

## Technical notes

### Evaluation of fungicides for the control of kikuyu yellows (*Verrucalvus flavofaciens*)

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Kikuyu yellows is the most important disease of kikuyu grass (*Pennisetum clandestinum* Hochst. ex Chiov.) in New South Wales and Queensland (Allen *et al.* 1975, Wong 1975). The disease is caused by a soilborne oomycete, *Verrucalvus flavofaciens* Wong and Dick (Dick *et al.* 1984, Wong 1975). The pathogen invades the roots of the grass and becomes systemic in the stem and leaves. The infected plants turn a bright yellow colour and subsequently die. The disease occurs as scattered patches, enlarging to several metres in diameter. The death of the grass in the centre of diseased patches results in weed invasion and the decline in productivity of the kikuyu pasture. Kikuyu yellows has also become a serious disease in home lawns on the central coast and in the north-west of New South Wales.

There are no entirely satisfactory methods to control the disease. Present recommendations by the New South Wales Department of Agriculture and Fisheries include killing the affected pastures with herbicides and re-sowing after a fallow period of a year. Recently, a new kikuyu cultivar, Noonan, has shown field tolerance to the disease (Wong and Wilson 1983). Affected paddocks may now be re-sown with this disease-tolerant cultivar to reduce the risk of severe disease in the new pasture. However, Noonan is an upright grass, which is unsuitable as lawn grass and other measures are required for the control of the disease in home lawns. A study was undertaken to investigate the effect of a range of fungicides, particularly those with systemic activity against oomycetes, on kikuyu yellows.

#### Materials and Methods

##### Fungicides

For *in vitro* studies, the active ingredients (technical grade) were used. The systemic fungicides were metalaxyl, furalaxyl, propamocarb and triadimefon. The non-systemic fungicides used were etridiazol, vinclozolin and quintozone. For glasshouse and field experiments, triadimefon (Bayer, Bayleton 25% WP) was used.

##### *In vitro* Experiment

The isolate of *V. flavofaciens* used was an isotype culture (DAR 55947). The fungus was plated on one-quarter strength PDA and incubated at 25°C for 6 days. Fungal discs (7 mm diam.) were taken from the margin of the colonies and placed in the centre of plates of one-quarter strength PDA or cornmeal agar (Difco) amended with 0, 1, 10 and 100 ppm of the active ingredient of the fungicides. There were 4 replicates per treatment of each chemical. The plates were incubated at 25°C and the diameters of the colonies were measured after 4, 5 and 6 days. After 14 days, the production of sporangia and oospores was recorded.

##### Glasshouse Experiment

As preliminary pot experiments had shown that only triadimefon was effective in controlling kikuyu yellows, this experiment was carried out to compare the effectiveness of various rates of triadimefon. The rates of application are shown in Table 2.

Clay pots (13 cm diameter) were filled to within 3 cm of the top with pasteurized potting mix (1:1 topsoil : sand, pH 7.0) and one 1/4 PDA plate of actively-growing *V. flavofaciens*, chopped up into 1 cm<sup>2</sup> pieces, was placed on the surface of the potting mix. The inoculum was covered with a 1 cm layer of potting mix. Three healthy runners of kikuyu (10 cm long) were planted per pot. There were three replicates for each treatment.

The fungicide was applied as a soil drench (100 ml/pot) immediately after planting. Controls received an equivalent amount of water. The pots were randomized in blocks in a glasshouse at 25° - 30°C. The pots were watered daily to field capacity. After 4 and 8 weeks the number of shoots bearing one or more yellow leaves per pot was recorded.

##### Field Experiment

The experiment was conducted on a home lawn with severe kikuyu yellows disease at Tamworth, N.S.W. The disease had been inadvertently and uniformly distributed through the lawn by regular lawn-mowing. Plots measuring 2 m x 1 m were pegged out in 3 blocks and the treatments randomized in the blocks. The following treatments were applied in early spring when the grass had resumed growth after winter dormancy:

- (1) triadimefon at 60 g a.i. 100 m<sup>-2</sup> once
- (2) triadimefon at 60 g a.i. 100 m<sup>-2</sup> every 4 weeks
- (3) triadimefon at 120 g a.i. 100 m<sup>-2</sup> once
- (4) triadimefon at 120 g a.i. 100 m<sup>-2</sup> every 4 weeks and
- (5) no chemical.

