INVESTIGATING THE IMPACT OF PROTECTANT FUNGICIDES ON INTEGRATED MITE CONTROL. II RESULTS OF THREE FIELD EXPERIMENTS

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SUMMARY

Mancozeb and metiram have been implicated in the disruption of integrated mite control based on the predator mite *Typhlodromus pyri*. Three field studies reported here confirm that these fungicides disrupt mite control by suppressing the development of *T. pyri* populations. Suppression of *T. pyri* increased as the period of use was extended and the number of applications increased. Consequently, mite damage was more severe as the number of applications increased from four to eight per season. Both metiram and mancozeb appeared to have delayed toxicity to immature stages of the first summer generation of *T. pyri*. Delayed toxic effects were more apparent with mancozeb than metiram.

INTRODUCTION

Integrated control of European red mite by the predator mite, *Typhlodromus pyri*, is a well recognised strategy for mite control in apple orchards. To operate an effective integrated mite control (IMC) programme an orchardist must avoid or minimise the use of pesticides which are toxic to the predator. Two fungicides, metiram and mancozeb, are reportedly disruptive to IMC based on information from field trials where these products have been used (Wearing and Ashley 1982; Wearing and Wilton 1984; Walker *et al* 1988).

In laboratory studies, attempts to determine the reason for this disruption have not revealed any major toxicity of these products to *T. pyri*. Baynon and Penman (1987) found that both metiram and mancozeb reduced the hatch rate of young *T. pyri* eggs but did not find significant mortality to adults, or immature stages, with field rates of either product. Further laboratory studies on the effect of these fungicides on *T. pyri* were completed by Hayes *et al* (1989). They supported the findings of Baynon and Penman (1987) but, apart from demonstrating a small decrease in predation rates with exposure to metiram, did not identify any other effect which could explain the levels of disruption observed in field studies.

In this paper we report the results of field experiments which examine the relationship between the seasonal development of *T. pyri* populations and increasing exposure to metiram and mancozeb. We also attempt to explain the specific impact of metiram and mancozeb on *T. pyri* populations in early summer under field conditions.

MATERIALS AND METHODS

Trials 1 and 2

These were conducted at the DSIR Research Orchard, Appleby, Nelson on a large block of mature apple trees (cv. "Sturmer Pippin") over two seasons. In each trial there were four treatments; four single tree replicates were used in each treatment and these were separated by guard trees. Different trees were used in the second season to avoid testing mite populations which might have been affected by activities in the preceding season.

Trial 1 was carried out during the 1980-81 season. There were three treatments, *Proc. 42nd N.Z. Weed and Pest Control Conf.*

comprising four, six or eight applications of mancozeb (Dithane M45 80% ai) and a "control" treatment, which received a fungicide programme based on captan throughout. Mancozeb treatments commenced on November 6 (petal-fall) and ceased on December 16, January 27 and March 16 for the four, six and eight applications respectively. The rate of mancozeb used for the first four applications on each of these treatments was 150 g/100 litres; subsequently the rate was 125 g/100 litres.

Trial 2 was conducted in the 1981-82 season. The design was identical to Trial 1 but metiram (Polygram 2000 80% ai) was substituted for mancozeb. As in Trial 1, metiram applications commenced on November 6 and ceased on the same dates in each treatment. The rate of metiram used for the first four applications on each of these treatments was 150 g/100 litres, subsequently the rate was 100 g/100 litres.

In both studies mite populations in each treatment were monitored by recording active stages of ERM and *T. pyri* from 100 leaf samples (25 leaves from each replicate). These were collected at approximately 2 weekly intervals from late October until mid-April. Leaves were brushed using a mite brushing machine and the mites counted using a binocular microscope.

To present this data, cumulative mite-days are used as an index of total mite damage occurring on trees with different treatments over the season. These were calculated using the method described by Hoyt and Tanigoshi (1983):

$$\frac{ERM_{T_1} + ERM_{T_2}}{2} \times T_2 - T_1$$

where ERM is the mean number of mites per leaf

T₁-T₂ is the difference in days between two sampling dates

Trial 3

This was conducted on a block of 38 trees (cv. Red Delicious) at the DSIR Research Orchard, Havelock North, during the 1985-86 season. The block was divided into three 12-tree blocks. Each block was treated with one fungicidal product at 10-18 day intervals, from October 11, 1985 until March 10, 1986. The fungicides used were metiram, mancozeb or captan which were applied by airblast sprayer at 150, 150 and 125 g/100 litres respectively. No insecticides were applied.

T. pyri populations were counted on both leaf spurs and fruit spurs from late September ("pink" flowering stage). Initially, five clusters from each of the trees in the block (60 clusters per treatment) were examined using a binocular microscope. Later, as mite populations increased, this was reduced to five clusters from six trees in each block (30 clusters per treatment). Samples were taken at weekly intervals in early summer and then at two-weekly intervals from mid-summer. Numbers of all life stages of *T. pyri* were recorded to detect any differences in their proportions resulting from the different fungicide programmes. ERM populations were also monitored using a conventional leaf sampling programme.

