FIELD EVALUATION OF A DISEASE MANAGEMENT SYSTEM FOR PEACH BROWN ROT IN HAWKES BAY

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SUMMARY

A disease management system (DMS) for peach and nectarine brown rot (*Monilinia fructicola* Wilson, Honey) control was evaluated over two seasons. The DMS utilised disease risk assessments based on crop, pathogen, disease and infection period monitoring and forecasts of infection periods. Risk assessments were used to determine the need for fungicide sprays and when these should be applied. Field experiments comparing grower with DMS spray programmes, were conducted to test and refine the DMS. In three experiments, disease risk identification improved spray timing or resulted in fewer spray applications over blossom, without compromising disease control at harvest. The DMS was shown to be a feasible alternative to continuous cover spraying over blossom.

Keywords: disease management systems, peaches, nectarines, brown rot, *Monilinia fructicola*

INTRODUCTION

This paper describes the results of two seasons evaluation of a disease management system (DMS) for brown rot in peaches and nectarines. DMS rationale and development approach was discussed in Manktelow and Tate (1991) and Kable (1991). Hawkes Bay orchards, while being regarded as setting a high standard of orchard management, tend to rely on intensive use of pesticides for disease control. There are however, large variations in pesticide usage between orchards that do not reflect differences in biological spraying requirements (K.G. Tate, unpublished data). The Hawkes Bay climate is relatively dry and does not always favour disease development. Given these factors, there appeared to be potential to reduce overall brown rot fungicide use, while refining spray scheduling to improve, or at least maintain, disease control.

MATERIALS AND METHODS

Experimental design

Field trials were established on two properties on 8-year old Golden Queen peach trees (Orchard A, 1990-1992; spacing 1.2 x 4.5 m) and Fantasia nectarines (Orchard B, 1991-1992; spacing 2.5 x 4.5 m). Treatments were applied to two blocks of approximately 300 trees each. In each block fifteen trees ('monitoring sites') were tagged for intensive monitoring of pathogen, disease and crop development. At each site the grower's protective spray programme (standard) was compared with the DMS responsive programme (reduced). All fungicides were applied by the grower in high volume (1,000-2,000 litres/ha) airblast sprays. Treatment blocks were buffered from adjacent airblast sprays by at least three rows. Fungicides selected for the reduced programmes matched those used in the standard grower programme (Fig. 1).

Pathogen, disease and crop monitoring

Percent open flowers were assessed at each monitoring site at 3-4 day intervals (Fig. 1). At the same time, mummified fruit, in the trees or on the ground, and overwintered twig cankers and peduncles on trees were sought as potential inoculum sources. When apothecia were detected their numbers, position and stage of development were recorded. Presence of contaminating spores on open flowers was determined by collecting 25 open blossoms per monitoring site, atomising with distilled water and incubating in enclosed, moistened food trays for 5 days at 15-20°C.

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Blossom and twig blight incidence were assessed at each monitoring site at late bloom and after bloom respectively. In 1990-91 brown rot and split fruit incidence were estimated at harvest from a single sample of 100 fruit per monitoring site. In 1991-92 these assessments were made over the harvest period on all fruit in and beneath monitoring sites. After the 1991-92 harvest, 900 fruit from each treatment in Orchard A were coolstored for 6 days while awaiting processing, then assessed for incidence of brown rot. In orchard B, 40 fruit from each monitoring site were held at ambient temperatures (20-25°C) and likewise recorded (rotting fruit were removed before sporulation commenced).

Infection period forecasting/monitoring

Each day newspaper and television weather situation and forecast maps were examined and the potential of approaching weather systems to produce significant rain assessed by applying a set of prediction rules (K.G. Tate, unpublished). Leaf wetness and air temperature were recorded on hempstring-type leaf wetness-temperature recorders in weather screens on or near each orchard site. An infection period was deemed to have occurred if the product of leaf wetness duration (hours) and mean temperature (°C) exceeded 100 (adapted from Weaver 1950).

Fungicide scheduling/use

Standard spray timing depended on each grower's concept of how frequently, and when, sprays should be applied to maintain a protective cover during critical growth stages. Fungicides and rates applied were chlorothalonil (Bravo) at 115-150 g/100 litres, triforine (Saprol) at 19 g, bitertanol (Baycor) at 25 g and flusilazol (Nustar) at 4 g/100 litres. In the reduced programme infection weather prediction rules, monitored infection periods, rainfall since the previous spray, percent flower opening and disease levels were used to assist in spraying decisions. Sprays were generally applied just before expected infection periods and omitted in the intervening dry periods or when cold fronts were expected.

Fungicide scheduling comparisons were made between treatments at each site in relation to infection period occurrence and disease control. Overall fungicide use was compared with average district use, which was calculated from at least twenty other properties each season.

