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A REVIEW OF THE INHERITANCE OF INSECTICIDE RESISTANCE BY THE TOBACCO BUDWORM (NOCTUIDAE: LEPIDOPTERA)

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Abstract

Review of literature found 19 patterns of inheritance of insecticide resistance by tobacco budworm strains and their reciprocal crosses. Patterns of inheritance were shown for three organophosphorus, one carbamate, two pyrethroid insecticides and a toxin of transgenic *Bacillus thuringiensis* (Bt). Tests were conducted from 1978 to 2001 and patterns were determined in the first generation. Resistant (R] and susceptible (S) strains and crosses were treated with topical applications of the chemical insecticides to determine LD50s. Bt toxin was added to diet prior to placing neonate larvae on the diet to determine LC50s. Overlapping or non-overlapping 95% confidence limits of the LD50s or LC50s were used to indicate dominance or incomplete dominance, sex-linkage and recessive inheritance patterns. Eighty-nine percent of the patterns showed significant differences between R and S strains. Incomplete dominance occurred in 58 % of the patterns. Dominance, recessivity and sex linkage were determined in 21 % of the patterns. In tests with monocrotophos one strain showed a pattern of dominant sex-linkage while a second strain showed a pattern of incompletely dominant sex-linkage. In three tests using methyl parathion, two strains showed incomplete dominant autosomal inheritance while one strain showed sex-linked dominant inheritance. Slope values >3.44 are suggested to indicate homozygosity of response (resistance or susceptibility) by strain or crosses of strains.

Introduction

Certain anti-cholinesterase and pyrethroid insecticides are effective against the tobacco budworm, *Heliothis virescens* (F.) (TBW) in cotton fields in the Americas. These include the pyrethroids, i.e. cypermethrin and permethrin, the organophosphorus insecticides, i.e. methyl parathion, monocrotophos and profenofos and the carbamate, i.e. methomyl. Transgenic Bt cotton has had wide acceptance with producers for the control of the TBW. Pyrethroids and anticholinesterase insecticides are effective against >70% of the larval populations of this species in the Americas. Resistance by the TBW has not been found in any of the transgenic Bt cotton fields in the Americas. Methyl parathion has been used for 50 y, monocrotophos for 40 y, profenofos and thiodicarb for 30 y, the pyrethroids for 20 y and transgenic Bt cottons for 10 y against this pest.

Inheritance patterns of resistance obtained by means of the response of reciprocal crosses and their parental strains of the TBW to cypermethrin, EPN, methomyl, methyl parathion, monocrotophos, permethrin, profenofos and a Bt toxin were taken from the literature. The objective was to relate these patterns for dominance, incomplete dominance, recessive and sex-linkage with the strains tested.

Materials and Methods

The insecticides and strains used in these inheritance studies were referenced by the authors (Table 1).

The 95% confidence interval of LD50s was used to indicate the inheritance category as dominant, incomplete dominance (also called co-dominate), recessive or sex linkage of the R strain in generation one. Only data with confidence intervals for both reciprocal crosses and both parents were used unless the author determined that there was not a significant difference between the reciprocal crosses. The female is listed first in all crosses. LD50s were equal when the confidence intervals overlapped.

In the TBW the female is heterogametic (say XO) and male is homogametic (say XX). Thus, in the reciprocal crosses, there are different expectations of resistance in the F1 progeny depending upon which parent carried the resistant alleles and upon the dominance relations of the alleles.

Herein, we shall designate susceptible alleles by the symbol S and resistant alleles by the symbol R. We will also assume homozygosity of both strains and equal sex ratios in the F1 progeny (these assumptions are necessary for simplification of the illustration). A slash symbol will indicate the chromosome, thus S/ indicates a female (hemizygous) with a susceptible allele, while R/R would indicate a homozygous resistant male. If a susceptible female (S/) were crossed with a resistant male (R/R) then all F1 female progeny would carry only the resistant allele (R/), while all of the F1 male progeny would be heterozygous (R/S).

If the resistance were inherited as a completely dominant character then all the F1 progeny (R/S males and R/females) would be similar to the resistant parent.

If the trait were incompletely dominant, the F1 progeny would show an average response lower than the dominant parent but much higher than the recessive parent (i.e., the females would be completely resistant, but the males, being heterozygous, would be intermediate in response).

If the resistant character is due to a recessive allele, then the males will all be like the susceptible parent, but the females will be like the resistant parent (since they have the genotype R/). The response curve will be quite flat and the resistance will appear to be inherited as an incompletely dominant character. Conversely, if a resistant female (R/) was crossed to a susceptible male (S/S), then all F1 males would be heterozygous for the trait (R/S) and all F1 females would be hemizygous for the susceptible allele (S/).

