Studies on the Mechanism of Diamondback Moth Resistance to Insecticides

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Abstract

Since the first report of insecticide resistance in diamondback moth, *Plutella xylostella* L, to DDT in 1953, the number of cases of insecticide resistance in *P. xylostella* has increased. Currently this insect shows resistance to more than 46 insecticides, including some synthetic pyrethroids. The cross resistance spectrum is different between populations and insecticide resistance selections. DDT resistance is considered to be controlled by a non-metabolic mechanism. Synthetic pyrethroids resistance seems to be mediated partly by metabolic and partly by non-metabolic mechanisms. Organophosphorus insecticide resistance also seemed to be mediated in part by metabolic (increased metabolism by carboxylesterase and glutathione-S-transferase) and non-metabolic (reduced susceptibility of cholinesterase) mechanisms. It is important not only to control resistant *P. xylostella* strains but also to retard the development of resistance.

Introduction

Resistance to insecticides is an evolutionary phenomenon brought about by intensive 'natural selection' of the insect pest after continuous massive applications of insecticides. Resistance is not limited to insects and insecticides. A range of other organisms, including rodents, fungi, and weeds develop resistance to chemicals intended to control them. The number of species of arthropoda resistant to insecticides has incresed almost linearly from 1 in 1908, 5 in 1928, 14 in 1948, 76 in 1957, 224 in 1967, 364 in 1975 and 432 in 1980 (Georghiou and Taylor 1977, Georghiou and Mellon 1980).

The diamondback moth (DBM), *Plutella xylostella* L (Lepidoptera: Yponomeutidae), is a cosmopolitan species of considerable importance as a pest of cruciferous plants. DBM has 14 to 28 generations in Malaysia (Ho 1965), 15 to 20 generations in Taiwan (Sun et al 1978) and 5 to 12 generations in Japan (Umeya and Yamada 1973) each year with overlapping of all developmental stages. DBM damage has become serious in Japan since 1960. Yamada (1977) pointed to the following reasons for this increase in damage: (1) year-round cultivation of crucifers, especially cabbage, which is an excellent host plant; (2) increase of the cabbage cultivation area; and (3) insecticide resistance caused by frequent application of insecticides. Recently Nemoto et al (1984) reported that methomyl might cause a resurgence in DBM populations through the stimulation of the reproductive potential. These factors will also explain the increasing problem of DBM in other countries.

Development of Insecticide Resistance in DBM

The first well documented case of insecticide resistance in DBM was demonstrated in Java, Indonesia, in 1953 by Ankersmit (1953). He observed that insects have developed

over time as much as seven times the level of resistance to DDT as they had exhibited when the compound was first used. Since then numerous cases of resistance to various kinds of insecticides have been reported from the Philippines (Barroga and Morallo-Rejesus 1974, 1981, Morallo-Rejesus and Eroles 1976), Japan (Asakawa 1975, Tokairin and Nomura 1975, Miyata et al 1982, Noppun et al 1983, Hama 1984), Malaysia (Lim 1974, Sudderudin and Kok 1978, Teh et al 1982), Taiwan (Sun et al 1978, Lee and Lee 1978, Liu et al 1981, 1982b, Chou and Cheng 1983), Thailand (Sinchaisri et al 1980), Singapore (Georghiou 1984) and so on. Some of the resistance patterns of DBM are shown in Tables 1 and 2. All these reports have indicated that DBM has developed resistance to various types of insecticides, including recently introduced synthetic pyrethroids.

Table 1. Susceptibility to some insecticides of susceptible, slightly resistant (Peng-hu), and highly resistant (Ban-chau) strains of DBM in Taiwan[a]

Insecticide	Susceptible	Peng-hu	Ban-chau
	LC_{50} (mg/ml)	R.R[b].	R.R.
Malathion	0.0274	536	>3,650
Diazinon	0.0293	43	413
Methyl parathion	0.00472	10,508	>21,000
Dichlorvos	0.0168	43	300
Cyanofenphos	0.00196	74	>50,000
Prothiophos	0.00041	400	5,854
Carbaryl	0.432	23	>230
Propoxur	0.338	>44	>300
Methomyl	0.0507	69	1,049
Permethrin	0.000776	4	110
Cypermethrin	0.00104	21	894
Deltamethrin	0.0002	56	2,235
Fenvalerate	0.000382	33	2,880
DDT	0.0348	635	>2,870
Cartap	0.025	16	199

[a] Source: Liu et al 1982b. Insecticides were sprayed on 4th instar larvae. [b] R.R.: resistance ratio.