RESULTS

Trials 1 and 2

The effect of mancozeb and metiram treatments on the seasonal changes in numbers of *T. pyri* active stages are shown in Figures 1a and 1b. For clarity of presentation the six applications treatment data are not presented but these did produce values intermediate to the four and eight applications data in both trials.

Numbers of *T. pyri* declined in the mancozeb treatments shortly after fungicide applications commenced and continued to decline until early January. In contrast, numbers of *T. pyri* increased in the captan treatment during this period so that by late December there was a 68-fold difference in predator numbers between this and each of the mancozeb treatments. Four weeks after the mancozeb applications ceased in the four applications programme (December 16) *T. pyri* numbers increased sharply in the presence of high ERM populations. In the eight applications programme some increase in *T. pyri* numbers did occur from mid-January but numbers were still much lower than in the four applications programme with a 9-fold difference recorded in mid-March.

Fig. 1a and 1b: The number of active stages of *T. pyri* per leaf resulting from either 4 (+—+) or 8 (□—□) applications of mancozeb or metiram respectively and compared to a captan (■—■) standard.

TABLE 1: Comparison of peak numbers of ERM and *T. pyri* per leaf for mancozeb (Trial 1) and metiram (Trial 2). The dates when *T. pyri* numbers first exceeded 1.5 per leaf are also shown for each treatment.

Treatment		Peak numbers per leaf		Time when <i>T. pyri</i> exceeded 1.5/leaf
		ERM	<i>T. pyri</i>	
Trial 1				
1.	mancozeb x4	87	3.9	10 March
2.	mancozeb x6	125	0.7	—
3.	mancozeb x8	52	0.4	—
4.	control	5	4.6	13 January
Trial 2				
1.	metiram x4	27	15.9	21 January
2.	metiram x6	32	11.1	4 February
3.	metiram x8	84	4.2	19 March
4.	control	3	6.0	24 December

Mancozeb applications consistently reduced the peak numbers of *T. pyri* as the number of applications increased (Table 1). They also delayed the time for *T. pyri* populations to reach 1.5 per leaf.

The effect of metiram applications on numbers of *T. pyri* produced results very similar to those obtained with mancozeb. Numbers of *T. pyri* declined in the metiram treatments in early summer, and by late December were 11 to 13-fold less than in the captan treatment. Numbers of *T. pyri* increased in the eight applications programme from mid-January but were still 7 times lower than in the four applications programme in mid-March. Peak numbers of *T. pyri* in the four and six metiram applications programme were higher than in the captan programme (Table 1), the consequence of higher ERM populations resulting from the former programmes. Additional metiram applications increasingly delayed the time for *T. pyri* populations to reach 1.5 per leaf.

The consequent disruption to IMC, expressed as cumulative mite-days, is shown in Figures 2a and 2b. Again, for clarity of presentation, the intermediate six application programme is not shown on these graphs. ERM populations in the mancozeb trial (Figure 2a) increased similarly in all treatments until mid-January. Thereafter the rate

Fig. 2a and 2b: The cumulative European red mite-days developing under either 4 (+—+) or 8 (□—□) applications of mancozeb or metiram respectively and compared to a captan (■—■) standard.

of increase in mite days in the captan treatment was markedly lower than in the mancozeb treatments. A similar pattern of mite population increase occurred in the metiram trial (Figure 2b) until early January when the captan treatment again departed from the metiram treatments.

Conspicuous leaf damage from ERM usually commences at around 400 mite-days (Walker, White unpublished data). In both trials mites in the captan treatments were adequately controlled by *T. pyri*; the total number of mite-days never exceeded 400 and no obvious leaf damage occurred. In contrast, 400 mite-days were exceeded in all mancozeb and metiram treatments by mid-February and trees receiving these fungicides suffered severe mite damage by late summer. Peaks in ERM numbers per leaf generally increased with the number of applications (Table 1).

Trial 3.

The effects of the fungicide treatments on *T. pyri* are shown by (a) the proportion of different life stages within each sample (Figures 3a-c), and (b) the total numbers of *T. pyri* per cluster during the season (Figure 3d).

Early season leaf sampling showed that the distribution of ERM was uniform and their numbers very low. Cluster sampling showed that the distribution of *T. pyri* throughout the block was uneven, being highest in the metiram block then declining through the mancozeb treated trees to be lowest in the captan block. Despite this population gradient, the ratio of the different life stages was similar in each treatment until early November. The overwintered fertilised females laid eggs and then gradually died. The effects of the fungicide treatments only became apparent as these eggs hatched.

Metiram treatment: Throughout November the population development of *T. pyri* followed an expected pattern, in which the proportion of eggs declined as they hatched, increasing the proportion of immature *T. pyri*. However, when these reached adult (2nd generation, late November-early December) oviposition was poor and did not increase progressively, as expected (Figure 3a). The total number of *T. pyri* per cluster declined steadily until mid-December (Figure 3d). Despite initially higher numbers of *T. pyri* present in the metiram treatment (Figure 3d), predators were more numerous in the captan treatment from early December until late January.

