

OVICIDAL EFFECTS OF SELECTED INSECTICIDES AGAINST  
PYRETHROID-SUSCEPTIBLE AND -RESISTANT TOBACCO BUDWORM<sup>1</sup>B. R. Leonard, J. B. Graves, and T. C. Sparks<sup>2</sup>Department of Entomology, Louisiana Agricultural Experiment Station  
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## ABSTRACT

A dipping technique was used to test the ovicidal activity of selected pyrethroid, carbamate, formamidine and organophosphate insecticides against pyrethroid-susceptible (PY-S) and pyrethroid-resistant (PY-R) laboratory strains and a field (FIELD-89) strain of tobacco budworm, *Heliothis virescens* (F.). The PY-R and FIELD-89 strains were determined to be 65-239 times and 1.3-3.1 times resistant, respectively, to pyrethroids relative to the PY-S strain using topical application procedures against third instar larvae.  $LC_{50}$ 's for all insecticides except profenofos on eggs of the PY-R strain were significantly higher than  $LC_{50}$ 's for the same insecticide on eggs of the PY-S strain. Furthermore, all insecticides except profenofos and methomyl were significantly less toxic to eggs of the FIELD-89 strain compared with their respective toxicity to eggs of the PY-S strain. Eggs of the PY-R strain exhibited resistance to esfenvalerate (13.6 times), *lambda*-cyhalothrin (34.9 times), and chlordimeform (5.4 times), while eggs of the FIELD-89 strain possessed resistance to *lambda*-cyhalothrin (11.7 times), chlordimeform (6.5 times), and SN 49844, *N*'-[2,4-dimethylphenyl]-*N*-methylformamidine (7.2 times). Chlordimeform and methomyl were generally the most toxic insecticides tested against eggs of tobacco budworm, regardless of pyrethroid susceptibility. Profenofos was significantly less toxic than other insecticides to eggs of the PY-S and PY-R strains and less toxic than all insecticides except thiodicarb to eggs of the FIELD-89 strain.

## INTRODUCTION

Pyrethroid resistance in field strains of tobacco budworm, *Heliothis virescens* (F.), has been reported in most of the cotton producing states in which this insect is a pest of cotton (Leonard et al. 1988a, Luttrell et al. 1987, Plapp et al. 1987). Management strategies have been developed to delay the widespread development of resistance to levels that will prohibit the use of the pyrethroids (Anonymous 1986, Plapp 1987, Graves et al. 1988). These strategies commonly recommend the use of an ovicide in combination with a larvicide and the timing of insecticide applications against infestations of tobacco budworm in the egg or neonate larval stages of development. Chlordimeform was an important component in mixtures with pyrethroids and provided both synergistic and ovicidal activity (Plapp 1976, 1979, Campanhola and

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Plapp 1987, Graves et al. 1988). The loss of chlordimeform during 1989 has caused researchers to intensify efforts in the evaluation of chlordimeform alternatives as ovicides and synergists.

Numerous studies have evaluated the ovicidal activity of a variety of selected organochlorine, organophosphate, carbamate, formamidine, and pyrethroid insecticides (Walker 1966, Tysowsky and Gallo 1977, Bull and House 1978, Campbell et al. 1979, Pitts and Pieters 1980, Horowitz et al. 1987, Bradley and Agnello 1988) as well as insect growth regulators (Masner et al. 1987) to eggs of tobacco budworm and bollworm, *Helicoverpa zea* (Boddie). Because tobacco budworm eggs may be present on cotton plants throughout much of the growing season, they may be exposed to a variety of insecticides that are applied for control of other cotton pests. These eggs are inadvertently under insecticide selection pressure that can lead to altered levels of susceptibility. In some of these studies, differences in the insecticide susceptibility of eggs from different tobacco budworm strains have been observed. Bull and House (1978) reported that chlordimeform was less toxic to eggs of a methyl parathion-resistant strain of tobacco budworm than to eggs of a methyl parathion-susceptible strain. Chlordimeform, amitraz, methomyl, monocrotophos, acephate, methyl parathion, and fenvalerate also were observed to be significantly less toxic to eggs from a field strain compared with eggs from a laboratory strain (Horowitz et al. 1987).