If the resistant allele was dominant to the susceptible allele, a strange response curve would result in similarity of all the F1 male progeny (R/S) with the female parent (resistant). All the F1 female progeny (S/) would be similar to the male parent (susceptible). The average response would make this look like an incompletely dominant character with a very flat response curve (just as in the case of a sex-linked recessive resistant allele.)

If the resistant allele was inherited as an incompletely dominant trait, then the F1 response curve would be closer to the susceptible parent than to the resistant parent (the male progeny would show an intermediate response while the female progeny would show susceptible response).

If the resistance was inherited as a recessive character, then all the F1 progeny would be susceptible and look like the response of the susceptible parent.

The key to interpreting sex-linked inheritance is that the reciprocal crosses will look so different from each other. For a dominant trait, if the resistant parent is the male, the F1 average will look like the resistant strain. For a recessive trait, if the susceptible parent is the male, the F1 progeny will look like the susceptible strain. An incompletely dominant resistance character using either sex as the resistant parent will give an intermediate result which will favor the response of the male parent.

Results

An inheritance pattern of incomplete dominance was found for methomyl against the TBW [Table 1). There were no significant differences between the crosses. LD50s were equal and intermediate to the parental strains (Roush and Wolfenbarger 1985). Dominance was indicated when LD50s of both reciprocal crosses and the R strain male were equal for methomyl [Wolfenbarger and Wolfenbarger 1999) (Table 1). The LD50 of methomyl by the R strain of Roush and Wolfenbarger (1985) was 1,000+ fold greater than the LD50 of the R strain of Wolfenbarger and Wolfenbarger (1999). In the United States of America and Mexico LD50s of methomyl for the R strains differed 287 fold from 1966 to 1983 while LD50s of the two S strains differed 143 fold (Wolfenbarger et al. 1987).

R and S parental strains and reciprocal crosses showed a clear sex-linked incompletely dominant pattern for profenofos (Ibrahim and Ottea 2001) (Table 1). The discussion of this pattern is somewhat confusing (Ibrahim and Ottea 2001). The authors relied on calculations of dominance relations of the genes rather than how sex-linked genes are passed to progeny. We believe that no additive autosomal locus was involved in their crosses.

The inheritance of resistance to monocrotophos was determined for two R strains (Wolfenbarger 1980) from southern Tamaulipas, Mexico in 1969 and 1971 (Table 1). Both R strains were crossed with the same S strain. One pattern showed sex-linked incomplete dominance. The other showed sex-linked dominant inheritance. One of the strains (Mante 1971) was almost 15X more resistant than the other strain which could account for the difference in dominance relationships for these experiments as well as those cited for methomyl (Bourguet, et al. 2000).

Three completely different inheritance patterns for methyl parathion were found in different inheritance experiments (Table 1). One strain, which had been selected for resistance to methyl parathion, showed incomplete dominance with sex-linkage [Wolfenbarger 1982]. A second strain (Estacion Cuauhtemoc) showed dominant sex-linkage and a third strain (Mante) was incompletely dominant (Wolfenbarger et al. 1997).

EPN showed an incomplete dominance inheritance pattern for a strain from Estacion Cuauhtemoc (Wolfenbarger 1997)(Table 1). LD50s of reciprocal crosses were combined because they were similar and differed from R and S strain by 3.2 and 2.6 fold, respectively.

All of the inheritance patterns for cypermethrin were autosomal recessive (Table 1). The inheritance patterns for permethrin were 80% incompletely dominant and 20% dominant. It is interesting that these similar insecticides would show such contrasting inheritance patterns. Patterns were determined by Payne et al. (1988), Watson and Kelly (1991), Firko and Wolfenbarger (1991), Ibrahim and Otea (2001) and Wolfenbarger (2001).

Although Firko and Wolfenbarger (1991) concluded that dominant effects were present, the inheritance patterns for cypermethrin demonstrated clear recessive inheritance. The data of Ibriham and Otea (2001) were inconclusive for inheritance of resistance by cypermethrin, but data were indicative of recessive inheritance.

The inheritance test for permethrin (Payne et al. 1988) is probably the most comprehensive and conclusive of any presented here. It should be noted that Payne et al. (1988) suggested that the resistance trait was inherited as a partially recessive character. We have chosen to call the inheritance incomplete dominance for consistency in our review. The difference between partially recessive and incompletely dominant is really a matter of interpretation of the placement of response curves.

