

# Inheritance of Resistance to Phenthoate and Fenvalerate in Diamondback Moth and Management of Insecticide Resistance

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## Abstract

The mode of inheritance of phenthoate and fenvalerate resistance in diamondback moth (*Plutella xylostella* (L.)) was incomplete dominant and incomplete recessive, respectively. Neither phenthoate resistance nor fenvalerate resistance was sex linked. Phenthoate and fenvalerate resistance was influenced by polygenic backgrounds. From results of mechanisms of phenthoate and fenvalerate resistance in diamondback moth, the management of resistance to insecticides is discussed. To retard or avoid the development of insecticide resistance, reduction of the selection pressure by the same insecticide is important. The rotational use of insecticides which do not show cross-resistance will be effective. The introduction of a combination of insecticides with negatively correlated cross-resistance will also be effective.

## Introduction

Although resistance to various organic insecticides in the diamondback moth (DBM), *Plutella xylostella* (L.) (Lepidoptera: Yponomeutidae) has been extensively documented (Sudderuddin and Kok 1978; Miyata et al. 1986). Little is known about the resistance mechanism and genetic basis for resistance (Liu et al. 1981; Hama 1989; Tanaka and Noppun 1989). Liu et al. (1981) and Hama (1989) found that the inheritance of fenvalerate resistance was controlled by autosomal incomplete recessive gene(s), and it was influenced by some polygenic backgrounds.

Noppun et al. (1986a, 1987b) obtained high levels of resistance to phenthoate and fenvalerate in DBM by successive selections in the laboratory. They found that phenthoate resistance in DBM is based on at least two mechanisms: (1) an efficient system of reduced cuticular permeability (Noppun et al. 1987e), and (2) an increased insensitivity of acetylcholinesterase (Noppun et al. 1987b). They found also that fenvalerate resistance is based on at least three mechanisms: (1) an efficient system of reduced cuticular permeability (Noppun et al. 1989b), (2) increased degradation of fenvalerate (Noppun et al. 1986b), and (3) an increased insensitivity of the nervous system (Noppun et al. 1986b). Therefore genetic studies were conducted to determine the mode of inheritance of phenthoate and fenvalerate resistance in DBM. From data obtained in this study and data obtained previously by us, we will discuss ways to manage the development of insecticide resistance in DBM.

## Materials and Methods

### Insects strains and maintenance

An experiment was conducted with two phenthoate-resistant (OKR-R and OSS-R), two phenthoate-susceptible (OKR-S and OSS-S), two fenvalerate-resistant (OKR-FR and KAR-FR) and two fenvalerate-susceptible (OKR and KAR) strains.

Phenthoate-resistant strains were those previously selected for resistance with phenthoate from the susceptible OKR and OSS strains, respectively (Noppun et al. 1986a). Two phenthoate-susceptible strains were selected for susceptibility with phenthoate from the susceptible OKR and OSS strains, respectively (Noppun et al. 1987d). Two fenvalerate-susceptible (OKR and KAR) strains which have been maintained without exposure to insecticides after collection were used for selection in the laboratory (Noppun et al. 1987c). Fenvalerate-resistant strains were those previously selected for resistance with fenvalerate from OKR and KAR strains (Noppun et al. 1987c).

The insects were mass-reared and maintained in a controlled room (25% RH, 16L:8D) as previously described (Noppun et al. 1983). The third instar larvae weighing an average 2.55 mg from the population were used for toxicity experiments.

### Continued selection

The phenthoate-resistant (OKR-R and OSS-R) strains were further selected for homogeneous resistance to phenthoate by the method reported by Noppun et al. (1986a) for the successive 12 generations. In this selection 6 ml of phenthoate solution was used instead of 3 ml in the previous selection (Noppun et al. 1986a) because spraying with 3 ml of phenthoate solution did not result in more than 50% mortality at 24 hours. The phenthoate-susceptible (OKR-S and OSS-S) strains were also selected to obtain more homogeneous susceptibility to phenthoate by the method reported by Noppun et al. (1987d) for the successive three generations.

### Genetic study

F<sub>1</sub> crosses were made in each strain by mass-mating of more than 100 adults of each sex according to the standard method reported by Georghiou (1969). To ensure the use of virgin insects, each pupa was isolated for eclosion individually in a test tube.