The relative insecticidal susceptibility of eggs from different tobacco budworm strains is not well known because standard monitoring procedures for insecticide resistance typically focus on the larval or adult stages of this insect. Current management strategies for tobacco budworm target insecticide applications to the egg and early larval stages. These practices coupled with the potential for resistance development in this pest justify determining the susceptibility of eggs from pyrethroid-resistant tobacco budworms to selected insecticides. The objective of this study was to evaluate the ovicidal toxicity of insecticides from several classes against strains of tobacco budworm with varying levels of pyrethroid susceptibility in the larval stage.

## MATERIALS AND METHODS

Insects. Two laboratory strains and one field strain (FIELD-89) were included in this study. The pyrethroid-susceptible laboratory strain, PY-S, was initiated from a collection of larvae obtained from a cotton field in Louisiana during 1977. This collection was made before widespread use of the pyrethroids in cotton and has been maintained in continuous culture in the laboratory without exposure to insecticides. The pyrethroid-resistant laboratory strain, PY-R, was obtained from ICI Americas Inc. (Wilmington, Del.) during September 1988. This strain had been continuously pressured with cypermethrin but was not further exposed to insecticides after being colonized in our laboratory. The egg bioassays were conducted during the F<sub>5</sub>-F<sub>6</sub> generations of the PY-R strain in our laboratory. The FIELD-89 strain was established from a collection of ca. 380 eggs taken from a cotton field on the Northeast Research Station, St. Joseph, La., on 24 and 25 August 1989. This strain represents the survivors of an insecticide trial (one application) to evaluate the ovicidal efficacy of selected pyrethroids. No other insecticide had been applied to this field, but the test site was in close proximity to several large fields of commercially produced cotton that received multiple applications of pyrethroids, organophosphates, and carbamates throughout the season.

The tobacco budworm strains were all reared in a similar manner according to the laboratory procedures described by Leonard et al. (1988a). Larvae were fed a modified pinto bean and wheat germ artificial diet (Shour and Sparks 1981). Adults were held in 3.78 L cardboard cartons covered with cotton gauze and were fed an aqueous (1:10 ratio, sugar:water) solution. Tobacco budworm adults oviposited on the cotton gauze and these eggs were used in the egg bioassays or placed on artificial

diet for larval rearing. Insects were held at a 14:10 (L:D) photoperiod,  $28 \pm 3^\circ\text{C}$  and 65-70% RH for the duration of this experiment.

**Insecticides.** Technical grade insecticides were used in the bioassays to determine pyrethroid toxicity to larvae, but formulated materials were used in the tests to compare ovicidal activity. Samples of cypermethrin (FMC Corp., Middleport, N.Y.), *lambda*-cyhalothrin (ICI Americas Inc., Wilmington, Del.) and esfenvalerate (E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.) were obtained from the manufacturers. Technical materials were diluted with acetone to a 10% stock solution and refrigerated at  $15^\circ\text{C}$  until needed for bioassays. The formulated insecticides evaluated for ovicidal activity were esfenvalerate (60 g/liter emulsifiable concentrate [EC]; E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.), *lambda*-cyhalothrin (120 g/liter EC; ICI Americas Inc., Wilmington, Del.), thiodicarb (384 g/liter flowable liquid [F]; Rhone-Poulenc, Research Triangle Park, N.C.), methomyl (288 g/liter liquid [L]; E. I. DuPont de Nemours & Co. Inc., Wilmington, Del.), amitraz (180 g/liter EC; NOR-AM Chemical Co., Wilmington, Del.), chlordimeform (480 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.), and profenofos (960 g/liter EC; CIBA-GEIGY Corp., Greensboro, N.C.). The numbered compound SN 49844 (BTS-27712, *N*'-[2,4-dimethylphenyl]-*N*-methylformamidine, 100% soluble powder [SP]), which was obtained from NOR-AM Chemical Co., Wilmington, Del., was also evaluated for ovicidal activity. The insecticides evaluated in this study were selected from those recommended in Louisiana for larvicidal or ovicidal control of tobacco budworm in cotton with the exception of amitraz and SN 49844 which are potential replacements for chlordimeform (Baldwin et al. 1989).

