MELON APHID (*APHIS GOSSYPII*) RESISTANCE TO PESTICIDES

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ABSTRACT

Eleven pesticides were applied at their recommended concentrations through a Potter Tower to melon aphid (*Aphis gossypii*), (Homoptera: Aphididae) from potted chrysanthemums (*Chrysanthemum* x *morifolium*) in Auckland. The aphids were either topically sprayed or placed on spray residues on chrysanthemum leaf discs embedded in agar. Mortality was assessed after 48 h. The LC_{50} for topical application of pirimicarb was also assessed and was over 10 times the recommended field spray concentration. The synthetic pyrethroids, lambda-cyhalothrin and tau-fluvalinate, caused low mortality. The residues of dichlorvos, demeton-s-methyl, methamidophos, acephate, methomyl, maldison and pirimiphos methyl/ permethrin mixture, gave lower mortality than topical applications. Mortality was similar for maldison and methomyl in both tests. Endosulfan gave 100% mortality in both tests. Growers are recommended to implement a melon aphid resistance management strategy.

Keywords: Melon aphid, *Aphis gossypii*, pesticide resistance, *Chrysanthemum*, bioassay

INTRODUCTION

There have been many reports of aphids resistant to pesticides (e.g. Georghiou 1981) but in New Zealand resistance has only been demonstrated in one species *Myzus persicae* (Homoptera: Aphididae) (Cameron and Walker 1988). In 1996, chrysanthemum (*Chrysanthemum* x *morifolium*) pot plants used to rear western flower thrips were found to be infested with melon aphid (*Aphis gossypii*). These aphids survived a thorough application of pirimicarb which was intended to remove them without harming the thrips. This paper reports tests for resistance to pirimicarb in a population of melon aphid in New Zealand and the susceptibility of the aphids to recommended concentrations of ten other pesticides with label claims for aphid control.

METHODS

Two bioassays were compared when testing the efficacy of 11 pesticides at their recommended concentrations (Table 1). Aphids were either exposed to pesticide residues on chrysanthemum leaf discs or directly sprayed with pesticide. The topical application bioassay was also used for a more detailed study of pirimicarb. Leaf discs (23 mm diam.) were cut from unsprayed chrysanthemum leaves and a single leaf disc embedded in 1% agar in small Petri dishes (30 mm internal diam. and 10 mm high). For the residue test, the leaf discs were sprayed in groups of five on the abaxial surface and dried before embedding in agar. Ten adult aphids were then put in each dish. In the topical test, 10 aphids were placed on each leaf disc embedded in the Petri dish and one dish at a time was sprayed. For each bioassay, 2 ml of pesticide was applied through a Potter tower at 68.9 kPa. A water only treatment was included. The closed Petri dishes had a disc of filter paper in the lid and were stored with the leaf surface upper most at 25°C and 16:8 h light:dark; aphid mortality was assessed after 48 h. Three dishes of each pesticide by application method were used for each test. The comparison of 11 pesticides in the two bioassays was repeated twice (n = 60 aphids per pesticide).

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A further bioassay was carried out to estimate the LC_{50} of pirimicarb to assess possible resistance in comparison to the recommended dose. The aphids were sprayed on a leaf disc. After a range finding test, mortality of melon aphids to five or six concentrations of pirimicarb was determined in four repetitions of the bioassay. Ten dishes containing ten aphids were used for each dose in each of the four repetitions.

The aphids were obtained from chrysanthemum plants grown by a commercial nursery. They were reared on plants from the same nursery. Voucher specimens were deposited in the NZ National Arthropod collection, Landcare, Auckland.

TABLE 1: Mortality of melon aphid in two bioassays following use of recommended concentration pesticides with label claims for aphid control. LSD (5%) = 15.75 (df=119).

Pesticide category	Common chemical name	Pesticide formulation	Recommended application rate (g or ml/ 100 litres)	Recommended application rate g a.i./ litre	Mortality of aphids (percent) Topical application	Mortality of aphids (percent) Pesticide residues
Carbamate	methomyl	Lannate	120 ml	0.24	97	85.5
	pirimicarb	Pirimor	25 g	0.125	3	10
Cyclodiene	endosulfan	Thiodan	200 ml	0.71	100	100
Organo-phosphate (OP)	dichlorvos	Dichlorvos	100 ml	1.0	100	20
	maldison	Malathion	200 ml	1.0	100	91.1
	demeton-S-methyl	Metasystox	100 ml	0.25	87.9	28.9
	acephate	Orthene	100 g	0.75	72.1	54.6
	methamidophos	Tamaron	150 ml	0.9	100	78.9
Synthetic pyrethroid (SP)	lambda-cyhalothrin	Karate	20 ml	0.02	4.7	8.4
	tau-fluvalinate	Mavrik	40 ml	0.05	18.6	6.4
Mixture SP & OP	permethrin (SP) pirimiphos-methyl (OP)	Attack	100 ml	0.025 0.475	100	48.1
	untreated (water only)				3.3	2.8

Percent mortality of aphids treated with the recommended concentration of 11 pesticides (residue and topical) was examined using analysis of variance (ANOVA). The standard checks of assumptions indicated that the data did not need transforming before analysis.