The degree of dominance (D) of resistance in F<sub>1</sub> offspring was calculated by Falconer's formula (1964) according to Stone (1968):

$$D = \frac{2LD_{50}(RS) - LD_{50}(RR) - LD_{50}(SS)}{LD_{50}(RR) - LD_{50}(SS)}$$

where RR, RS and SS represent the resistant, heterozygote and susceptible populations, respectively.

Males and females of F<sub>1</sub> progeny were backcrossed to resistant (RR) females and to susceptible (SS) males, respectively. F<sub>1</sub> progeny were also allowed to intercross with F<sub>2</sub> progeny. The expected dose-response curve of the backcross progeny, assuming monofactorial inheritance, was calculated as followed (Georghiou 1969):

- (1) for backcross progeny to SS or RR parents:

$$X_y = W_{(SR)}0.50 W_{(SS \text{ or } RR)}0.50$$

- (2) for F<sub>2</sub> progeny:

$$X_y = W_{(SS)}0.25 W_{(SR)} 0.50 W_{(RR)}0.25$$

where X = the expected response at a given concentration y; and W = the observed response of SS, SR and RR genotypes at concentration y, obtained directly from the respective regression lines.

The single-gene hypothesis was tested by exposing the F<sub>2</sub> and the backcross progeny to a full range of concentrations of phenthoate and fenvalerate, respectively. If a single gene is responsible for insecticide resistance then plateaus will occur in the F<sub>2</sub> regression line at about 25 or 75% mortality. Goodness of fit of observed to expected mortalities was tested by Chi-square analysis and the *t* distribution at 95% confidence limits.

### Toxicity tests

The third instar larvae were topically applied with 0.52 µl of acetone solution of insecticide. The details of a standard method are given in Noppun et al. (1983). However, for the genetic study 20 larvae were used at one dose level with three replications, instead of 10 larvae in the standard method. At least nine dose levels of insecticide were employed and mortalities at 24 hours after topical application were taken and subjected to probit analysis (Finney 1971) using a microcomputer (NEC PC-9801VX, NEC Co. Ltd., Tokyo).

## Results and Discussion

### Continued selection

The continued selections of the phenthoate-resistant (OKR-R and OSS-R) strains for 12 generations resulted in a further increase in resistance levels. After the last selection, the OKR-R and the OSS-R strains showed higher LD<sub>50</sub> values to phenthoate (48,400 and 44,800 µg/g) than those reported previously (16,200 and 13,600 µg/g) (Noppun et al. 1986a), respectively. On the other hand, continued selection of the phenthoate-susceptible (OKR-S and OSS-S) strains (Noppun et al. 1987d) for more susceptibility with phenthoate for three generations did not give a further increase in phenthoate susceptibility.

### Genetic study

Results of toxicity tests with phenthoate against the parental and hybrid populations are given in Tables 1 and 2. Resistance to phenthoate of the F<sub>1</sub> progenies of OKR-R × OKR-S and OKR-R × OKR-S crosses had fallen in the mid-parent range (D of LD<sub>50</sub> and LD<sub>95</sub> values = 0.15 and 0.15, and 0.47 and 0.55, respectively). Similarly, resistance of the F<sub>1</sub> progenies of OSS-R × OSS-S and OSS-R × OSS-S crosses to phenthoate was also in the mid-parent range (D of LD<sub>50</sub> and LD<sub>95</sub> values = 0.15 and 0.11, and 0.50 and 0.32, respectively). When

Table 1. Susceptibility to phenthoate in the phenthoate-resistant (OKR-R) strain, the phenthoate-susceptible (OKR-S) strain and various interstrain crosses.