According to Georghiou (1981), the cases of DBM resistance to insecticides had increased to 36 insecticides and 14 countries by 1980 (Table 3). Hama (1983) compared the susceptibility of the field-collected Miinohara strain and the susceptible strain (a strain reared in laboratory without exposure to insecticides) against 26 insecticides. Fifteen insecticides showed more than 20 fold resistance (Table 2).

Resistance stability and selection for resistance

Noppun et al (1984b) reported a decrease of insecticide resistance in DBM after withdrawal of chemicals. Strains collected in Okinawa and Aichi showed high levels of resistance to phenthoate, prothiophos, and cyanophenphos, and a moderate level of resistance to acephate, methomyl, and cartap when tested after 12 months and 5 months of laboratory rearing following field collection, respectively (Noppun et al 1983). However, there were no significant differences between resistance levels among field-collected strains and the susceptible strain after another 12 months laboratory rearing without insecticide selection (Noppun et al 1984c). A similar phenomenon was also observed by Ankermit (1953). The susceptibility of a DDT-resistant DBM strain to DDT increased at the eighth generation of laboratory rearing. Sun et al (1978) observed that the susceptibility of field-collected DBM to diazinon increased about three fold after

Table 2. Toxicity of various insecticides to susceptible and resistant (Miinohara) strains of DBM in Japan

Insecticides	LD_{50} (ug/larva)		Resistance ratio
	Susceptible	Resistant	
Fenvalerate	0.0073	0.0093	1.3
Phenothrin	0.030	0.034	1.1
Cyanofenphos	0.031	29.0	936.0
Dimethylvinphos	0.046	1.9	41.0
Methidathion	0.068	1.6	24.0
Profenofos	0.073	5.6	77.0
Prothiophos	0.089	24.0	270.0
Cyanophos	0.10	9.6	96.0
Phenthoate	0.13	8.3	64.0
Isoxanthion	0.14	>45.0	>321.0
Cartap	0.16	1.2	7.5
Thiocyclam	0.19	0.39	2.1
Salithion	0.43	11.0	26.0
Pirimiphos-methyl	0.48	8.7	18.0
Dialifor	0.71	>45.0	>63.0
Dichlorvos	0.73	9.6	13.0
Methomyl	0.86	>45.0	>52.0
Chlorvinphos	1.1	7.7	7.0
Chlorpyrifos-methyl	1.3	>45.0	>35.0
EPN	1.5	37.0	25.0
Diazinon	1.6	>45.0	>26.0
Fenitrothion	1.6	>45.0	>28.0
Acephate	1.7	>4.5	>2.6
Chlorpyrifos	2.3	>4.5	>2.0
Dimethoate	2.4	—	—
Trichlorfon	13.0	>45.0	>3.5
Malathion	20.0	>45.0	>2.3
BPMC	4.5	—	—
Carbaryl	10.0	—	—
Oxyphinofos	45.0	—	—

[a] Source: Hama 1983. Insecticides were topically applied to 4th instar larvae.

having been reared for 14 generations in the laboratory without insecticides. Lee and Lee (1979) also reported that susceptibility levels of DBM to malathion, dichlorvos, diazinon, phenthoate, mevinphos, and endosulfan increased three to seven fold after laboratory rearing for 20 generations. The loss of insecticide resistance during laboratory rearing causes some difficulties in insecticide resistance studies of DBM. On the other hand, Hama (1983) mentioned that the Miinohara strain did not show any significant loss of resistance (except to a few insecticides) after laboratory rearing for more than 15 generations.

Noppun et al (1984c) selected the Osaka susceptible strain and the Okinawa strain which lost resistance to phenthoate. High levels of resistance to phenthoate were obtained after selection repeated eight times during nine generations (Figure 1). At LD_{50} and LD_{95} levels, the phenthoate-selected Okinawa strain exhibited 172 and 287 fold resistance, while the Osaka susceptible strain exhibited 194 and 289 fold resistance, respectively. No significant difference in the rate of development of phenthoate resistance between the two selected strains was observed.

