

## INSECTICIDE RESISTANCE IN DIAMONDBACK MOTH IN NEW ZEALAND

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### SUMMARY

Samples were collected from four different populations of diamondback moth and these were used to establish laboratory colonies. The F1 generation from these colonies was tested for insecticide resistance using leaf dip assays and a discriminating dose. Resistant strains were identified in Pukekohe and Palmerston North.

### INTRODUCTION

The diamondback moth (*Plutella xylostella*) is a cosmopolitan species of considerable importance as a pest of cruciferous plants. Diamondback moth has 14-28 generations per year in Malaysia, 15-20 in Taiwan, 5-12 in Japan and 6-7 in New Zealand, with overlapping of all development stages (Miyata *et al* 1986; Valentine 1975). The females of each generation can lay up to 100 eggs (Valentine 1975). This means that this species is capable of a rapid growth in numbers and has a high potential for the development of insecticide resistance. Since 1953 numerous cases of insecticide resistance by diamondback moth to various types of insecticide have been reported from around the globe. By 1986 resistance to 23 insecticides in 16 countries had been confirmed (Georghiou 1981; Sun *et al* 1986). The highest levels of resistance have been found in the Ban Chau strain in Taiwan where the resistance ratio (LD<sub>50</sub> of the resistant population/LD<sub>50</sub> susceptible population) ranges from 2 for *Bacillus thuringiensis* to 50,000 for cyanofenphos, with intermediate values for eleven other insecticides (Sun *et al* 1986). In 1987 the New Zealand Committee on Pesticide Resistance identified diamondback moth as a potential resistance problem in this country (Elliot *et al* 1987).

The aim of this work was to establish whether or not diamondback moth was developing a resistance to insecticides in New Zealand.

### METHOD

Populations of diamondback moth were collected from a forage brassica crop on a Massey University farm (with a history of no insecticide use), and from market gardens in Pukekohe (2 sites), Levin and Palmerston North. The populations were sampled by collecting third and fourth instar larvae and pupae. The colonies were then synchronised by placing the larvae on potted cabbage plants and any pupae collected were held at 4°C. As the larvae pupated they were removed from the host plant and placed into cold storage until all of them had pupated. They were then removed from the cold store and allowed to continue their development at room temperature. Larvae from the Massey University Farm were collected in a transect across the paddock. In the other cases sufficient larvae could only be found by searching a high proportion of brassica plants showing evidence of diamondback moth damage as in all cases these crops had been treated with insecticide within 5 days prior to sampling and the numbers of surviving insects was low (ca 10% of plants showed some evidence of damage).

The adult insects were held in cages with a supply of cabbage plants cv "Flower of Spring" for oviposition. Once sufficient eggs had been laid on any plant it was removed from the cage and placed in an incubator at 60% RH and 25°C on a 12:12 light:dark cycle. The plants were held in the incubator until the majority of the insects had reached the third instar.

The colony from the Massey farm was used to develop a dose mortality curve for susceptible insects. The larvae were removed by shaking the plant until they dropped down on a thread. They were then counted into small vials in groups of approximately 10.

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A leaf dip test was conducted by cutting 7 cm diameter leaf discs from young fully expanded cabbage leaves and dipping them into a solution or suspension of the commercially available formulation of one of three insecticides (Table 1) in water. The treated discs were hung in a fume cabinet until dry (ca 1 hour). Once dry the discs were placed in a petri dish on top of three small polystyrene blocks so that the larvae had access to both sides of the leaf disc. Ten third instar larvae were then placed onto the centre of each leaf disc. Four discs were used for each treatment. The petri dishes were held at 60% RH and 25 °C for 24 hours.

After 24 hours the larvae were assessed as being either alive or dead. Dead larvae were those that showed no signs of movement when prodded gently with a dissecting needle. Data were analysed by probit analysis using the computer program Polo (Robertson *et al* 1980). The LD<sub>50</sub>, LD<sub>90</sub> and LD<sub>95</sub> were calculated for each insecticide.

The colonies collected from properties regularly exposed to insecticides were tested for resistance using a discriminating dose. That is, samples of third instar larvae from the colony were exposed to concentrations of different insecticides (Table 2) that could be expected to kill almost all of the susceptible individuals in the colony. This method is claimed by Roush and Miller (1986) to be more sensitive than the alternative method of using a range of dose rates to establish the LD<sub>50</sub> of the population and hence the resistance ratio. The LD<sub>95</sub> from the Massey Farm colony was selected as the discriminating dose.

One of the colonies from Pukekohe was maintained for five generations and then retested using the discriminating dose technique to see if the level of resistance declined in the absence of insecticide selection pressure.

#### RESULTS AND DISCUSSION

High levels of parasitism (71% - 97%) by the larval parasite *Diadegma cerophagus* were found on all properties, including those regularly treated with pesticides. This may indicate that the parasite has become resistant to a range of insecticides but further work is needed to confirm this. It is possible that a behavioural response by the parasitized insect means that they pupate in more exposed positions on the plant than the healthy insects. Therefore the sampling method used to collect the insects for evaluation lead to a disproportionate number of parasitized pupae being found and collected.