**Bioassays.** Tests to determine pyrethroid susceptibility in tobacco budworm larvae were done according to a standard method for determining insecticide resistance in tobacco budworm and bollworm (Anonymous 1970). Ten to fifteen larvae (third stage, mean weight =  $25 \pm 4$  mg) were treated with each of four to six concentrations of insecticide in acetone. Each dose was replicated at least three times. One microliter of the insecticide solution or acetone (controls) was applied to the dorsal surface of the thorax of each larva. Treated insects were individually held in 29.7-ml plastic cups containing artificial diet. Mortality data were recorded 72 h after treatment. A larva was considered dead if it failed to upright itself after being overturned with a blunt probe. Dose-mortality regressions were determined with a microcomputer based probit analysis (MicroProbit 3.0, T. C. Sparks and A. P. Sparks, unpublished, Greenfield, Ind.). All data were corrected for mortality (< 5%) in the controls (Abbott 1925). Significant differences among  $\text{LD}_{50}$  values were determined by the failure of 95% confidence limits (CL) to overlap. Pyrethroid  $\text{LD}_{50}$ 's were determined for larvae of the PY-S strain within the same generation and these results are adapted from a previous study (Leonard et al. 1988b). Larvae of the PY-R strain were tested with pyrethroids one generation before the ovicidal evaluation. Larvae of the FIELD-89 strain were bioassayed with cypermethrin and esfenvalerate in the  $F_1$  generation and with *lambda*-cyhalothrin during the  $F_2$  generation.

Insecticide toxicity to eggs was evaluated using a dipping technique modified from previous methods (Horowitz et al. 1987). Sheets of cotton gauze covering the adult oviposition chambers were removed daily, placed in inflated plastic bags and held for 24 h to allow further egg development. Pieces of cotton gauze were cut to contain a sample of 15 to 30 fertile eggs. Fertility (embryonic development) was indicated by the presence of a red band (germ band) around the circumference of the eggs. These samples were submerged into an insecticide distilled water (pH = 6.4-6.8) solution for 4 s and air dried for 30 min in a fume hood. After drying, the treated egg samples were placed on No. 1 filter paper in plastic petri dishes (10 cm). Four to seven doses in three replications were used to obtain a dose-mortality line for each insecticide. Egg mortality was determined at 72 h after treatment. Any larva that fully eclosed from the chorion was recorded as a hatched egg. All data were analyzed with the

same procedures used in the larval tests. Control mortality in the egg bioassays ranged from 0 to 12%.

## RESULTS AND DISCUSSION

Larval LD<sub>50</sub>'s for the PY-R strain to cypermethrin, esfenvalerate, and *lambda*-cyhalothrin were 207, 239, and 65 times higher, respectively, than LD<sub>50</sub>'s for the PY-S strain (Table 1). The larval LD<sub>50</sub>'s for cypermethrin and esfenvalerate on the FIELD-89 strain were much lower than those of the PY-R strain being only 3.1 and 3.0 times, respectively, higher than those reported for the PY-S strain. There was no significant difference in the toxicity of *lambda*-cyhalothrin between the PY-S and FIELD-89 strains. Slope values of the dose-response lines determined for the pyrethroids against larvae were somewhat higher for the PY-S strain compared to the slopes of the PY-R and FIELD-89 strains. The higher slope values for the PY-S strain are to be expected for a laboratory strain that has not been exposed to insecticides for many generations.

TABLE 1. Toxicity of Selected Pyrethroids to Larvae of Pyrethroid-Susceptible (PY-S), Pyrethroid-Resistant (PY-R) and Field (FIELD-89) Strains of Tobacco Budworm.

Insecticide	Strain <sup>a</sup>	No. Tested	Slope ± SE	LD <sub>50</sub> <sup>b</sup> (95% CL)	RR <sup>c</sup>
Cypermethrin	PY-S	150	3.50 ± 0.60	1.61 (1.32-1.93)	---
	PY-R	237	1.89 ± 0.26	333.89 (268-416)	2074
	FIELD-89	221	1.87 ± 0.24	4.96 (3.92-6.46)	3.1
Esfenvalerate	PY-S	150	1.74 ± 0.38	0.42 (0.11-0.49)	---
	PY-R	142	1.43 ± 0.28	100.41 (61.08-146)	239.0
	FIELD-89	294	1.73 ± 0.17	1.25 (1.00-1.58)	3.0
<i>lambda</i> -Cyhalothrin	PY-S	150	2.64 ± 0.37	0.93 (0.68-1.14)	---
	PY-R	178	1.34 ± 0.22	60.42 (33.09-89)	65.0
	FIELD-89	190	1.49 ± 0.21	1.17 (0.84-1.69)	1.3

<sup>a</sup> Results for LSU PY-S strain adapted from Leonard et al. (1988b).