Sun et al (1978) selected the field-collected DBM strain with diazinon and methomyl pressure. Strains with resistance ratios of 14.4 and 17.5 to diazinon and methomyl

Table 3. Geographical distribution of the occurrence of insecticide resistance in DBM[a]

Insecticide group	Country, area	Insecticide group	Country, area
DDT	Barbados, Indonesia, Malaysia, Philippines, Singapore, South Africa, Sri Lanka, Taiwan, Vietnam	Malathion	Antigua, Barbados, Jamaica Malaysia, Philippines, South Africa, Taiwan, Vietnam
		Mevinphos	Philippines, Singapore
BHC/cyclodienes	Venezuela	Monocrotophos	Vietnam
Aldrin/dieldrin	Vietnam	Naled	Vietnam
Endosulfan	South Africa	Parathion	Sri Lanka, Taiwan
Endrin	Sri Lanka, Vietnam	Phenthoate	Japan, Vietnam
Isobenzan	Malaysia	Phosphamidon	Vietnam
Lindane/BHC	Malaysia, Singapore, Sri Lanka	Prothiophos	Taiwan
Acephate	Japan	Trichlorfon	Japan, Malaysia
Chlorpyrifos-methyl	Malaysia	Carbaryl	Barbados, Malaysia, Taiwan, Vietnam
Cyanophos	Taiwan	Isoprocarb	Taiwan
Diazinon	Japan, Philippines, Taiwan, Vietnam	Methomyl	Barbados, Japan, Malaysia, Taiwan
Dichlorvos	Japan, Malaysia, Philippines, Taiwan, Vietnam	Propoxur	Taiwan
		Pyrethroids	Malaysia, Thailand
Dimethoate	Barbados	Cypermethrin	Taiwan
EPN	Japan	Deltamethrin	Taiwan
Fenitrothion	Vietnam	Fenvalerate	Taiwan
Leptophos	Malaysia, Vietnam	Permethrin	Taiwan
Methamidophos	Malaysia	Resmethrin	Malaysia
Methyl parathion	Philippines, Taiwan, Vietnam	Cartap	Malaysia, Taiwan

[a] Source: Georghiou 1981.

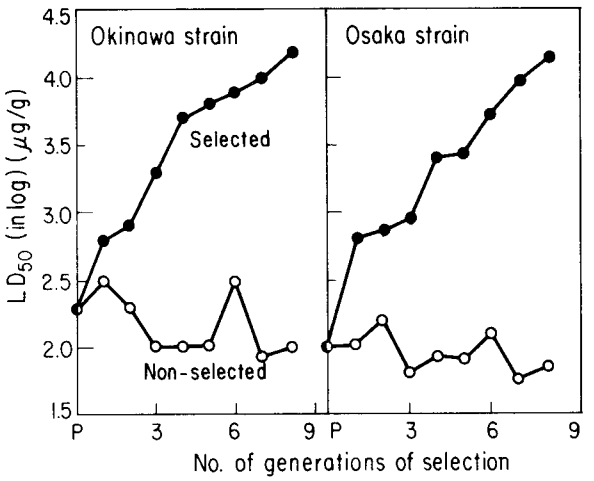

Figure 1.
Changes in LD_{50} values of phenthoate in the phenthoate-selected and non-selected strains from Okinawa and Osaka

respectively were developed in the laboratory by continuous selection for 14 and 19 generations, respectively. Chou and Cheng (1983) also selected the field-collected susceptible IL strain of DBM with carbofuran, fenvalerate, and cartap. The resistance to carbofuran developed 36 fold in eight generations. The other two insecticides showed smaller levels of resistance: 15 fold for fenvalerate in 12 generations and 10 fold for cartap in 10 generations. Noppun et al (1983) selected the field-collected Okinawa strain with fenvalerate 15 times during 22 generations and recorded an 8.7 fold resistance. However, Liu et al (1981, 1982b) reported higher levels of resistance in Taiwan strains of DBM.

