THE EFFECTS OF MANCOZEB AND METIRAM ON THE PREDATORY MITE, *TYPHLODROMUS PYRI*

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SUMMARY

Using the slide-dip method the fungicides, mancozeb and metiram, did not kill adults and nymphs of the predatory mite *Typhlodromus pyri* at recommended field rates. Exposure of *T. pyri* to residues of mancozeb had no effect on egg hatch, fecundity, development rate or mortality. Both fungicides reduced egg hatch when directly sprayed at field rates onto 0-24 h old eggs. Metiram had a greater effect than mancozeb, while at half field rates there were no differences in egg hatch between fungicides. Older eggs were less susceptible to both fungicides. The implications of these results for interpretation of field trials are discussed.

INTRODUCTION

Integrated mite control (IMC) using the organophosphorus-resistant predatory mite *Typhlodromus pyri* for control of the European red mite (*Panonychus ulmi*) requires a compatible spray programme. Insecticides must allow predator survival, and selective miticides and fungicides must have minimal effect on predator biology (Wearing and Ashley 1982). Organophosphorus insecticides are generally compatible with IMC as are the organotin miticides, so effective insect and mite control can be based around these chemical groups. Control of fungal diseases, black spot (*Venturia inaequalis*) and powdery mildew (*Podosphaeria leucotricha*), is also very critical in apple growing in New Zealand with up to 20 fungicide applications made annually depending on weather conditions (Hancox and Penman 1982). Compatibility with fungicides is therefore essential for predator survival given this intense chemical pressure.

A current issue pertaining to IMC in New Zealand is the use of the ethylene-bisdithiocarbamate (EBDC) protectant fungicides, mancozeb and metiram. Field evidence has suggested a suppression of *T. pyri* numbers in orchards where mancozeb has been used (Collyer 1980). Suppression was particularly severe following repeated use early in the season (Wearing and Wilton 1984) with predator populations recovering only slightly following cessation of applications.

Metiram is an alternative protectant fungicide to mancozeb. Wearing and Proffitt (1982) found that metiram suppressed *T. pyri* populations but to a lesser extent than mancozeb, and Wearing and Wilton (1984) noted a suppression with early season use followed by population recovery when metiram applications ceased.

Reports indicate a low toxicity of mancozeb and metiram to *T. pyri* in laboratory studies (Watve and Lienk 1975). This paper reports on a laboratory study into the effects of mancozeb and metiram on various life stages of *T. pyri*.

MATERIALS AND METHODS

A *T. pyri* culture was begun from field collections from apples (cv. 'Gala') at Appleby Research Orchard, D.S.I.R., Richmond. Active life stages were transferred to black acrylic plates (120 x 70 mm) and reared as per the method described by Markwick (1986). Two-spotted spider mites and pollen were used as food.

Direct toxicity to adults and nymphs

Direct toxicity of mancozeb (Dithane M45 — 80% ai) and metiram (Polyram 2000 — 80% ai) was evaluated at the recommended field rate (0.1 g ai/100 ml) by the slide-dip method (Anon. 1968). Twenty adults and nymphs were tested in each replicate, with five replicates for adults and three for nymphs. Mites were placed ventral side up on double-sided sticky tape on a glass slide. Slides were dipped in the fungicide suspension

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for 5 sec. A control was prepared by dipping mites in water. The mites were held at $25 \pm 1^\circ\text{C}$ and $73 \pm 5\%$ relative humidity. Mortality was assessed after 24 h with death being the absence of leg movement following prodding with a camel's hair brush. Data were adjusted for control mortality using Abbott's (1925) formula.

Direct toxicity to eggs

Newly laid eggs were removed from the rearing plates, placed on plastic petri dishes (90 mm diam.), and either treated immediately (0-24 h old eggs), or held for a further 24 h for treatment of 24-48 h old eggs. Eggs were sprayed under a Potter Tower at a pressure of 103 kPa. An aqueous fungicide suspension was sprayed on each replicate and a settling period of 10 sec. was allowed. After spraying the dishes were placed in a sealed chamber at $25 \pm 1^\circ\text{C}$ that maintained humidity at 75% using a 30.14% H_2SO_4 solution. Egg hatch was assessed 72 h after treatment for 0-24 h old eggs and 48 h for 24-48 h old eggs. Six concentrations of mancozeb ranging from $0.25 \times$ to $2 \times$ field rate plus a distilled water control were applied to 0-24 h old eggs. Each treatment was replicated 10 times with 20 eggs per replicate. Metiram was applied at two rates: $0.5 \times$ and field rate. Twenty-four to 48 h old eggs were treated at field rates of mancozeb and metiram. For the latter two experiments, five replicates, each of 40 eggs, were treated.

