single, recessive gene inheritance. Resistance developed in 101 generations or 12.6 years when the model included selection for resistance during two of eight generations. Resistance would develop much faster (13 generations) if the insect was exposed to insecticides during all eight generations per year. This illustrates the importance of a refuge for species that concentrate most of their population on crops treated with insecticide. The prolonged development of resistance over several years also supports the general opinion that pyrethroid insecticides were largely responsible for insecticide resistance development in tarnished plant bug populations in the Midsouth. Pyrethroids have been used for ca. 20 years in Midsouth cotton. Current recommendations to avoid unnecessary selection of tarnished plant bug populations with pyrethroids seem warranted. Caution in excessive use of other insecticide chemistries is also warranted because of the presence of multiple resistance to different insecticide classes in some tarnished plant bug populations (Snodgrass and Elzen 1994, Snodgrass 1996). Development of resistance management strategies and an overall concern for insecticide resistance in tarnished plant bug is important because of the increasing pest status of the insect.

Given that insecticide resistant populations have been documented in the field, effective life of any available insecticide, particularly a pyrethroid, is shortened. Recommendations cautioning that effective control may not be achieved are warranted. However, increasing the number of applications will result in more rapid resistance evolution. Manipulating selection intensity and effective mortality levels by reducing exposure within and among generations will prolong the effective life of all insecticides. Avoiding multiple selection events and following resistance management guidelines seems to be prudent actions. More research is needed on population structure of tarnished plant bug populations and the genetics and toxicology of observed resistance.

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Table 1. Evolution of resistance management recommendations for the tarnished plant bug in Mississippi (MCES 1986-1997).

Year	Event or Change in Recommendations
1986	Pyrethroid resistance detected in field populations of tobacco budworm in Mississippi. (Luttrell et al. 1987). Midsouth recommendations for pyrethroid resistance management published (Anonymous 1986).
1987	Recommendations stated that pyrethroids should be targeted at heliothines and that use of insecticides against early season pests may increase problems with pyrethroid resistance in tobacco budworm. Tolerance of tarnished plant bug to dimethoate detected in different regions of Mississippi (Snodgrass and Scott 1988).
1988	The window strategy for use of pyrethroids was continued. Pyrethroids were recommended for use only after first bloom. Prebloom recommendations for control of plant bugs included organophosphorous insecticides and oxamyl.
1989	Similar to 1988. Pyrethroids recommended after first bloom. Plant bug recommendations included organophosphorous insecticides and oxamyl.
1990	Sulprofos and profenofos were no longer recommended prior to first bloom for heliothine control. Statements about switching chemistries when resistance to tobacco budworm was observed appeared in recommendations. Pyrethroids directed at heliothines post bloom were considered effective plant bug control measures.
1991	Pyrethroids were not recommended for late season use.
1992.	Statements about preventing outbreaks of secondary pests associated with use of organophosphorous insecticides, especially aphids, appeared in recommendations.
1993	Pyrethroid resistance detected in plant bug populations in cotton in Mississippi (Snodgrass 1994). Insecticide resistance problems with most pests recognized in recommendations. Recommendations strongly stated that pyrethroids and organophosphorous insecticides profenofos and sulprofos not be used prior to first bloom. Recommendations also stated that pyrethroids should not be used late in the year.
1994	Statements that varieties should be chosen for insect control appeared. Recommendations to mow the border of fields at correct times were included as non-insecticidal controls for plant bugs. Statements about not panicking in the face of insecticide control failures were included. Insecticide resistance in the plant bug thought to be associated with selection from pyrethroids. Cross resistance to all pyrethroids and multiple resistance to other insecticide classes suggested (Snodgrass and Elzen 1995).

Table 1. Cont.