<sup>b</sup> Dosages are reported in µg insecticide per g larval weight.

<sup>c</sup> RR (resistance ratio) = (LD<sub>50</sub> PY-R or FIELD-89 strain)/(LD<sub>50</sub> PY-S strain).

Esfenvalerate and *lambda*-cyhalothrin were significantly more toxic to eggs of the PY-S strain than other insecticides except for chlordimeform and SN 49844 (Table 2). Based on LC<sub>50</sub> values, chlordimeform was one of the most toxic insecticides to eggs of the three tobacco budworm strains, while profenofos was consistently the least toxic. All insecticides except profenofos were more toxic to eggs of the PY-S strain compared with the toxicity of the same insecticide to eggs of the PY-R strain. All insecticides except for profenofos and methomyl were more toxic to eggs of the PY-S strain when compared with their toxicity to eggs of the FIELD-89 strain. The highest level of resistance was found with esfenvalerate (13.6 times) and *lambda*-cyhalothrin (34.9 times) in eggs of the PY-R strain. Relatively low levels of resistance were found to all other insecticides except chlordimeform (5.4 times) in this strain.

TABLE 2. Toxicity of Insecticides to Eggs of Pyrethroid-Susceptible (PY-S), Pyrethroid-Resistant (PY-R) and Field (FIELD-89) Strains of Tobacco Budworm.

Insecticide	PY-S Strain			PY-R Strain			FIELD-89 Strain		
	n	Slope ± SE	LC <sub>50</sub> (mg[A.I.]/ml)	n	Slope ± SE	LC <sub>50</sub> (mg[A.I.]/ml)	n	Slope ± SE	LC <sub>50</sub> (mg[A.I.]/ml)
<b><u>PYRETHROIDS</u></b>									
Lambda-Cyhalothrin	527	1.95 ± 0.18	0.017 (0.014-0.019)	413	1.34 ± 0.14	0.593 (0.467-0.774)	206	1.98 ± 0.27	0.199 (0.063-0.358)
Esfenvalerate	439	2.12 ± 0.15	0.043 (0.036-0.050)	368	1.31 ± 0.15	0.586 (0.446-0.821)	NB <sup>b</sup>		
<b><u>CARBAMATES</u></b>									
Methomy1	302	1.77 ± 0.20	0.081 (0.063-0.103)	350	1.92 ± 0.18	0.157 (0.130-0.192)	1.9	2.15 ± 0.37	0.108 (0.091-0.130)
Thiodicarb	1173	2.49 ± 0.14	0.218 (0.198-0.241)	712	2.41 ± 0.19	0.287 (0.256-0.323)	1.3	2.63 ± 0.16	0.851 (0.596-1.406)
<b><u>FORMAMIDINES</u></b>									
Chlordimeform	792	1.79 ± 0.14	0.016 (0.014-0.020)	839	1.03 ± 0.10	0.086 (0.069-0.106)	5.4	2.17 ± 0.22	0.104 (0.088-0.124)
SN 49844	542	1.34 ± 0.11	0.043 (0.033-0.054)	473	1.03 ± 0.12	0.125 (0.096-0.166)	2.9	1.87 ± 0.19	0.309 (0.251-0.379)
Amitraz	635	1.88 ± 0.20	0.122 (0.098-0.145)	959	2.92 ± 0.16	0.331 (0.277-0.403)	2.7	4.61 ± 0.55	0.367 (0.289-0.463)
<b><u>ORGANOPHOSPHATE</u></b>									
Profenofos	495	1.48 ± 0.16	1.087 (0.858-1.479)	350	1.30 ± 0.16	1.099 (0.825-1.590)	1.0	1.82 ± 0.17	1.182 (0.790-1.64)

<sup>a</sup> RR (resistance ratio) = (LC<sub>50</sub> PY-R or FIELD-89 strain)/(LC<sub>50</sub> PY-S strain).

<sup>b</sup> Not bioassayed.