RESULTS

Pathogen, disease and crop monitoring

Apothecia, present from the first open flower, were the only primary inoculum sources observed to produce spores during blossom. Apothecia were found on the weedy verges of blocks on Orchard A and scattered throughout Orchard B in denser weedy patches under the trees. Blossom incubations indicated that spores had contaminated unequal proportions of flowers from each treatment (Table 1). Blossom blight incidence was low or nil in each treatment, except in the standard treatment on Orchard B (1991-92). In each year blighted blossoms developed into twig cankers.

Brown rot incidence at harvest was comparable between treatments at each site, but overall levels ranged from about 1 to 12% between sites/seasons (Table 1). On Orchard B (1991-92) the reduced treatment had a lower incidence of twig blight than the standard, but a slightly higher incidence of harvest fruit rot (Table 1).

Infection period forecasting/monitoring

The 1990-91 (Orchard A) blossom period was relatively dry and infection periods occurred only at the beginning of flowering and after 100% petal fall (Fig. 1). In the intervening period cool, showery, southwesterly conditions persisted, with five brief rainfalls recorded. After bloom the summer was relatively dry until the harvest period, when four infection periods were recorded. In contrast, the 1991-92 bloom period commenced with warm dry weather causing rapid flower opening, with two marginal infection periods at mid bloom and four significant wet periods at late bloom (Fig. 1). The rest of the season was relatively wet with a total of 28 infection periods recorded, compared with 12 in 1990-91.

TABLE 1: Inoculum, brown rot and sprays applied in three spray programme comparisons.

Data Obtained	Means ($\pm$ SE) of records from monitoring sites					
	Orchard A 1990		Orchard A 1991		Orchard B 1991	
	Standard	Reduced	Standard	Reduced	Standard	Reduced
% spore contam. bloss.	11.7(1.9)	32.1(2.9)	1.3(0.6)	5.1(1.1)	32.0(3.2)	14.4(2.3)
% blossom blight	0.2	0.3	0	0	2.1(0.5)	0.8(0.3)
No. twig cankers per site	1.1(0.3)	1.3(0.5)	0	0	6.1(1.1)	2.2(0.5)
% split fruit	7.9(1.3)	9.5(1.7)	7.2(1.4)	7.9(0.7)	9.0(1.3)	10.6(0.9)
% harvest rot	0.5(0.1)	0.9(0.2)	12.1(1.6)	11.9(0.8)	4.6(0.7)	6.2(0.5)
% storage rot	3.0	2.3	1.1	1.3	45.0	36.8
No. blossom sprays	5	3	6	4	5	4
Total no. of sprays	8	6	11	8	8	7

Fig. 1: Spray timing in relation to infection periods during blossom, Orchards A and B, 1990 and 1991. Arrows indicate elapsed time from spraying to potential infection periods.**Fungicide scheduling/use**

In 1990-91 on Orchard A, five blossom blight sprays were applied in the standard treatment compared with three in the reduced. Over the blossom period there was an interval of 21 days between sprays in the reduced treatment (Fig. 1). While eight official forecasts for rain occurred over this period, only two resulted in rain and both were brief showers. Our infection prediction rules enabled us to withhold DMS sprays

for its duration and the final spray after 100% petal fall was only applied because of the presence of twig blight. Sprays were not particularly well coupled to infection periods in either programme.

In 1991-92 on Orchard A, both treatments completely controlled blossom blight, with two fewer sprays applied to the reduced programme block (Table 1). One less application was made in the Orchard B reduced treatment and sprays were applied closer to the start of infection periods than standard sprays (Fig. 1).

In the preharvest period for the standard treatment, both growers opted to follow the spray scheduling methodology used for the reduced programme. Therefore, no treatment comparisons were possible, except in 1990-91 (Orchard A), where an additional standard spray was applied when rain fell soon after application.

Over the three comparisons reduced programmes saved an average of two sprays (Table 1), with less than a 1% difference in brown rot at harvest. On average over both seasons nine applications were made on other orchards, compared with an average of seven to the reduced treatments and nine to the standards.

DISCUSSION

These trials were established to obtain data required to build and refine a DMS for stonefruit brown rot and to field test the system at its present stage of development. A problem in commercial scale trials lies in balancing trial logistics and cost with the need for adequate replication. Due to risks of airblast spray drift and the growers mis-applying sprays, we opted to use single, large, uniform adjacent blocks, with data collected from many monitoring sites in each block. The lack of within site replication presented some problems, especially where apothecial production was not uniformly distributed. However, we ultimately have to prove the DMS approach on many sites/seasons and comparisons between the three trials to date provide a form of treatment replication.