The inheritance pattern for a strain of Bt toxin was incompletely dominant [Sims and Stone 1991) (Table 1). Significant differences were shown between the reciprocal crosses and both parents.

A pattern of inheritance of incompletely dominance for resistance was shown for monocrotophos, methomyl, methyl parathion, permethrin and Bt toxin. The inheritance of resistance appeared to be consistent regardless of the mode of action of these toxicants. This observation does not mean we believe the same loci are involved, but that the development of insecticide resistance may follow similar patterns over time.

Homozygosity and heterozygosity for response to anticholinesterase, pyrethroid insecticides and Bt toxins have not been defined for these strains of this insect. They need to be defined for all strains. Steepness of slope may be used to define homozygosity (Whitten 1978). Both Whitten (1978) and Watson and Kelly (1991) define homozygosity of S strains with slope values of >3.44 for methyl parathion and permethrin, respectively. Ibrahim and Otea (2001) showed slopes >3.44 for both S and R strains when treated with profenofos. Neither cross showed slopes indicating homozygosity in this test. This is interesting because the crosses showed heterozygous slopes when both parents showed homozygous slopes. For cypermethrin the reverse was true; slopes were heterozygous for the parents, but they were homozygous for the crosses (Ibrahim and Otea 2001). Of the 19 inheritance patterns only 11 % showed regressions which were homozygous.

Bourguet et al. (2000) has discussed the relationships of the ways that dominance levels of the alleles that affect insecticide resistance vary in insects. One important point made by them is that dominance describes the relationship between the phenotypes of three genotypes, which may vary between traits and environments.

The method of Bourguet et al. (2000) depends upon the position of the mortality curve for the heterozygous individuals (F1 progeny) relative to the homozygous individuals at a given mortality level (labeled as DLC in Bourguet et al. 2000). The actual dominance ratios for the different experiments were not calculated because of the variability of the types of populations which were used in them. This variability in parental strains is reflective of the variability of insecticide resistance in the field. Any given population of TBW has probably been exposed to multiple combinations of insecticides, some of which are applied to control the TBW population and some of which are used to control other insects. The effects of these mixtures of chemicals on the genetics of any particular population cannot be determined.

Because of this variability of exposure in time, place and environment, (and the genetic structure of the population), it is almost impossible to predict what the response of a population to continued exposure to a given insecticide might be. We measured the distribution of the inheritance patterns. Incomplete dominance is the most common pattern for all of the insecticides examined. If one considers the way populations are exposed and selected for insecticide resistance, this conclusion is not surprising. These genotypes may vary because of location and environment (Bourguet et al. 2000). Dominance may also be modified because of alleles at linked and unlinked loci. Modifiers may be generalist, affecting several dominant traits simultaneously, or specific, affecting the dominance level of one trait.

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Table 1. Inheritance of resistance patterns of organophosphorus and pyrethroid insecticides and transgenic *Bacillus thuringiensis* in first generation of crosses of strains of the tobacco budworm. 1982-2001.

Difference between parents	Inheritance patterns in first generation			
	Sex-linked[1]	Incomplete dominance	Dominant	Recessive
		Profenofos		
		Ibrahim and Ottea (2001)		
yes	yes	yes	no	no
		Methomyl		
		Roush and Wolfenbarger (1985)		
yes	no	yes	no	no
yes	no	yes	no	no
		Wolfenbarger and Wolfenbarger (1998)		
yes	no	no	yes	no
		Monocrotophos		
		Wolfenbarger (1980)		
yes	yes	no	yes	no
yes	yes	yes	no	no
		Methyl parathion		
		Wolfenbarger et. al. (1982)		
yes	no	yes	no	no
		Wolfenbarger (1997)		
yes	yes	no	yes	no
yes	no	yes	no	no
		EPN		
yes	no	yes	no	no
		Cypermethrin		
		Firko and Wolfenbarger (1991)		
yes	no	no	no	yes
yes	no	no	no	yes
		Ibrahim and Ottea (2001)		
no	no	no	no	yes
		Permethrin		
		Watson and Kelly (1991)		
yes	no	yes	no	no
		Payne et al. (1988)		
yes	no	yes	no	no
yes	no	yes	no	no
		Wolfenbarger (2001)		
no	no	no	yes	no
yes	no	yes	no	no
		Transgenic *Bacillus thuringiensis*		
		Sims and Stone [1999]		
yes	no	yes	no	no

[1]If a trait is not sex-linked autosomal inheritance is suggested.