Table 1 shows a comparison of the LC<sub>50</sub> values for three insecticides trialed against the Massey population and the LC<sub>50</sub>'s of a number of laboratory colonies reported as susceptible. It can be seen that the Massey population was more susceptible to diazinon and permethrin than the laboratory colonies but it appeared to be less susceptible to carbaryl.

**TABLE 1: Comparison of LC<sub>50</sub> values for the Massey population with other published data based on leaf dip assay (mg/ml).**

Insecticide	Population		
	LC <sub>50</sub>	95% C.L.	Published* (various sources) LC <sub>50</sub>
diazinon	0.033	0.015 - 0.054	0.19 - 0.75
permethrin	0.006	0.003 - 0.008	0.017 - 0.02
carbaryl	5.758	4.230 - 7.731	3.2

\*Data from Tabashnick *et al* (1987)

Table 2 sets out the results of the discriminating dose test against the different populations. The Pukekohe 1 colony showed some resistance to all of the insecticides evaluated with the exception of methomyl. The highest level of resistance displayed was to carbaryl where only 16% of the larvae were killed with the discriminating dose as compared to 93% for the Massey colony. This indicates that approximately 84% of the population were resistant individuals. The Pukekohe 2 colony showed similar trends but

**TABLE 2: Discriminating dose test giving the percent mortality at the LD<sub>50</sub> level for the susceptible population (Massey), and two Pukekohe and one Palmerston North population.**

Insecticide	% Mortality $\pm$ 95% C.L.			
	Massey	Pukekohe 1	Pukekohe 2	Palm. Nth
esfenvalerate	92 $\pm$ 9	47 $\pm$ 15	59 $\pm$ 15	81 $\pm$ 13
permethrin	90 $\pm$ 9	38 $\pm$ 16	55 $\pm$ 16	61 $\pm$ 15
diazinon	88 $\pm$ 10	37 $\pm$ 19	40 $\pm$ 15	100
dichlorvos	98 $\pm$ 4	62 $\pm$ 15	90 $\pm$ 9	52 $\pm$ 15
mevinphos	98 $\pm$ 5	66 $\pm$ 14	75 $\pm$ 13	
methomyl	95 $\pm$ 7	82 $\pm$ 13	74 $\pm$ 14	57 $\pm$ 14
carbaryl	93 $\pm$ 7	16 $\pm$ 11	37 $\pm$ 15	41 $\pm$ 15

Insecticides used: esfenvalerate — Hallmark; permethrin — Ambush 50ec; diazinon — Basudin 80ec; dichlorvos — Nuvan 100; mevinphos — Phosdrin; methomyl — Lannate; carbaryl — Carbaryl 80.

with reduced resistance levels. Both of these properties were sprayed with permethrin on a 7-14 day schedule whenever growers found insects in the crop. It is likely then, that the same resistant individuals were responsible for the initiation of several applications of pesticide at close intervals while they completed their life cycle. The colony collected in Palmerston North showed some degree of resistance but in a different pattern to the Pukekohe colonies indicating that the cross resistance spectra are different, and by implication that the resistance mechanism may also be different. The Palmerston North population was sprayed with permethrin on a 7-14 day schedule and with mevinphos close to harvest. Sun *et al* (1986) found that both metabolic and nonmetabolic mechanisms could be responsible for insecticide resistance in diamondback moth and that differences in the resistance spectra between strains could be due to variation in the relative importance of each of these mechanisms. The colony collected from Levin (not included in the table) showed no signs of insecticide resistance.

**TABLE 3: Percent mortality in discriminating dose test after one and five generations of the Pukekohe 1 population.**

Insecticide	% Mortality ( $\pm$ 95% C.L.)	
	F1	F5
esfenvalerate	47 ( $\pm$ 15)	85 ( $\pm$ 11)
permethrin	38 ( $\pm$ 16)	71 ( $\pm$ 14)
diazinon	37 ( $\pm$ 19)	97 ( $\pm$ 3)
dichlorvos	62 ( $\pm$ 15)	97 ( $\pm$ 3)
mevinphos	66 ( $\pm$ 14)	93 ( $\pm$ 7)
methomyl	82 ( $\pm$ 13)	93 ( $\pm$ 7)
carbaryl	16 ( $\pm$ 11)	60 ( $\pm$ 14)

Table 3 shows the results of the discriminating dose test on the first and fifth generations of the Pukekohe 1 colony. After only five generations in the absence of selection pressure the level of resistance regressed to the point where only permethrin and carbaryl were still showing impaired control.

It would seem then that in Pukekohe and Palmerston North diamondback moth have become resistant to a range of insecticides. As yet this has not lead to control problems despite the fact that the growers are unaware of the situation. The incidence of resistant individuals in the crops is low. However this situation may change in the future although the high level of parasitism found may be controlling the spread of the resistant insects.

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