Pathogen monitoring in both orchards suggested that ascospores, produced by apothecia arising from buried fruit mummies, were the source of primary bloom inoculum. No conidial production was observed from overwintered mummies, twig cankers or fruit peduncles during intensive monitoring over the spring period of the seasons under study. These observations support those made in Hawkes Bay by J.B. Corbin (unpublished data) in 1969. DMS apothecial observations indicated similar patterns of emergence and discharge to those reported by Tate (1979) from the Horowhenua district. Observations of the spatial distribution of apothecia suggested that orchard floor treatments (e.g. close mowing or superficial ground disturbance) could be applied to eradicate, or at least greatly reduce, apothecial/ascospore production. Experiments to evaluate potential treatments and their benefits for disease control are planned.

The period of apothecial emergence was closely coupled to blossom opening and fine weather release of ascospores was observed (Tate 1979). This has important implications for blossom blight control. With regular release of ascospores, filaments of freshly opened blooms would soon become contaminated with spores, allowing germination to commence at the start of the next wet period. This could allow infection to occur from relatively short, even overnight, wet periods. Differences between blocks in percent spore-contaminated blossoms would have resulted partly from differences in apothecia distribution in each block and partly from the timing of flower sampling in relation to prior blossom sprays. In 1990, flowers were sampled on September 4 and only the former factor applied, while in 1991, sampling on August 29 meant that spray timing also applied (Fig. 1).

In the orchard B (1991-92) standard programme, despite more sprays being applied over blossom, more blossom blight resulted than on the reduced programme. This difference can be attributed to the higher level of viable spore contamination of blooms observed in the standard programme block, caused partly by the less precise spray timing that occurred in this treatment. Filaments in blooms that open after fungicide application are unlikely to be protected by non-systemic fungicides so they need to be applied as close as possible before the start of an infection period. Particular care

needs to be taken during the periods of rapid bloom opening (e.g. in the 1991-92 season, from August 22 to September 3, when 90% of blooms opened). Thus flower monitoring can be an additional disease risk assessment tool. Experiments planned for the 1992-93 season will combine bloom assessments, infection period forecasts and fungus monitoring to schedule bloom fungicide applications.

Harvest rot differences between treatments in each trial were comparable, but the higher levels of harvest rot (12%) in Orchard A, 1992, following virtually nil blossom blight was unexpected. However, a very low level of green fruit decay was observed in the Orchard A trial by 3 weeks preharvest, and this combined with 7-8% fruit split and five significant infection periods in the last few weeks may explain these levels of decay. If so it highlights the significance of even very scattered inoculum sources in a block by preharvest when other factors favour disease increase (Ogawa and English 1991). While blossom blight was not detected at any monitoring site, an exhaustive search through the blocks did reveal a trace. This was followed by a much wetter than normal fruit development period with 20 potential infection periods identified by 3 weeks preharvest, providing optimum conditions for twig blight and green fruit rot to develop. Spray timing during preharvest was generally good, but incidence of fruit split increased with each rainfall and this can expose even sprayed fruit to new infection. However, the additional spray after rainfall in the standard treatment did not improve control, suggesting that other factors such as dicarboximide resistance selection may also have been involved (Elmer and Gaunt 1990). Blossom blight control remains important in the DMS as heavy blossom blight development can result in major fruit losses at harvest (Tate and Corbin 1978).

Differences in postharvest rots observed in 1991-92 reflect different fruit handling and the reduced treatments did not compromise fruit storage life before processing (Orchard A) or fresh sale (Orchard B).

DMS spray timing currently relies on reliable prediction of rainfall that could result in infection. Bloom and pathogen monitoring reduce this dependence by indicating periods when disease risks are high, and hence when fungicide scheduling decisions must be conservative (risk adverse). However, the infection period forecasting system used has provided good results and is being pursued. Infection period prediction rules are being developed from observations/experience of 'infection weather' and disease monitoring on the Heretaunga Plains.

The rules aim to predict extended wet (infection) periods, rather than wet periods associated with showers. On September 4 and 13, 1991, marginal infection periods occurred as a result of showers that occurred at dusk, allowing trees to remain wet long enough for infection to occur. The first of these was not predicted by our forecasting rules. However, Wilcox (1990) demonstrated the potential for eradicant fungicide applications to control infection after short duration infection periods. Eradicant fungicide use is possible up to about 24 hours post infection, where disease is established in the filaments, but has not progressed into the receptacle. Eradicant applications would only be possible after infection periods of short duration.

The rule used to monitor infection period occurrence (Weaver 1950) worked well despite its crudity. On a number of occasions infection periods were confirmed by the appearance of blossom blight several days after these conditions were met.