Strains	SRE <sup>a</sup>	LD <sub>50</sub> (µg/g)	RF of <sup>b</sup> LD <sub>50</sub>	D of <sup>c</sup> LD <sub>50</sub>	LD <sub>95</sub> (µg/g)	RF of <sup>d</sup> LD <sub>95</sub>	D of <sup>e</sup> LD <sub>95</sub>
OKR-R (R)	1.95	48400	1870	—	338000	2000	—
F11 (R × S)	1.21	1950	75.3	0.15	44500	265	0.47
F12 (R × S)	1.10	1980	76.5	0.15	61100	364	0.55
F12 × R	1.11	14800	571	—	448000	2660	—
F12 × S	0.87	490	18.9	—	38200	227	—
Intercross F11	0.84	1290	49.8	—	116000	690	—
Intercross F12	1.20	1070	41.3	—	25300	151	—
OKR-S (S)	2.03	25.9	1.0	—	163	1.0	—

<sup>a</sup>Slope of regression equation. <sup>b</sup>Resistance factor of LD<sub>50</sub> = LD<sub>50</sub> of RR or RS population/LD<sub>50</sub> of SS population. <sup>c</sup>Degree of dominance of LD<sub>50</sub>. <sup>d</sup>Resistance factor of LD<sub>95</sub> = LD<sub>95</sub> of RR or RS population/LD<sub>95</sub> of SS population. <sup>e</sup>Degree of dominance of LD<sub>95</sub>.

Table 2. Susceptibility to phenthoate of the phenthoate-resistant (OSS-R) strain, the phenthoate-susceptible (OSS-S) strain and various interstrain crosses.

Strains	SRE <sup>a</sup>	LD <sub>50</sub> ( $\mu$ g/g)	RF of <sup>b</sup> LD <sub>50</sub>	D of <sup>c</sup> LD <sub>50</sub>	LD <sub>95</sub> ( $\mu$ g/g)	RF of <sup>d</sup> LD <sub>95</sub>	D of <sup>e</sup> LD <sub>95</sub>
OSS-R(R)	1.96	44800	1200	—	309000	1410	—
F <sub>11</sub> (R $\sigma$ $\times$ S $\phi$ )	1.21	2180	58.3	0.15	49400	226	0.50
F <sub>12</sub> (R $\phi$ $\times$ S $\sigma$ )	1.43	1920	51.3	0.11	26900	123	0.32
F <sub>12</sub> $\sigma$ $\times$ R $\phi$	1.35	9680	259	—	161000	735	—
F <sub>12</sub> $\phi$ $\times$ S $\sigma$	1.10	382	10.2	—	11800	53.9	—
IntercrossF <sub>11</sub>	0.96	1670	44.7	—	87100	398	—
IntercrossF <sub>12</sub>	0.98	1340	35.8	—	62900	287	—
OSS-S(S)	2.14	37.8	1.0	—	219	1.0	—

Footnotes as in Table 1.

D values are compared between two different crossings in each strain, the difference is small. Results from the present study revealed that inheritance of resistance to phenthoate in DBM was derived by more than one gene, incomplete dominant genes and no sex linkage. From studies on the mechanism of phenthoate resistance in DBM, it was also found that reduced cuticular penetration and reduced sensitivity of acetylcholinesterase are involved in the mechanism of phenthoate resistance (Noppun et al. 1987b, e).

The observed dose-response curves of the backcross progeny (F<sub>12</sub>(R $\phi$   $\times$  S $\sigma$ )) $\sigma$   $\times$  S $\phi$ ), (F<sub>12</sub> (R $\phi$   $\times$  S $\sigma$ ) $\phi$   $\times$  S $\sigma$ ) and intercrosses of F<sub>11</sub> and F<sub>12</sub> progenies differ significantly from the curves which are expected on the basis of monofactorial inheritance. Computation of the 95% confidence limits confirms the significant differences between the observed and expected curves except for a small region near LD<sub>50</sub> (data are not shown). It is thus concluded that phenthoate resistance in DBM is influenced by polygenic backgrounds.

Results of toxicity tests with fenvalerate against the parental and hybrid populations are given in Tables 3 and 4. Resistance of the F<sub>1</sub> progenies of OKR-FR $\sigma$   $\times$  OKR $\phi$  and OKR-FR $\phi$   $\times$  OKR $\sigma$  crosses to fenvalerate had fallen in the mid-parent range (D of LD<sub>50</sub> and LD<sub>95</sub> values = -0.50 and -0.17, and -0.53 and -0.07, respectively). Similarly, resistance of the F<sub>1</sub> progenies of KAR-FR $\sigma$   $\times$  KAR $\phi$  and KAR-FR $\phi$   $\times$  KAR $\sigma$  crosses to fenvalerate was also in the mid-parent range (D or LD<sub>50</sub> and LD<sub>95</sub> values = -0.53 and -0.39, and -0.44 and -0.41, respectively). D values seemed to be different between two crossings in OKR-FR and OKR strains. Since the difference in the slope of regression equations is statistically nonsignificant, it is therefore concluded that there is no sex-linkage in the inheritance of fenvalerate resistance.