Mancozeb treatment: The overwintered *T. pyri* females laid eggs during October but, in comparison with metiram treatment, immature stages were rarely found in samples taken during late October and November. In early November most of the

Fig. 3a, 3b and 3c: The proportion of the *T. pyri* population on apple leaf and fruit clusters consisting of eggs (■), immatures (XXXX) or adult (///) stages in each of metiram, mancozeb and captan treatments respectively.

overwintered females had died and the population was mainly in the egg stage. The proportion of immature stages was under-represented compared with both metiram and captan treated samples. Subsequently the number of *T. pyri* per cluster declined and, from late November until late summer, their number remained low (Figure 3d).

Captan treatment: Numbers of *T. pyri* were very low until mid-November when most were immature stages as in the metiram treated trees. As this spring generation of predators matured, the increasing proportion of adults was followed by an increasing proportion of eggs. Unlike metiram treated trees, there was no obvious suppression of any stage at the start of the second generation in late November. Consequently there was a rapid increase in the number of *T. pyri* per cluster from early December (Figure 3d).

The effect of each fungicide programme on *T. pyri* numbers in Trial 3 is summarised in Table 2. The mean numbers of *T. pyri* per cluster in early October are compared with samples taken in December while ERM populations were still uniformly low in each treatment. These data show that there was no significant increase in predator numbers during this period in either the metiram or mancozeb programmes. In contrast, the increase in numbers of *T. pyri* per cluster in the captan treatment was highly significant.

Fig. 3d: The mean number of *T. pyri* per cluster (all life stages) in each of the metiram (□—□), mancozeb (+—+) and captan (■—■) treatments.

TABLE 2: Comparison between the mean numbers of *T. pyri* per cluster in the first two samples taken in October and two samples taken in mid-December.

Fungicide	Mean no. <i>T. pyri</i> per cluster		P value
	October	December	
metiram	0.53	0.70	0.45 NS
mancozeb	0.20	0.15	0.64 NS
captan	0.17	1.82	0.0002 ***

DISCUSSION

The data from Trials 1 and 2 clearly indicate that both mancozeb and metiram suppressed the seasonal development of *T. pyri* populations when compared with captan. The suppression was related to either the period of use or the total number of applications; greater suppression occurred with increasing use. Suppression persisted for 4-5 weeks after the final application. Sensitivity analysis with the ERM/*T. pyri* simulation model of Hayes (1989) has shown that even a small reduction in *T. pyri* numbers in early summer may seriously disrupt IMC in late summer (Hayes *et al* 1989). This conclusion is supported by field data here which showed significant disruption of mite populations in mid-late summer after just four mancozeb or metiram applications.

The detailed effect of metiram and mancozeb on *T. pyri* populations in Trial 3 was more difficult to determine. There appeared to be little direct toxicity to *T. pyri* apparent in laboratory studies. Field results, assessing the impact on life stages of *T. pyri*, suggest that suppression is probably due to cumulative and delayed effects of exposure to metiram and mancozeb.

There was little obvious effect of repeated exposure to metiram on overwintered female *T. pyri*, their oviposition or subsequent egg hatch. The reasons for variable and low proportions of *T. pyri* eggs in late November and December were investigated using

the simulation model of Hayes (1989). The *T. pyri* population in the metiram treatment was simulated throughout November and December based on the number of stages present in late October. This revealed a difference between the field data and the simulation in the number of second generation adults. Field sampling found only 50% of the expected numbers in late November. Similarly, simulations of the *T. pyri* population in the mancozeb treatment suggested there were greatly reduced numbers of second generation nymphs in the field than could be expected. In contrast, simulation of the *T. pyri* population in the captan treatment found good agreement with field data.

This therefore suggests that repeated metiram applications affected the developing, immature stages of the first generation of *T. pyri* in early summer. Subsequently, adults were less numerous than predicted, which may explain the lower proportions of eggs present in the metiram treatments in late November and early December. Repeated exposure to mancozeb also appeared to affect immature *T. pyri* developing from eggs laid by the overwintering females. These immature stages appeared to die as young nymphs and the toxic effects were more apparent than in the metiram treatment.

All three trials confirm that mancozeb and metiram suppression of *T. pyri* is the primary reason for disruption of IMC by these fungicides. The effect of cumulative exposure of *T. pyri* populations to these fungicides is an increasingly uncoupled interaction with ERM as the season progresses. This inevitably leads to ERM outbreaks in mid and late summer.

Laboratory bioassays have identified relatively small toxic and behavioural effects on *T. pyri* caused by these fungicides (Baynon and Penman 1987; Hayes *et al* 1989). These field studies show that repeated applications result in more significant effects. Field studies also found mancozeb to be slightly more toxic to *T. pyri* than metiram. This result contrasts with the conclusions drawn in laboratory bioassays and suggests that delayed toxic effects are more significant with mancozeb than metiram. This study therefore highlights the problem of identifying subtle delayed, or cumulative toxic, effects of pesticides on *T. pyri*, and the consequence on IMC, based solely on laboratory bioassays.

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