Cross Resistance

The term cross resistance denotes the resistance of a strain of insects to compounds other than those they were selected against. The term multiple resistance denotes the resistance of a strain of an insect developed during the simultaneous or consecutive use of several insecticides (Yamasaki 1972). However, Oppenoorth and Welling (1976) state that the term cross ressistance denotes the resistance of a strain of insects to compounds other than those they were selected against, when resistance is due to the same mechanisms. In contrast, they assert that multiple resistance is the resistance of a single strain to several different compounds but resulting from different mechanisms. Multiple resistance often results from the simultaneous or consecutive use of several insecticides. However, it is sometimes difficult to discriminate between cross resistance and multiple resistance, since genetic linkage may result in different cross resistances, particularly during selection in the laboratory. If selection with an insecticide favors a rare gene A, it will simultaneously select the gene-alleles that are linked with it. If another resistance gene B already occurs in the strain, either this gene or its S-allele may be linked with gene A. Selection for gene A can thus increase or decrease gene B, depending on its accidental occurrence, whereas in actuality multiple resistance is present. Therefore, in this paper, we follow Yamasaki's definition (1972).

Sasaki (1982) selected the Osaka susceptible strain of DBM with dichlorvos, prothiophos, and cyanophos for more than 15 generations. He also conducted negative selection and obtained highly susceptible strains for each insecticide selection. Kuwahara (1983) reported that the Osaka susceptible strain was 2.5 to 4.1 times less sensitive than a strain from Uruguay to some insecticides. Cross resistance spectra of each selected

Table 4. Pattern of cross resistance to insecticides in dichlorvos-, prothiophos-, and cyanophos-selected DBM strains[a]

Insecticides	RR[b] of strains selected for resistance to		
	Dichlorvos	Prothiophos	Cyanophos
Dichlorvos	37.0	17.0	11.0
Cyanophos	7.0	158.0	381.0
Dimethylvinphos	1.7	2.3	5.0
Phenthoate	2.4	3.9	2.0
Prothiophos	2.5	98.0	29.0
Cyanofenphos	0.5	57.0	—
Diazinon	0.6	44.0	66.0
Dibrom	26.0	—	—
Isoxanthion	1.5	32.0	37.0
Salithion	12.0	12.0	6.0
Mecabram	13.0	8.0	—
Dialifor	1.3	1.5	0.7
Dimethoate	2.7	6.0	13.0
Fenitrothion	10.0	40.0	—
Tetrachlorvinphos	7.0	6.0	—
Acephate	3.1	1.6	—
Phosalone	1.4	3.9	—
EPN	2.8	4.2	—
Pyridaphenthion	2.5	1.2	—
BPMC	4.1	2.5	—
Pyrethrins	2.1	2.9	—
Fenvalerate	1.4	—	—
Pirimiphos-methyl	—	54.0	53.0

[a] Source: Sasaki 1982. [b] RR: resistance ratio.

strain are shown in Table 4. Each selected strain showed cross resistance to a wide range of insecticides. But cross resistance was lowest in the dichlorvos selected strain.

Liu et al (1981) demonstrated obvious cross resistance to synthetic pyrethroids (permethrin, cypermethrin, deltamethrin and fenvalerate). The methomyl-selected strain showed 3.8 fold resistance to fenvalerate, while this strain showed only 0.5 to 0.2 fold resistance to the other three synthetic pyrethroids. Hama (1983) reported more than 28 fold resistance to diazinon in the Miinohara strain, but this strain showed no resistance to fenvalerate and phenothrin (Table 2).

Chou and Cheng (1983) reported that the carbofuran-selected IL strain of DBM showed very little cross resistance to another 19 insecticides. On the other hand, stronger cross resistance was observed in both cartap-and fenvalerate-selected strains. Cheng et al (1984) also selected the susceptible IL strain with mevinphos for 20 generations and obtained only five to eight fold resistance. The mevinphos-selected strain showed a broad cross resistance to other organophosphorus insecticides and cartap, but this resistance was not extended to most of the synthetic pyrethroids.

On the other hand, Noppun et al (1984a) compared the cross resistance spectra of two DBM strains selected with phenthoate (Table 5). The phenthoate-selected Okinawa

Table 5. Cross resistance spectra of phenthoate-selected DBM strains[a]