The lawn was mown close to the ground (3 cm) and the chemicals were applied as a drench. The lawn was then fertilized with a commercial lawn fertilizer (Multigro<sup>®</sup>) and watered as required with a sprinkler. The percentage area of each plot showing symptoms of yellow leaves was recorded after 4, 8 and 12 weeks.

#### Results

##### *In Vitro* Experiment

Of the 7 fungicides, triadimefon prevented all growth of *V. flavofaciens* at 10 ppm (Table 1). Of the systemic fungicides with known activity against oomycetes, furalaxyl and metalaxyl inhibited growth at 100 ppm but were ineffective at 10 ppm. Propamocarb, vinclozolin and quintozone were ineffective at these rates.

In unamended plates, sporangia and oospores were formed after 14 days incubation at 25°C. Triadimefon inhibited production of both sporangia and oospores at 1 ppm. At 10

Table 1 Effect of fungicides on the growth of *V. flavofaciens* on agar plates

Fungicide	Mean radial growth rate (mm day <sup>-1</sup> ) at 25°C*		
	1 ppm	10 ppm	100 ppm
Triadimefon	3.0 ± 0.0**	0.0	0.0
Metalaxyl	7.0 ± 0.0	6.0 ± 0.0	0.0
Furalaxyl	5.0 ± 0.0	4.0 ± 0.0	0.0
Etridiazole	6.0 ± 0.0	3.0 ± 0.0	0.0
Quintozone	5.8 ± 0.4	3.3 ± 0.4	2.0 ± 0.0
Propamocarb	7.0 ± 0.4	5.8 ± 0.4	3.5 ± 0.9
Vinclozolin	6.0 ± 0.0	5.0 ± 0.0	4.0 ± 0.0

\* Unamended agar = 7.0 ± 0.0 mm/day.

\*\* Standard error of mean.

**Table 2 Effect of rates of triadimefon on kikuyu yellows in a pot experiment**

Treatment	Mean number of diseased shoots per pot at	
	4 wks	8 wks
Uninoculated control	0	0
No chemical	4.7 ± 2.3*	11.7 ± 2.4
Triadimefon (15 g a.i. 100 m <sup>-2</sup> )	0	3.0 ± 1.6
Triadimefon (30 g a.i. 100 m <sup>-2</sup> )	0	2.0 ± 1.0
Triadimefon (60 g a.i. 100 m <sup>-2</sup> )	0	0
Triadimefon (120 g a.i. 100 m <sup>-2</sup> )	0	0

\* standard error of mean

ppm, all the other fungicides except quintozene permitted the production of oospores. In addition, sporangia were produced in the presence of 10 ppm of metalaxyl and furalaxyl. At 100 ppm of chemicals, oospores were still produced in plates with propamocarb and sporangia were produced in plates with quintozene.

#### Glasshouse experiment

At 4 weeks, none of the plants in pots treated with triadimefon were diseased (Table 2). At 8 weeks, there was no disease with 60 g a.i. 100 m<sup>-2</sup> or 120 g a.i. 100 m<sup>-2</sup>. There was disease at the lower rates, but the amount of disease was significantly less ( $P < 0.05$ ) than that present in inoculated pots not treated with the chemical.

#### Field experiment

At 4 weeks, disease symptoms were present over the whole of the untreated plot areas as well as in the plots treated with triadimefon at 60 g a.i. 100 m<sup>-2</sup>.

In plots treated with twice this rate, only 50% of the plot areas were diseased. At 8 weeks, the plots treated with one application of triadimefon at 120 g a.i. 100 m<sup>-2</sup> were completely diseased while the plots, which were treated twice at this rate, showed disease over 80% of the plot areas. At 12 weeks, all plots were completely diseased.

#### Discussion

Although several chemicals inhibited *V. flavofaciens* on agar plates (Table 1), only triadimefon controlled the disease in pot experiments (Table 2). This is surprising since triadimefon is not reputed to have any activity against oomycetes. The effectiveness of triadimefon in suppressing *V. flavofaciens* may not only be due to its inhibition of mycelial growth but also of sporulation and oospore production. However, it is not known whether these structures are suppressed in the soil. The infection process of this pathogen has not been studied and requires examination.

The attempt at field control of the disease on a home lawn failed. The reasons for not obtaining results in the field comparable to those in the glasshouse are not known. Higher rates of triadimefon were not used because they would not have been economic even on a home lawn. It appears at this stage that chemical control may not be feasible and attempts at selecting for disease-tolerant cultivars of kikuyu with suitable turf characteristics may be more worthwhile.

#### References

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