1995	Statements that resistance to insecticides were now detectable in aphid, plant bug, and beet armyworm populations were included in recommendations. Resistance management strategies and justification expanded to include other pests, especially aphids and plant bugs. Goals of resistance management now included slowing the rate of resistance development in plant bugs. Strong recommendations to avoid use of pyrethroids late in the year were included. Insecticide resistance in plant bugs in Arkansas (Hollingsworth et al. 1997), Louisiana (Pankey et al. 1996), and wide-spread resistance in plant bugs in Mississippi (Snodgrass and Scott 1996, Snodgrass 1996a) detected.
1996	Expanding problems with insecticide resistant plant bugs created a sense of concern for plant bug control. The following statement appeared in recommendations: “Some pyrethroids have activity against plant bugs and, when applied against tobacco budworm/bollworm as the primary target, will provide control of low to moderate levels of plant bugs that may be present. Because resistance to both pyrethroids and organophosphates has been documented in populations of plant bugs at some locations, do not assume that treatments targeting tobacco budworm/bollworm will always provide effective control of plant bugs.” Statements about reduced use of insecticide in transgenic cotton expressing endotoxin protein of *Bacillus thuringiensis* and probable need for more insecticide targeted specifically at plant bug appeared. Vegetation management was again emphasized as a non-insecticidal control for plant bugs.
1997	Additional concerns for plant bug resistance to insecticide were evident. The following statement was made in the 1997 recommendations: "Because of insecticide resistance and/or difficulty obtaining adequate coverage in larger cotton, a single application of insecticide may not provide effective control of heavy established populations of plant bugs. Multiple applications applied at 4 to 5 day intervals may be required in such cases."

Table 2. Parameters used in simple spreadsheet model of insecticide resistance development in tarnished plant bug in the Mississippi Delta.

initial gene frequency-----	variable; 10^{-6} used in standard simulation (Table 3)
inheritance----------	single gene; recessive gene used in standard simulation
number of generations per year----------	8 generations per year; standard simulation included 6 generations on hosts not treated with insecticide and 2 on cotton where selection from insecticide application occurred
mortality----------	variable rates of natural mortality and insecticide mortality were included for each generation; natural mortality was similar for all genotypes and was reduced when insecticide applications were made; insecticide mortality varied by genotype and was a combined measurement of mortality given exposure and proportion of population exposed
population growth and fecundity----------	fecundity and natural mortality were manipulated to developa seasonal population pattern similar to those described byCleveland (1982) and Snodgrass et al. (1984); resistantgenotypes were less fecund than susceptible genotypes (Snodgrass 1996a); all mating was random and gene frequencies were redistributed by Hardy-Weinburg ratios following survival and mating within each generation
overwintering----------	population growth did not occur during the winter months (i.e. after 8th generation each year); natural mortality reduced population numbers of overwintering adults (Cleveland 1982) near those observed emerging from overwintering the previous year, thus there was little change in population densities among years.

Table 3. Values of parameters used in standard simulation.

Generation	Natural Mortality	Insecticide Mort.			Eggs per Female			Resulting Popluation
		SS	RS	RR	SS	RS	RR	
1st(Mar-Apr)	0.95	0.0	0.0	0.0	200	150	100	5,000
2nd(Apr-May)	0.98	0.0	0.0	0.0	200	150	100	10,000
3rd(May-Jun)	0.85	0.9	0.7	0.3	100	75	50	7,500
4th(Jun-Jul)	0.85	0.9	0.7	0.3	100	75	0	5,625
5th(Jul-Aug)	0.98	0.0	0.0	0.0	200	150	100	11,250
6th(Aug-Sep)	0.99	0.0	0.0	0.0	200	150	100	9,000
7th(Sep-Oct)	0.98	0.0	0.0	0.0	200	150	100	6,750
8th(Oct-Nov)	0.85	0.0	0.0	0.0	0	0	0	1,012
Initial Gene Frequency = 10^{-6}					Initial Population Density = 1000			

Table 4. Simulated effect of intensity of insecticide selection on time to resistance (50% of alleles are resistant; initial gene frequency of 10^{-6}).