The highest level of resistance in the FIELD-89 strain was to *lambda*-cyhalothrin (11.7 times). This strain also exhibited a moderate level of resistance to chlordimeform (6.5 times) and SN 49844 (7.2 times). There was no significant difference among  $LC_{50}$ 's of the tobacco budworm strains to profenofos. The slopes for dose-response lines of insecticides are slightly steeper for eggs of the FIELD-89 strain compared to slopes determined for the same insecticide tested against eggs of the PY-S and PY-R laboratory strains. Also, there was much more variation in the range of slope values for the FIELD-89 strain compared to the range of values for the PY-S and PY-R strain.

Although different procedures were used to measure pyrethroid susceptibility in eggs and third instar larvae, both techniques detected a significant decrease in pyrethroid toxicity to the PY-R and FIELD-89 strains compared with the PY-S strain. The variation in the susceptibility of eggs from the different tobacco budworm strains to esfenvalerate and *lambda*-cyhalothrin is probably the result of differential selection pressure in the field (FIELD-89 strain) and laboratory (PY-R strain) with the pyrethroids. The levels of resistance to *lambda*-cyhalothrin in third instar larvae (65.0 times) and eggs (34.9 times) were much higher than the resistance levels found in larvae (1.3 times) and eggs (11.7 times) of the FIELD-89 strain. Pyrethroid resistance levels in larvae of the PY-R strain were higher than resistance levels in eggs which could be related to selection pressure being applied to larvae of a laboratory adapted strain. Conversely, pyrethroid resistance in eggs of the FIELD-89 strain may be higher than in larvae of this strain because it was a field collection that was exposed to insecticides as eggs, larvae and adults. The PY-R strain has only been exposed to cypermethrin as a selecting agent, but resistance to esfenvalerate and *lambda*-cyhalothrin was observed in larvae and eggs of this strain. In a previous study, larvae of a pyrethroid-resistant (labeled ICI) strain from the same source as the PY-R strain were found to be resistant to all pyrethroids to which they were exposed (Campanhola and Plapp 1987).

The toxicities of most of the non-pyrethroid insecticides against the PY-S strain were similar to that obtained against eggs of the PY-R and FIELD-89 strains. The FIELD-89 strain had been exposed to a variety of insecticides before its removal from the field and was generally less susceptible than the PY-S strain to most of the insecticides tested in this study. Using a similar technique on eggs, Horowitz et al. (1987) found significant differences in the  $LC_{50}$ 's of pyrethroids, formamidines, carbamates and organophosphates between eggs of a field tobacco budworm strain collected in Southern California and eggs of a laboratory tobacco budworm strain. Using an insecticide coated vial test, Bagwell and Plapp (1988) found chlordimeform to be significantly more toxic than amitraz to eggs of pyrethroid-resistant and pyrethroid-susceptible laboratory tobacco budworm strains.  $LC_{50}$  values for eggs of the pyrethroid-resistant strain were determined to be 3.9 and 3.3 times higher for chlordimeform and amitraz, respectively, compared to eggs of the pyrethroid-susceptible tobacco budworm strain. Eggs of a methyl parathion-resistant tobacco budworm strain were less susceptible (2.2 times) to chlordimeform than eggs of a methyl parathion-susceptible laboratory tobacco budworm strain (Bull and House 1978). The results of these and similar studies indicate that while eggs of field-collected or insecticide-pressured tobacco budworm strains are less susceptible to insecticides compared with eggs of insecticide-susceptible tobacco budworm strains, the levels of insecticide resistance in eggs among tobacco budworm strains is often quite low.

The relative toxicity of different insecticides to tobacco budworm eggs in the present study indicated chlordimeform was the most toxic formamidine and methomyl was the most toxic carbamate tested on the PY-S strain. Other studies (Pitts and Pieters 1980, Gonzales and Allen 1985, Horowitz et. al. 1987, Bradley and Agnello 1988) have also found chlordimeform, methomyl, and thiodicarb to be highly toxic to