Emphasis in DMS work to date has been placed on improving fungicide scheduling during the blossom period. We aimed to prevent blossom blight totally since any blighted blooms can provide the inoculum required to initiate mature fruit decay (Ogawa and English 1991). While blossom blight was not always eliminated, fewer sprays were used in our reduced programmes without increase in disease. Successful spray timing requires accurate prediction of infection periods and the application of fungicide just before the event to optimise fungicide efficacy. This approach assumed that a fungicide 'cover' was not necessary during dry periods, even if spores were present on blooms, since a moisture film is required for spore germination and infection (Wilcox 1989). While dew can provide the necessary moisture for this process, the duration of wetness is not usually long enough at the low (1-8°C) overnight spring temperatures experienced or even at harvest (Kable 1972). We therefore believe that the usual grower

practice of spraying after rain to re-establish fungicide cover is not efficient or necessary (unless aimed at eradication of filament infections).

Blossom spray timing in the reduced DMS treatments was generally better matched to infection period occurrence than those of the standard treatments and, at all sites, provided comparable blossom blight control with fewer applications. Despite this success, it was possible to identify areas where spray timing could have been improved. In the 1990-91 season the initial application in both programmes was made approximately 8 days too late (due to a delay in starting the experiment) and the final spray could have been avoided if the initial spray had been applied earlier. In the 1991-92 season, more attention to bloom phenology would have suggested that a spray application was justified on Orchard A when bloom opening peaked, so removing the need for the final application.

Currently the DMS protocol eliminates spray applications from the end of bloom until 3 weeks preharvest, provided blossom blight is prevented. We have not yet established rules/thresholds for spray scheduling in the event of different levels of blossom blight occurrence. During the preharvest period fungicide scheduling has been based on infection period forecasts. We found that both growers gradually adopted the reduced treatment spray scheduling system on their standard treatments. This, plus the reductions in bloom sprays achieved to date indicate the potential benefits to be gained from a DMS approach to brown rot control.

Whether growers will adopt the DMS will depend on their perception of the risk involved and the value of the crop. Indications are that process peach growers are reducing the number of sprays applied each season (Tate unpublished data) but that they will have to pay more attention to timing to maintain adequate control of brown rot. Extensive pathogen and disease monitoring was conducted in these trials. In a commercial DMS we envisage that the cost of this would be considerably reduced. A suitable bloom monitoring programme would include; 1) daily weather map surveillance, 2) continuous wetness and temperature logging, with field verification of infection and 3) monitoring flower opening and apothecial emergence at 3 to 4 day intervals. Between shuck fall and harvest, disease monitoring would occur after; 1) thinning, 2) occurrence of fruit splitting and 3) significant rainy periods. If blossom blight occurred it would be necessary to either prune out and destroy blighted twigs and/or adopt a weather-based protectant spray programme for the rest of the season. It is anticipated that much of the monitoring required could be cost effectively supplied to growers by independent, specialist consultants.

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REFERENCES

- Elmer, P.A.G., and Gaunt, R.E., 1990. Population dynamics of dicarboximide resistant strains of *Monilinia fruticola* and conservation of the dicarboximides for brown rot control. *Proc. 43rd N.Z. Weed and Pest Control Conf.*: 109-113.
- Kable, P.F., 1972. Brown rot of stone fruits on the Murrumbidgee Irrigation Areas. III. Influence of weather conditions during the harvest period on disease incidence in canning peaches. *Aust. J. Agric. Res.* 23: 1035-44.
- Kable, P.F., 1991. Crop disease management with fungicides - an overview of its origins, progress, current status and future development using modelling and climate data. *Plant Protection Quarterly* 6: 19-28.
- Manktelow, D.W., and Tate, K.G., 1991. Development of disease management systems (DMS) for Hawkes Bay pip and stone fruit production. *Proc. 44th N.Z. Weed and Pest Control Conf.*: 216-219.

- Ogawa, J.M., and English, H., 1991. Diseases of temperate zone tree fruit and nut crops. *Univ. Calif. Div. Agric. Nat. Res. Publ. 3345*. 461pp.
- Tate, K.G., 1979. *Sclerotinia fructicola* ascospore fluctuations in a peach orchard during blossom. *N.Z. J. Agric. Res.* 22: 355-60.
- Tate, K.G., and Corbin, J.B., 1978. Quiescent fruit infections of peach, apricot and plum in New Zealand caused by the brown rot fungus *Sclerotinia fructicola*. *N.Z. J. Exp. Agric.* 6: 319-25.
- Weaver, C.O., 1950. Effect of temperature and relative humidity on occurrence of blossom blight of stone fruits. *Phytopathol.* 40: 1136-53.
- Wilcox, W.F., 1989. Influence of environment and inoculum density on the incidence of brown rot blossom blight of sour cherry. *Phytopathol.* 79: 530-34.
- Wilcox, W.F., 1990. Post infection and antispore activities of selected fungicides in control of blossom blight of sour cherry caused by *Monilinia fructicola*. *Plant Disease* 74: 808-11.