Selection	Time to Resistance
Standard Simulation (selection in 3rd and 4th generation)	101 generations (12.6 years)
Selection Every Generation (1st-8th generations)	13 generations (1.6 years)
Selection in Three Generations (2nd, 3rd, 4th generations)	79 generations (9.9 years)
Selection in Three Generations (3rd, 4th, 5th generations)	62 generations (7.8 years)
Selection in Only One Generation (3rd or 4th generation)	158 generations (19.8 years)

Table 5. Simulated effect of effective dominance on time to resistance (50% of alleles are resistant; initial gene frequency of 10^{-6}).

	Insecticide Mortality			
	SS	RS	RR	Time to Resistance
Standard Simulation	0.9	0.7	0.3	101 generations (12.6 years)
More Recessive	0.9	0.8	0.3	244 generations (30.5 years)
Less Recessive	0.9	0.5	0.3	78 generations (9.8 years)
Near Complete Dominant	0.9	0.4	0.3	53 generations (6.6 years)

Table 6. Simulated effect of insecticide mortality (probability of mortality given exposure times proportion of population exposed) on time to resistance (50% of alleles are resistant, initial gene frequency of 10^{-6}, relative ratios of genotypes held constant.

	Insecticide Mortality			
	SS	RS	RR	Time to Resitance
Standard Simulation (95% mort., 95% exposed)	0.9	0.7	0.3	101 generations (12.6 years)
Increased Exposure (95% mort., 100% exposed)	0.95	0.74	0.32	62 generations (7.8 years)
Smaller Increase in Exposure (95% mort., 99% exposed)	0.94	0.73	0.31	70 generations (8.8 years)
Reduced Exposure (95% mort., 90% exposed)	0.86	0.66	0.28	109 generations (13.6 years)
Reduced Exposure and Mortality (80% mort., 90% exposed)	0.72	0.56	0.24	126 generations (15.8 years)
Further Reduced Exposure (95% mort., 50% exposed)	0.48	0.37	0.16	150 generations (18.8 years)
Further Reduced Exposure and Mortality (80% mort., 50% exposed)	0.40	0.31	0.13	156 generations (19.5 years)

Table 7. Simulated effect of initial gene frequency on time to resistance (50% of alleles are resistant; standard selection with 2 generations on cotton (Table 3)).

Initial Gene Frequency	Time to Resistance
10^{-6} (one resistant gene in one million)	101 generations (12.6 years)
10^{-5} (one resistant gene in one hundred thousand)	85 generations (10.6 years)
10^{-4} (one resistant gene in ten thousand)	62 generations (7.8 years)
10^{-3} (one resistant gene in one thousand)	37 generations (4.6 years)
10^{-2} (one resistant gene in one hundred)	12 generations (1.5 years)

Table 8. Simulated effect of manipulating selection intensity (number of generations and proportion of population exposed) on time to resistance (number of generations until 50% of alleles are resistant, standard simulation parameters in Table 3) once resistant genes are as common as one in a thousand (10^{-3}) to one in a hundred (10^{-2}).

		One Selection (3rd Generation)		Two Selections (3rd and 4th Generation)	
	Fraction	Initial R Frequency		Initial R Frequency	
Mort.	Exposed	10^{-3}	10^{-4}	10^{-3}	10^{-2}
0.95	0.95	55 (6.8 yr)	18 (2.2 yr)	37 (4.6 yr)	12 (1.5 yr)
0.95	0.90	61 (7.6 yr)	20 (2.5 yr)	38 (4.8 yr)	13 (1.6 yr)
0.95	0.80	66 (8.2 yr)	22 (2.8 yr)	46 (5.8 yr)	15 (1.8 yr)
0.95	0.50	75 (9.4 yr)	26 (3.2 yr)	58 (7.2 yr)	22 (2.8 yr)

(time to resistance is 2 generations under all conditions tested with a R frequency of 10^{-1})