tobacco budworm and bollworm eggs from field collections and laboratory strains. In our study, amitraz was significantly less active than chlordimeform against tobacco budworm eggs of all strains. Pitts and Pieters (1980) found amitraz to be significantly less toxic than chlordimeform to tobacco budworm eggs, but several field studies have shown that the ovicidal activity of amitraz is equal to that of chlordimeform (Elzen 1989, Leonard et al. 1989, Micinski et al. 1989). A comparison of the toxicity of selected formamidines to tobacco budworm eggs in a laboratory test indicated chlordimeform to be slightly, but not significantly more toxic than SN 49844 to bollworm eggs (Gemrich et al. 1976). The pyrethroids, esfenvalerate and *lambda*-cyhalothrin, were highly active ovicides against the PY-S and FIELD-89 strains in this study. Several studies have demonstrated the toxicity of various pyrethroids to tobacco budworm eggs (Pitts and Pieters 1980, Jany 1984, Horowitz et al. 1987, Leonard et al. 1989). Profenofos exhibited the lowest ovicidal activity in this study, but field tests have indicated that the contact toxicity of profenofos to tobacco budworm eggs was not significantly different from that of chlordimeform and thiodicarb (Leonard et al. 1989, Micinski et al. 1989).

The mode of insecticidal action in insect eggs is not well understood and at least two types of mortality are associated with death of the developing larva. The embryo in the egg may be killed or the developing larva dies as it feeds on the chorion during eclosion. Both types of mortality were observed in this study, but no attempt was made to differentiate between the two. These variations in embryo death have been observed in studies that have evaluated the ovicidal toxicity of several different classes of insecticides including formamidines, carbamates and organophosphates (Walker 1966, Gemrich et al. 1976, Bradley and Agnello 1988). Studies with chlordimeform-treated *Spodoptera littoralis* (Boisduval) eggs have shown that larval survival can be increased if the larva is removed from the chorion before it begins to feed during eclosion (Salvisberg et al. 1980). For insecticide-treated ova, the ingestion of egg materials during the hatching process contributes significantly to intoxication of neonate larva. In our experiment, the eggs were completely submerged in the insecticide-water solution and all of the chorion should have been exposed to the insecticide. Although insecticides are taken up by the eggs at the time of treatment, they may not become lethal until further development, depending on the dose that actually reaches the embryo. They may be retained in the egg until further embryonic development or the dose of insecticide becomes lethal through continuous exposure and/or metabolism. Storage either within the egg or on the chorion would increase the potency of the toxicant by increasing the overall amount received by the embryo. Although the structural characteristics of the chorion are designed to prevent desiccation, there are openings in the chorion (aeropyles and micropyle) for gas exchange to occur (Smith and Salkeld 1966). Insect eggs have an extensive respiratory system that may allow uptake and storage of a toxicant without affecting the embryo until a later stage of development. As the embryo undergoes significant changes in physiology from an immobile physical stage to one that is capable of movement, stress levels are increased. The formamidines at sublethal doses are capable of disrupting behavioral patterns in insects, thereby increasing stress and accentuating the potential for starvation and dehydration (Lund et al. 1979, Benedict et al. 1990).

Insecticides used against tobacco budworm and bollworm in cotton generally target the larval stages. The recommended use rates are higher than the amount that is lethal to the egg stage. In a field study in Australia, researchers found no significant differences in egg or early instar larval (< 3 day old) mortality between pyrethroid-susceptible and pyrethroid-resistant *Heliothis armigera* (Hubner) when eggs were exposed to fenvalerate (Daly et al. 1988). In this case, the field dose may actually be an overdose of insecticide for this developmental stage because resistance was observed in older larvae (> 4 day old). However, in other field studies, *lambda*-cyhalothrin at recommended field rates was less toxic to eggs and neonate larvae of

a laboratory selected pyrethroid-resistant tobacco budworm strain compared with eggs and neonate larvae of a pyrethroid-susceptible strain (Treacy et al. 1988, Hopkins et al. 1989). Thus, pyrethroid resistance in eggs may be detected in laboratory bioassays but may not be recognized in the field if recommended rates of insecticides are being used. The ovicidal activity of insecticides to tobacco budworm should not be underestimated for use as a resistance management tool as pyrethroid resistance management strategies emphasize the use of ovicides and application timing to coincide with the egg or early larval stages.

Consideration should be given to a standardized system of monitoring insecticide susceptibility in the egg stage of tobacco budworm. A monitoring system is fundamental to extending the usefulness of insecticides and allowing appropriate measures to be taken before such a problem would become unmanageable.

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