The observed dose-response curves of the progeny of the backcrosses, (F<sub>11</sub>(R $\sigma$   $\times$  S $\phi$ ) $\sigma$   $\times$  S $\phi$ ), (F<sub>11</sub>(R $\sigma$   $\times$  S $\phi$ ) $\phi$   $\times$  R $\sigma$ ), (F<sub>12</sub>(R $\phi$   $\times$  S $\sigma$ ) $\sigma$   $\times$  S $\phi$ ) and (F<sub>12</sub>(R $\phi$   $\times$  S $\sigma$ ) $\phi$   $\times$  R $\sigma$ ) and intercrosses of F<sub>11</sub> and F<sub>12</sub> progenies differ significantly from the curve which might be expected on the basis of monofactorial inheritance (data are not shown). Computation of the 95% confidence limits confirms the significance of differences between the observed and expected curves except for a small region near the LD<sub>50</sub>.

It is thus concluded that fenvalerate resistance in DBM is due to more than one gene. Liu et al. (1981) and Hama (1989) also reported that fenvalerate resistance is inherited through partially recessive genes with no sex linkage. According to Noppun et al. (1986b, 1989a, b), fenvalerate resistance is controlled by at least three different mechanisms: (1) increased metabolism (esterase and mixed function oxidases), (2) reduced cuticular permeability, and (3) reduced sensitivity of the central nervous system.

## Management of insecticide resistance

Yamada (1977) pointed to the following reasons for the increase in DBM damage: (1) year-round cultivation of crucifers, especially cabbage which is an excellent host plant; (2) increase

Table 3. Susceptibility to fenvalerate of the fenvalerate-resistant strain (OKR-FR), the fenvalerate-susceptible strain (OKR) and various interstrain crosses.

Strains	SRE <sup>a</sup>	LD <sub>50</sub> ( $\mu\text{g/g}$ )	RF of <sup>b</sup> LD <sub>50</sub>	D of <sup>c</sup> LD <sub>50</sub>	LD <sub>95</sub> ( $\mu\text{g/g}$ )	RF of <sup>d</sup> LD <sub>95</sub>	D of <sup>e</sup> LD <sub>95</sub>
OKR-FR (R)	0.92	877	1620	—	53500	16200	—
F <sub>11</sub> (R $\times$ S )	1.71	3.47	6.43	-0.50	31.9	9.67	-0.53
F <sub>12</sub> (R $\times$ S )	1.16	11.3	21.7	-0.17	305	92.4	-0.7
F <sub>11</sub> $\times$ R	0.64	9.18	17.0	—	3480	1050	—
F <sub>11</sub> $\times$ S	2.38	0.787	1.46	—	3.86	1.17	—
F <sub>12</sub> $\times$ R	0.57	15.1	28.0	—	12200	3700	—
F <sub>12</sub> $\times$ S	2.45	0.966	1.79	—	4.54	1.38	—
Intercross F <sub>11</sub>	0.87	0.013	0.02	—	0.774	0.23	—
Intercross F <sub>12</sub>	1.01	0.014	0.08	—	1.73	0.52	—
OKR(S)	2.10	0.54	1.0	—	3.30	1.0	—

Footnotes as in Table 1.

Table 4. Susceptibility to fenvalerate of the fenvalerate-resistant strain (KAR-FR), the fenvalerate-susceptible strain (KAR) and various interstrain crosses.