Mortality from the pirimicarb dose-response experiments was modelled using a logistic dose-response curve in natural log of dose:

$$\% \text{ Dead} = C + \frac{100 - C}{}$$

$$1 + e^{b(\ln(Dose) - \ln(LC_{50}))}$$

where b is related to the steepness of the curve at dose=LC_{50}, C is % natural mortality (at dose=0), and LC_{50} is the dose which kills half of the remaining (100-C)% insects. This model was fitted to the data as a Generalised Linear Model (McCullagh and Nelder 1989) using Genstat (Genstat 5 Committee 1993), taking account of the binomial nature of mortality data. The model was fitted several times with the parameters (LC_{50}, C and b) either being the same for, or allowed to vary between, the four experiments. The results of fitting these various models were compared to test whether any or some of the parameters varied between the four sets. A logistic regression was also fitted to estimate the mean mortality for each dose in each experiment. This also gave an estimate of the underlying random variability in the data around the means (dispersion), and, by comparison, allowed a test of goodness of fit for the other models (i.e. how well any model described the mean responses).

RESULTS

ANOVA of mortality of aphids caused by application of the recommended concentration of pesticides showed significant ($P<0.001$) interaction between pesticides and bioassay type (Table 1). Residues on leaf discs of five pesticides, acephate, demeton-S-methyl, dichlorvos, methamidophos, and a mixture of pirimiphos methyl and permethrin gave reduced mortality ($P<0.05$) compared with topical application. In the topical application bioassay, mortality caused by three pesticides, lambda-cyhalothrin, pirimicarb and tau-fluvalinate, was not different ($P>0.05$) from that for untreated aphids. In the residue test, only two pesticides, endosulfan and maldison, caused mortality not different ($P>0.05$) from 100%.

There was considerable variation between the dose-response experiments with pirimicarb (Table 2), with the LC_{50} and P (but not b) varying ($P<0.05$) between them. Thus, the LC_{50} for each experiment is presented. There was no lack of fit for this model, indicating that it described the mean responses adequately. In all cases, the LC_{50} was more than 10 times the recommended concentration, confirming that the poor kill from spraying the aphids on chrysanthemum plants was due to resistance. The variability between tests may have been due to the continued exposure of the aphids to pesticides on the pot plants on which they were reared, and variability in the quality of the unsprayed chrysanthemum leaves used for the bioassays.

TABLE 2: Mortality of melon aphid 48 h after spraying aphids on chrysanthemum leaves with different concentrations (g a.i./litre) of pirimicarb on four occasions.Control mortality, (C), varied between 2.3 and 16.4%, b = 2.010 (S.E. 0.134), df=234. Standard errors and confidence limits are based on the dispersion estimated from the logistic regression.

Date tested	Dose (g/litre)		
	LC_{50}	S.E.	95 % confidence limits
15 Oct 96	1.44	0.17	1.11 - 1.78
22 Oct 96	3.38	0.36	2.78 - 3.98
5 Nov 96	8.57	0.76	7.08 - 10.07
21 Nov 96	3.94	0.34	3.28 - 4.60

DISCUSSION

Although it is unlikely that several pesticides, e.g. pirimicarb, acephate and the pyrethroids, will give field control of melon aphid, there is no measure of level of resistance in the population from chrysanthemums, because the susceptibility to pesticides was not compared to a known susceptible population. Different populations of melon aphid may have various combinations of resistance to pesticides (e.g. Hollingsworth *et*

al. 1994; Silver *et al.* 1995). Overseas populations are resistant to endosulfan and maldison, but in this present study both chemicals gave high mortality.

The bioassay involving topical application of pesticide to the aphid gave higher aphid mortality than when they were exposed to the same dose of pesticide as a residue on leaves, even for systemic pesticides. Bioassays using residues, (e.g. leaf dips or spray deposit on leaves) may underestimate the ability of the pesticide to kill insects, but may indicate persistence of activity once the deposit has dried. Topical tests measure both the direct effect and the effect of the residues. The relative merits of both types of bioassay should always be considered in light of the questions being asked.

Although melon aphid has a wide host range, it is possible that a resistant strain adapted to chrysanthemums would grow poorly on an alternative host such as cucurbits. We carried out comparative fecundity assessments with the resistant strain on chrysanthemum and cucumber leaves (Martin and Workman unpubl.) which showed that fecundity over 48 h was similar (respectively 3.8 and 2.3 juveniles /aphid) on these host plants. This demonstrates that cucurbits such as greenhouse cucumbers and squash are potential hosts of this resistant strain of aphid. There has been a recent report of melon aphid on asiatic lilies in Nelson not being controlled by pirimicarb, acephate and a synthetic pyrethroid insecticide.

It has not been possible to trace the origins of the resistant melon aphid, but preceding its discovery, the grower had regularly sprayed crops with acephate and tau-fluvalinate with occasional applications of pirimiphos-methyl/permethrin. No pirimicarb has ever been used. Because of the risks from this aphid, all growers should immediately implement an appropriate pesticide resistance management strategy (Martin and Cameron 1997). Our findings also suggest that even where aphid species are not known to be resistant to pesticides, growers should adopt aphid resistance prevention and management strategies as part of standard crop production practice.

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