CONTROL OF BLOSSOM BLIGHT IN STONEFRUIT WITH DIFENOCONAZOLE

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Blossom blight cause by *Monilinia fructicola* (Wint.) Honey is a common disease of stonefruit in New Zealand. Blossom blight is the first stage of attack by the disease with blossoms susceptible at any stage of flowering. All blossom parts are susceptible and the whole flower is rapidly enveloped by the fungus. The second stage is on the maturing fruit and will occur if weather conditions are warm and wet. The severity of fruit rot is influenced by the extent of blossom blight. Control measures are based on the general strategy of the use of fungicides at blossom and pre- and post-harvest along with good orchard hygiene. The strategy being that DMI fungicides, preferably in mixture with a protectant fungicide, are applied a maximum of three times over flowering up to shuck fall, with non-DMI fungicides to be used at other times (Prince *et al* 1989). Elmer and Gaunt (1986) reported on the development of the insensitivity of *Monilinia fructicola* to dicarboximide and benzimidazole fungicides. This has led to research into other chemical groups that may be used for blossom blight and brown rot control. A range of DMI fungicides was tested by Gawith and Tate (1989) for efficacy against blossom blight in nectarines. They showed that all triazoles tested were effective with some significantly better than the standard DMI triforine. In this series of trials we report on further developments with difenoconazole (Score) for the control of blossom blight in stonefruit.

Two trials were undertaken in 1988 in Hawkes Bay and Waikato orchards and a single trial was conducted in Hawkes Bay in 1990. The experimental design was a randomised complete block with plots of single trees and four-six replicates. In 1988 Redhaven peaches were used in the Waikato and Golden Queen peaches in the Hawkes Bay. Firebrite nectarines were used in the Hawkes Bay in 1990.

Four fungicide applications were made at each site to cover the blossom period in 1988 and three in 1990 (Table 1). Applications were made to run-off (500-1000 litres/ha) using a CO_2 pressurised sprayer with solid cone nozzles at 340-500 kPa. Spraying was carried out during calm conditions. The use of small trees and, where necessary, polythene screens, prevented contamination of neighbouring trees. All treatments (Tables 2 and 3) were applied with the addition of a wetting agent (Citowett or Multifilm X-90) at 0.25% v/v conc. Assessments were carried out on 10 laterals/tree for infected blossoms 4 to 5 days after the last application. Monitoring of the tree for growth effects was also carried out 3-4 weeks after the last application.

Blossoming laterals were taken from the field trials in Hawkes Bay in 1988 and 1990 and placed in a controlled environment using the method outlined by Gawith and Tate (1989). The procedure in 1988 was that two to six blossoming laterals were cut

TABLE 1: Date and flowering stage of crop when fungicides were applied in 1988 and 1990.

Hawkes Bay		Waikato
1988	1990	1988
30 August popcorn	22 August late popcorn	5 September early bloom
7 September early bloom	31 August full bloom	15 September full bloom
16 September full bloom	10 September petal fall	26 September early petal fall
29 September petal fall		7 October petal fall

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from each plot to achieve a minimum of 120 blossoms per treatment. The laterals were taken 3 days after application of fungicides. Two mm of rain had fallen. The laterals were placed in a container of water, all blossoms were inoculated with *M. fructicola* pure culture suspension (2-3 x 10^5 conidia/ml) and enclosed in a plastic bag to encourage high humidity. They were then placed in a laboratory at an average temperature of 20 °C for 8 days. A similar method was carried out in 1990 with the following modifications. The blossoming laterals were taken 48 hours after treatment. Eight mm of rain had fallen in the interim. They were placed in an Oasis block standing in a container of water, inoculated, bagged and placed in a glasshouse at an average temperature of 16 °C for 8 days.

Assessments of the amount of blossom blight in the controlled environment tests were made following the latent period. The total number of blossoms were counted and each blossom placed in a diseased or clean category. Statistical analysis was by analysis of variance with mean separation by Duncan's multiple range rests ($P<0.05$, $P<0.01$) with arcsine transformation where appropriate.

In 1988 the level of disease in the Hawkes Bay field trials was very low but there was a moderate level of natural infection in the Waikato orchard with a high level of variability between replicates (Table 2). Difenoconazole gave comparable levels of control of blossom blight to procymidone at all the rates tested. Both fungicides reduced the level of disease observed compared with untreated. No symptoms of blossom burning or irregular growth effects on the foliage were noted in any trial.

TABLE 2: Percentage of blossoms infected with *Monilinia fructicola* in the field (Waikato) and on inoculated laterals in a controlled environment (H. Bay) in 1988 after fungicide application.

Treatment	g ai/100 litres	% infection Waikato	H. Bay
difenoconazole (Score WP)	10	2.9 bB*	18.0 bB
difenoconazole	15	9.0 abAB	7.2 cB
difenoconazole	20	7.1 bAB	9.0 cB
procymidone (Sumisclex 25)	37.5	5.1 bB	13.4 bcB
untreated	—	14.5 aA	74.6 aA

*Means in each column followed by the same letter are not different ($P<0.05$ in lower case, $P<0.01$ in capitals).

The controlled environment tests produced good infection levels in the untreated (Tables 2 and 3), similar to the levels of disease reported by Gawith and Tate (1989) using the same method. There was little petal or blossom fall during the latent period, especially in 1990, probably due to the high natural light levels. All the chemicals tested gave a reduction ($P<0.01$) in the level of disease. In 1988 difenoconazole at 10-20 g/100 litres was equivalent to the standard promycidone (Table 2). Difenoconazole at 15 and 20 g/100 litres gave a lower level of disease than did the 10 g/100 litre rate ($P<0.05$). In 1990 difenoconazole was equivalent to triforine for blossom blight control (Table 3).

TABLE 3: Blossom infection with *M. fructicola* in the Hawkes Bay orchard on inoculated laterals in a controlled environment after fungicide treatment of nectarine trees in a Hawkes Bay orchard in 1990.

Treatment	g ai/100 litres	% infection
difenoconazole	10	6.9 cC
triforine (Saprol EC)	19	9.2 cC
iprodione (Rovral FL)	37.5	13.8 bB
untreated	—	68.3 aA

*Means in each column followed by the same letter are not different ($P<0.05$ in lower case, $P<0.01$ in capitals).

Both DMI fungicides gave a higher level of disease control ($P<0.01$) than the protectant iprodione.

Difenoconazole at 10-20 g/100 litres gave good control of blossom blight in the field and under controlled environmental conditions. The efficacy of difenoconazole was comparable to procymidone and triforine but better than iprodione.

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