Insecticide	Strain	RR[b] based on	
		LD_{50}	LD_{95}
Phenthoate	Selected Okinawa	109.0	116.0
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	166.0	178.0
	Non-selected Osaka	1.0	1.0
Dichlorvos	Selected Okinawa	7.7	9.0
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	7.5	8.3
	Non-selected Osaka	1.0	1.0
Prothiophos	Selected Okinawa	120.0	839.0
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	7.6	8.8
	Non-selected Osaka	1.0	1.0
Cyanophos	Selected Okinawa	95.0	680.0
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	10.9	11.7
	Non-selected Osaka	1.0	1.0
Acephate	Selected Okinawa	3.4	3.8
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	4.5	4.6
	Non-selected Osaka	1.0	1.0
Methomyl	Selected Okinawa	318.0	1380.0
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	130.0	274.0
	Non-selected Osaka	1.0	1.0
Cartap	Selected Okinawa	3.7	14.8
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	2.5	9.3
	Non-selected Osaka	1.0	1.0
Fenvalerate	Selected Okinawa	1.5	1.9
	Non-selected Okinawa	1.0	1.0
	Selected Osaka	1.2	1.7
	Non-selected Osaka	1.0	1.0

[a] Source: Noppun et al 1984a. [b] RR: resistance ratio.

strain showed high cross resistance to prothiophos, cyanophos, and methomyl and showed low cross resistance to dichlorvos and cartap. The phenthoate-selected Osaka strain also showed high cross resistance to methomyl, however, it showed low cross-resistance to dichlorvos, prothiophos, cyanophos, and cartap. This result indicates that even if the selection starts at the same insecticide susceptibility levels, when the strains are different the resultant selected strains do not necessarily show the same cross resistance spectra.

Resistance Mechanism

Susceptible and multiple resistant (Banchou, resistance factor for DDT = 200) strains of DBM were used to determine the effect of a microsomal oxidation inhibitor, piperonyl butoxide (PB), and a DDT-dehydrochlorinase inhibitor, 1,1-bis (p-chlorophenyl) ethanol (DMC). The absence of synergism by PB and DMC raised the possibility of the existence of a non-metabolic mechanism of DDT (Liu et al 1982a). However, against the multiple resistant strains (with high resistance to synthetic pyrethroids) collected from Banchou, synergism of synthetic pyrethroid toxicity by PB, but not by DEF (S,S,S-tributyl phosphorothioate), was observed (Table 6). The results seemed to indicate that oxidative metabolism mediated by microsomal oxidases contributed, at least in part, to synthetic pyrethroid resistance in DBM, whereas hydrolytic metabolism might not be involved. But, among synthetic pyrethroids, PB synergism was the lowest against permethrin (Liu et al 1981). Teh et al (1982) reported only a slight synergism with PB against permethrin in DBM from the Cameron Highlands (resistance ratio against permethrin >700).

Table 6. Effect of piperonyl butoxide (PB) and DEF on the susceptibility to several synthetic pyrethroids of Ban-chou (resistant) strain of DBM[a]

Insecticide		LC_{50} (mg/ml)				LC_{50} (mg/ml)		
	RR[b]	No PB	+PB[c]	SR[d]	RR	No DEF	+DEF[c]	SR
Permethrin	14.1	0.48	0.18	2.7	77.6	2.64	1.24	2.1
Cypermethrin	87.3	2.88	0.17	16.7	316.4	10.44	6.71	1.6
Deltamethrin	67.1	0.94	0.02	47.0	714.3	2.22	2.04	1.1
Fenvalerate	207.7	2.70	0.09	30.0	701.5	9.12	8.15	1.1

[a] Source: Liu et al 1981. [b] RR: resistance ratio. [c] Larvae were sprayed with 1.0 mg of PB/ml one hour prior to insecticide treatment. [d] SR: synergistic ratio.

From genetic analysis, it was demonstrated that fenvalerate resistance in DBM was partially recessive and was due to more than one gene (Liu et al 1981).

In some organophosphorus insecticide resistant species, resistance correlates with esterase activities (Motoyama and Dauterman (1974). Sun et al (1978) demonstrated a qualitative difference in esterase activity between susceptible and methomyl- or diazinon-resistant DBM strains in Taiwan. However, Miyata et al (1984) could not demonstrate a qualitative difference in esterase between Japanese strains of DBM.

