BOYSENBERRY DECLINE: IDENTIFICATION AND CONTROL OF A SERIOUS DISEASE OF BOYSENBERRIES

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ABSTRACT

The fungus *Cercosporella rubi*, which causes rosette of boysenberry, was identified in 1998 as the cause of boysenberry decline in New Zealand. A fungicide trial was carried out in the summer of 1998/99 to determine the efficacy of several fungicides for controlling rosette. The fungicides azoxystrobin, benomyl, bitertanol, difenconazole, mancozeb and pyrimethanil were each applied five times to boysenberry primocanes in a commercial planting of boysenberries from November through to February. Difenconazole was the most effective fungicide for reducing rosette symptoms, while azoxystrobin and benomyl also gave good control of the disease. This trial has demonstrated that several fungicides from different chemical groups could be used for control of rosette.
Keywords: *Cercosporella*, control, boysenberry, rosette.

INTRODUCTION

Boysenberry decline was first noted in New Zealand in the early 1980s, and caused the demise of boysenberry growing in the Auckland district. It also seriously affected production in the Bay of Plenty, Waikato and Taranaki, and spread into the main production region of Nelson (Wood et al. 1999). For much of this time, the cause of the disease was thought to be a phytoplasma (Wood & Charles 1989; Wood et al. 1992; Wood & Langford 1996). The symptoms included stunted multiple shoots emerging from individual buds (witches broom) and deformed flowers from which fruit did not develop normally.

In spring 1998, some boysenberry flower samples from Whakatane with rosette symptoms were sent for diagnosis of a possible *Cercosporella rubi* infection (rosette) to the MAF National Plant Pest Reference Laboratory at Lincoln. Examination of these flowers confirmed the presence of *C. rubi*. This identification was validated (Dr E. McKenzie, pers comm.) and a sample was lodged in the Landcare Herbarium (PDD 69804). Subsequent testing of additional samples confirmed the original identification (Wood et al. 1999).

Previous trials on the control of *C. rubi* in USA indicated that benomyl was likely to be effective for controlling the disease (Moore et al. 1977; Marroquin et al. 1990). Based on this information, a trial was established to determine whether other chemicals might be effective in controlling rosette in boysenberry.

MATERIALS AND METHODS

A trial was carried out on the property of Mr Richard Somerfield, in the Bay of Plenty region of New Zealand, on a boysenberry block severely affected by boysenberry decline.

The fungicide treatments and rates of application used in the trial are shown in Table 1. Each fungicide was applied five times in the 1998/99 season (26 November, 10 and

21 December, 6 January and 4 February) onto the primocanes only of boysenberry plants. The fungicide treatments were applied using the growers' standard spraying equipment. There were four replicates for each treatment, in a randomised complete block design, with one row representing a treatment in each replicate. Most rows consisted of 21 bays of two plants per bay. However the last replicate consisted of seven rows, because of the shape of the block, and the number of bays per row in this replicate ranged from 13 to 18.

TABLE 1: Fungicide treatments applied to rosette-affected boysenberries in a field trial.

Chemical	Product name	Rate (g ai/100 litres)
Mancozeb	Mancozeb	160
Benomyl	Benlate®	25
Bitertanol	Baycor®	25
Difenoconazole	Score®	5
Pyrimethanil	Scala®	30
Azoxystrobin	Amistar®	12.5
Untreated	-	Nil

The following spring (9 September 1999), assessments were made of the buds affected by rosette in two ways:

1. In bays two and four of each row, the number of buds showing symptoms of rosette (i.e. multiple shoots) and the number of buds showing normal shoots, were counted for all canes above the middle wire. From this, the proportion of buds showing rosette symptoms, were calculated.
2. For each bay in all rows, a score was given representing the estimated proportion of buds showing rosette symptoms. Before starting the scoring, bays two and four were scored and the score was compared with the actual counts from bud counts (above), to ensure scoring accuracy. To provide a cross check of the scoring system, two people scored all bays independently on two of the replicates.

The rosette severity scores used were:
1 = <5% buds with symptoms
2 = 5-20% buds with symptoms
3 = 20-40% buds with symptoms
4 = 40-60% buds with symptoms
5 = >60% buds with symptoms

RESULTS AND DISCUSSION

The mean proportions of buds with rosette symptoms from the bud counts in bays two and four for the different fungicide treatments are shown in Table 2. The mean scores representing severity of rosette symptoms in all bays for the different treatments are also shown in Table 2. Both sets of results show clearly that there was a statistically significant reduction in disease from application of most of the fungicides ($P<0.001$ and $P=0.001$ for bud counts and severity scores respectively).

Considering both sets of data, difenconazole was the most effective of the fungicide treatments for control of rosette, with benomyl, azoxystrobin and bitertanol next most effective. The others, particularly pyrimethanol and possibly mancozeb, were less effective, but all of the fungicides tested gave some degree of control of the disease. The effectiveness of several fungicides will allow two or three effective chemical fungicides to be used in alternate applications. This should reduce the likelihood of developing insensitivity in *C. rubi* to particular fungicides. Most boysenberry growers in New Zealand have not used benomyl for many years because resistant *Botrytis* strains had developed and this probably contributed to the spread of decline. Benomyl has now been withdrawn from the New Zealand market, but alternative benzimidazoles are likely to be effective.

Whilst different chemical rates were not investigated in our trial, recommended label rates were used. The rate for benomyl was within the range shown to be effective for control of rosette in the USA (Marroquin et al. 1990). In our trials, fungicides were

applied only to the primocanes whereas fungicides were applied to whole plants in the USA trials (Moore et al. 1977; Marroquin et al. 1990). It appears that targeted application was as effective as a blanket spray application. Target application has the advantage of applying smaller amounts of fungicide per hectare and has potential for reducing or eliminating any chemical residues in fruit that might be suitable for harvest in the season when fungicide is applied.

TABLE 2: Mean proportion of buds (%) showing boysenberry decline symptoms in bays 2 and 4, and mean rosette severity scores for all bays in boysenberries treated with different fungicides in a field trial.

Treatment	Proportion of buds infected	Rosette severity score
Benomyl	17	2.5
Difenconazole	18	2.2
Azoxystrobin	25	2.3
Bitertanol	25	2.8
Mancozeb	35	2.5
Pyrimethanol	42	3.9
Untreated	69	4.5
Overall mean	33	3.0
SED	6.6	0.48
P-value	0.000	0.001

Several growers in the Bay of Plenty region of New Zealand, who were on the verge of discontinuing with boysenberry production, have since been able to control boysenberry decline sufficiently to enable them to continue growing the crop. Whilst blackberry breeding in the southern USA focuses on development of rosette-resistant varieties (Gupton 1999), New Zealand growers consider that boysenberry decline is sufficiently under control to render incorporation of plant resistance to this disease as a low priority in the New Zealand boysenberry breeding programme.

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