Strains	SRE <sup>a</sup>	LD <sub>50</sub> ( $\mu\text{g/g}$ )	RF of <sup>b</sup> LD <sub>50</sub>	D of <sup>c</sup> LD <sub>50</sub>	LD <sub>95</sub> ( $\mu\text{g/g}$ )	RF of <sup>d</sup> LD <sub>95</sub>	D of <sup>e</sup> LD <sub>95</sub>
KAR-FR (R)	0.61	3330	5840	—	192000	53000	—
F <sub>11</sub> (R $\times$ S )	1.33	4.36	7.65	-0.53	75.2	20.8	-0.44
F <sub>12</sub> (R $\times$ S )	1.57	7.93	13.9	-0.39	88.1	24.3	-0.41
F <sub>11</sub> $\times$ R	0.53	45.2	79.3	—	59600	16500	—
F <sub>11</sub> $\times$ S	2.35	0.944	1.66	—	4.73	1.31	—
F <sub>12</sub> $\times$ R	0.46	84.3	148	—	146000	40300	—
F <sub>12</sub> $\times$ S	2.24	0.891	1.56	—	4.84	1.34	—
Intercross F <sub>11</sub>	1.10	0.031	0.05	—	0.954	0.26	—
Intercross F <sub>12</sub>	1.38	0.081	0.14	—	1.00	0.28	—
KAR (S)	2.04	0.57	1.0	—	3.62	1.0	—

Footnotes as in Table 1.

in the area of cabbage cultivation; and (3) insecticide resistance caused by frequent application of insecticides, especially in tropical countries where the number of DBM generations is more than 20 (Ho 1965; Sun et al. 1978).

To manage the insecticide resistance, one must (1) manage cultivation of crucifer crops; (2) introduce control methods that do not use insecticides to reduce the insecticide pressure; and (3) reevaluate conventional control methods that use insecticides. In this paper, only the third point will be discussed.

There are two ways to overcome the problem of development of insecticide resistance: (1) avoid or retard the development of insecticide resistance, and (2) control the insecticide-resistant DBM. The results of studies on the mechanisms of resistance to phenthoate and fenvalerate in DBM are given in Table 5. From these data, the rotational use of insecticides that have no cross-resistance will be important to reduce the selection pressure by the same insecticide. It would certainly be preferable to increase the time interval between applications of the same insecticide. The introduction of insecticide combinations that have negatively correlated cross-resistance will be effective, as reported in the rice-leaf and planthoppers by Miyata and Saito (1984). In DBM, chitin synthetase inhibitors seem to show negatively correlated cross-resistance with juvenile hormone analogs (Sinchaisri et al. 1990) as reported in *Spodoptera littoralis* (El-Guindy et al. 1983). Introduction of a new type of insecticide or the use of a synergist is effective to overcome insecticide resistance, however, more than two different resistance mechanisms are involved

in the resistance (Table 5), and it is not easy to increase the susceptibility of the resistant strain to the level of the susceptible one with a synergist. If they are used extensively, DBM can develop resistance to them (Sun et al. 1986; Takeda et al. 1986).

According to the simulation study by Tabashnik (1986), DBM develops resistance to insecticides in a short period, so it is important to extend the application interval of the same insecticide as much as possible.

The insecticide resistance level is reported to decrease after relaxation of insecticidal pressure (Noppun et al. 1984; Sun et al. 1986; Hama 1989). Possible explanations for the phenomenon include: (1) some genetic variance in insecticide resistance among individuals, (2) some genetic variance of reproductive ability among individuals; and (3) a possible negative correlation between insecticide resistance and reproductive ability of an individual (Tsubaki et al. 1988). Development of simple monitoring methods for resistance is also necessary, especially methods to detect individual resistance levels (Miyata 1983; Miyata and Saito 1984; Miyata 1989).

Table 5. The mode of resistance to phenthoate and fenvalerate in DBM.

Phenthoate resistance	Fenvalerate resistance
Reduced cuticular penetration	Reduced cuticular penetration Increased metabolism
Increased sensitivity of AChE	Reduced sensitivity of CNS
High cross-resistance to: prothiophos, cyanophos and methomyl	High cross-resistance to: pyrethroids
Low cross-resistance to: dichlorvos and cartap	Cross-resistance to: phenthoate, prothiophos, cyanophos and methomyl
No cross-resistance to: acephate and fenvalerate	No cross-resistance to: cartap
Synergism with TPP	Synergism with TPP and PB
Unstable resistance	Unstable resistance

(Source: Noppun et al. 1984, 1986b, 1987a, 1987b, 1987e, 1989a, 1989b).

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