Residual effects of mancozeb

Four rearing plates plus pollen and portions of dwarf bean (*Phaseolus vulgaris*), leaves used as predator refuges, were sprayed with the field rate of mancozeb. Each plate was then divided into six sections using a sticky polybutene-based substance. Three newly emerged, mated female *T. pyri* were placed in each section. Daily observations were made of female mortality, oviposition and development rate of nymphs over a 15 day period. Identical unsprayed plates were established as controls. Plates were held at $24 \pm 1^\circ\text{C}$ and $75 \pm 5\%$ R.H.

RESULTS

Direct toxicity to adults and nymphs

Mancozeb and metiram caused no significant mortality of *T. pyri* adults and nymphs when tested at the field rate (Table 1). The low mortality at field rates did not justify the development of complete dosage-mortality relationships.

TABLE 1: Toxicity of mancozeb and metiram applied at field rates (0.1 g ai/100 ml) in slide-dip tests to adults and nymphs of *Typhlodromus pyri*.

Stage	untreated	Percentage mortality	
		mancozeb	metiram
Adults	0.8	1.2	1.2
Nymphs	4.0	6.0	5.4

($P > 0.10$)

Direct toxicity to eggs

Spraying 0-24 h old eggs with mancozeb significantly ($P < 0.05$) reduced hatching (Fig 1.). Observed hatch was 92% for unsprayed eggs with hatch declining in a linear relationship with increasing doses. At the field rate, 73% hatched.

Metiram treated eggs gave hatch values of 81% at $0.5 \times$ field rate and 51% at field rate ($P < 0.01$) in contrast to a 93% hatch for the untreated eggs. Comparing hatching of 0-24 h old eggs treated at the field rate for mancozeb and metiram indicated a significantly greater ($P < 0.01$) effect of metiram. At $0.5 \times$ field rate, differences were not significant.

Treatment of 24-48 h old eggs at field rates of mancozeb and metiram gave hatch values of 88% and 76% respectively in comparison to untreated eggs at 94% ($P < 0.01$). The effects on egg hatch were less ($P < 0.01$) for 24-48 h old treated eggs than for 0-24 h old treated eggs.

Residual effects of mancozeb

No female mortality was recorded and oviposition rates were identical between untreated and treated plates. Percentage hatch was not affected by eggs being laid on

Fig. 1: Percentage hatch of 0-24 h old eggs of *Typhlodromus pyri* sprayed with mancozeb.

mancozeb residues. Development rate from egg to adult did not differ between treatments with generations being completed in 8 days.

DISCUSSION

Compatible spray programmes are essential for effective IMC. Disruption of predator effectiveness following use of protectant fungicides, such as mancozeb and metiram, has been suggested (Wearing and Wilton 1984). Field observations indicated that the *T. pyri* numbers declined following several fungicide applications suggesting that chronic rather than direct toxicity may be the main cause.

Direct mortality of the adult and nymphs of *T. pyri* is not a factor, confirming the results of Watve and Lienk (1975) in New York. Treatment of the eggs showed that young (0-24 h) eggs were more susceptible than older (24-48 h) eggs to both mancozeb and metiram. Contrary to reported field observations (Wearing and Ashley 1982; Wearing and Wilton 1984) field rates of metiram were relatively more toxic than mancozeb to young eggs. The reduction in susceptibility of older eggs to both fungicides suggests that timing of application may be critical.

Field rates of both fungicides are only moderately toxic to young eggs. Unaffected eggs hatch normally and our experiments on the effect of mancozeb residues on development of *T. pyri* indicate no subsequent effects of residues. Field responses may, therefore, be a result of cumulative egg mortality following several applications of fungicide through the main black spot infection period.

The concentration-mortality relationship with mancozeb and young eggs and the lower mortality from metiram at half-rates suggest the fungicides could be used at lower rates provided they remain effective against target pathogens. More frequent fungicide applications at lower rates may lessen disruption to *T. pyri* numbers. Further studies are required on rates, timing and frequency of application of metiram and mancozeb to minimise their disruptive effects on IMC.

Degradation products of mancozeb and metiram may be the cause of egg mortality. Since ethylene thiourea is a known degradation product of EDBC's, causing suppression of ovarian development in houseflies (Mitlin and Baroody 1958; Newsome 1980), we treated young eggs with thiourea. The reduction in egg hatch to 23% following application of 0.1 g/100 ml of thiourea suggests the need for further study of

the role of metabolites. Given the inherently unstable nature of the EDBC's (Newsome 1980), storage time and conditions may be an additional factor complicating field responses.

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