Noppun et al (1984a) studied the effect of PB, triphenyl phosphate (TPP), and fenvalerate on the toxicity of phenthoate to phenthoate-selected and non-selected DBM strains (Table 7). High co-toxicity coefficient values were obtained by the mixture of phenthoate and TPP combinations against phenthoate-selected strains, but not against non-selected strains. This suggests that an enhanced carboxyl-esterase degrading phenthoate might be involved as one of phenthoate resistance factors. However, the mixture of TPP and phenthoate could not completely suppress phenthoate resistance, suggesting that other resistance mechanism(s) could be involved. *In vitro* acetylcholin-

Table 7. Joint toxicity of phenthoate to non-selected and phenthoate selected DBM strains[a]

Strain	LD50 or CC[b] of LD50 of phenthoate							
	+ PB[c]	CC	+ TPP[d]	CC	+ TPP[e]	CC	+ Fenval[f]	CC
Sel[g] Okinawa	12800	112.0	3270	713	3570.0	664	0.721	189
Non-sel Okinawa	452	92.5	165	129	96.3	222	0.477	190
Sel Osaka	12600	107.0	2610	759	2980.0	664	0.633	170
Non-sel Osaka	400	58.4	97	123	55.8	213	0.330	263

[a] Source: Noppun et al 1984a. [b] Co-toxicity coefficient, [c] Pineronyl butoxide. [d] Phenthoate + TPP (simultaneous treatment). [e] Phenthoate + TPP (pretreatment). [f] Fenvalerate. [g] Selected.

esterase inhibition by phenthoate-oxon indicated that acetylcholinesterase of phenthoate-selected DBM strains is less sensitive to phenthoate-oxon than non-selected strains. Sun et al (1978) also mentioned that acetylcholinesterase of the diazinon-resistant strain was less sensitive to diazinon than that of a susceptible strain.

The role of glutathion-S-transferase as an organophosphorus insecticide detoxification mechanism was investigated by Cheng et al (1983, 1984). The results indicated that glutathione-S-transferase activities in the organophosphorus-resistant strains were three to four times higher than in the susceptible IL strain.

Liu et al (1981) mentioned that several mechanisms, including differential penetration, metabolism by microsomal oxidases and soluble enzymes, and possibly acetylcholin-esterase insensitivity, were involved in DBM resistance to diazinon and methomyl. However, this could not provide an explanation for the observed cross resistance patterns of diazinon-resistant and methomyl-resistant strains to some synthetic pyrethroids.

Measures to Overcome Insecticide Resistance

It has become increasingly difficult to develop new insecticides. In the United States 1800 compounds were screened to develop one new compound in 1956; 3600 in 1965; and 10,000 in 1972. The cost of $20 million to develop each marketable compound, estimated in 1977, was almost 17 times the cost estimated in 1956 ($1,196,000). The conclusion is that it will become increasingly difficult and expensive to discover and develop new pesticides (Metcalf 1980).

Therefore, it has become more difficult to introduce alternative insecticides which have no cross resistance when insects show resistance to certain insecticides. The use of synergists is a common measure to overcome insecticide resistance. As far as we know at this stage, we have no effective synergist to overcome insecticide resistance in DBM (Liu et al 1981, Noppun et al 1984a).

Certain combinations of insecticide mixtures and insecticide rotation are promising approaches for retarding insecticide resistance (Georghiou et al 1983). Miyata and Saito (1984) reviewed many successfull cases of suppression of resistance by a combination of synergistic insecticides and alternative use of chemical combinations with negatively correlated cross resistance in the green rice leafhopper, *Nephotettix cincticeps* Uhler (Hemiptera: Deltocephalidae), the brown planthopper, *Nilaparvata lugens* Stal (Hemiptera: Delcephalidae) and the smaller brown planthopper, *Laodelphax striatellus* Fallen (Hemiptera: Delphacidae).

El-Guindy et al (1983) studied cross resistance patterns to certain insecticides and insect growth regulators in the diflubenzuron-resistant strain of *Spodoptera littoralis* F (Lepidoptera: Noctuidae). The diflubenzuron resistant strain showed slight cross resistance to synthetic pyrethroids (fenvalerate and cypermethrin) and endrin, but

extremely low cross resistance to insect growth regulators (resistance ratio for methoprene 0.006 and for tripren 0.05).

However, no good combination of insecticides has yet been found against insecticide-resistant DBM (Sasaki 1982). Even though the insecticide resistance problem in DBM has become a worldwide phenomenon, the study of resistance mechanisms is not yet complete. The promotion of basic studies will result in progress in monitoring for the development of insecticide resistance and the development of new countermeasure, to control resistant DBM, or at least to retard the development of insecticide resistance.

As mentioned previously, insecticide resistance is caused by insecticide selection. Therefore the establishment of integrated pest management programs is also very important for the control of DBM.

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