BOTRYTIS STORAGE ROT OF KIWIFRUIT: EFFICACY OF PRE-HARVEST SPRAYS IN ORCHARDS WITH DICARBOXIMIDE-RESISTANT BOTRYTIS POPULATIONS

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ABSTRACT

A high frequency of resistance to dicarboximide fungicides was found in samples of *Botrytis cinerea* collected from many kiwifruit orchards in 1994. Experiments conducted during the 1994 season on two orchards with a high frequency and one with a moderate frequency of dicarboximide-resistant strains of *B. cinerea* determined the efficacy of pre-harvest dicarboximide applications in reducing incidence of botrytis storage rots. Treatments were water-sprayed and unsprayed, and a dicarboximide application. The water spray provided the greatest reduction in storage rot incidence in each trial, suggesting that the water on the surface of the fruit reduces infection, possibly by preventing transfer of spores from the fruit surface to the picking wound.

Keywords: *Botrytis cinerea*, kiwifruit, fungicide resistance, dicarboximide, efficacy

INTRODUCTION

Kiwifruit (*Actinidia deliciosa* (Chev.) C.F. Liang et A.R. Ferguson) become infected with *Botrytis cinerea* Persoon at the time of harvest and subsequent stem-end rots appear 4 to 12 weeks after fruit have been placed in cool storage (Pennycook and Manning 1993). Fruit are harvested from the vine by snapping off from the pedicel, leaving a picking wound. An infection process is initiated when inoculum (conidia, mycelial fragments) contacts the freshly exposed picking wound. Under favourable conditions an infection is established at the wound and continues to develop in fruit stored at 0°C.

The *B. cinerea* population increases in the canopy prior to harvest on necrotic leaf tissue, and spores are dispersed into the air and accumulate on the fruit surface (Manning and Pak 1992). Transfer of spores onto the picking wound can occur during handling of the fruit immediately after harvest (Brook 1991). Fungicide applied as a pre-harvest orchard spray can reduce the incidence of botrytis storage rots (Pennycook 1984), although it is not known how this reduction is achieved.

Current dicarboximide fungicide label recommendations include a pre-harvest application to reduce the incidence of botrytis storage rot. However, efficacy data required to register the dicarboximide fungicides was acquired prior to the development of dicarboximide resistance in populations of *B. cinerea* in kiwifruit orchards. Resistance to dicarboximide fungicides is now widespread. The aims of these experiments were to determine the impact of fungicide resistance on the effectiveness of the pre-harvest spray and to investigate the mechanism by which pre-harvest sprays reduce the incidence of storage rot.

METHODS

Trials were established on two kiwifruit orchards in the Bay of Plenty (Trials 1 and 2) and one in South Auckland (Trial 3), using single vine plots with five replicates per treatment. Treatments were as follows:

Treatment 1: Dicarboximide application - iprodione (Rovral FLO) at 37.5 g/100 litres

Proc. 48th N.Z. Plant Protection Conf. 1995: 22-26

Treatment 2: Water and Citowett (25 ml/100 litres)

Treatment 3: Unsprayed

Vines were sprayed to run off (approximately 10 litres per vine) using an engine-driven pump (300 p.s.i.) with hand lance. Three trays (36 fruit/tray) of fruit were harvested from each plot prior to spray application and again 4 h after treatments had been applied. Fruit were picked into a canvas picking bag, jostled 10 times, and packed into export trays (wooden trays, plastic liner and plastic pocket-pack). Within 1 h of harvest the trays of fruit were cooled rapidly in a coolstore at 0°C, with gaps between trays to allow for air movement.

Twenty fruit from each treatment were weighed individually within 1 h of each harvest, spread on a bench in a single layer at 20°C, and re-weighed after 24 h to determine the free surface moisture of fruit at harvest: Wetness (%) = $(W1 - W2)/W2 \times 100$ [W1 = initial fruit weight, and W2 = 24 h fruit weight].

Fifty 9 mm discs of necrotic leaf tissue were sampled from each plot immediately before treatment and again 4 hours after treatment. Counts were made of the number of discs on which fresh *Botrytis* sporulation developed during overnight humid incubation.

Fruit were assessed for botrytis storage rots after 4, 6, 8, 10, 12, 16 and 20 weeks in coolstorage at 0°C. Rotted fruit were removed at each assessment, and isolations were made from many of the rots in order to confirm the identity of the pathogen. The cultures were tested for resistance to iprodione using the methods described by Beever *et al.* (1989). Since the time of day when the fruit is picked is known to affect the level of storage rots (Brook 1991), the rot incidences in Treatments 1 and 2 were standardised for the proportional change in the level of rots of the unsprayed fruit between the two harvest times. Efficacy of the sprays was calculated separately for each replicate from the change in standardised incidence of storage rots.

RESULTS

The levels of storage rot incidence and fungicide resistance of each of the three orchards are in Table 1. Storage rot incidence ranged from low (Trial 2) to very high (Trial 3). The two properties with a high frequency of dicarboximide-resistant strains of *B. cinerea* had different levels of storage rot incidence.

TABLE 1: Mean incidence of botrytis storage rots from all treatments after 20 weeks coolstorage at 0°C, and frequency of dicarboximide-resistant strains of *B. cinerea* in each of the three trial blocks.

Trial	Dicarboximide resistance (%)	Storage rots (%)
1	100	10.7
2	55	1.4
3	87	55.2

The efficacy of the water and dicarboximide treatments relative to the unsprayed is in Table 2. In each trial, the water spray had the highest efficacy, significantly greater than unsprayed. For the dicarboximide treatment, a significant efficacy was obtained only in Trial 1, an orchard with a high frequency of dicarboximide resistance. In Trial 2, where resistance was of moderate frequency, the dicarboximide treatment performed poorly.

The primary source of inoculum for fruit infection is necrotic leaf tissue colonised by *B. cinerea* (Manning and Pak 1992). The incidence of *B. cinerea* on necrotic leaf tissue before and after application of the treatments is shown in Table 3. None of the treatments had a significant effect on the incidence of *B. cinerea* in the field.

Fruit wetness as achieved by the treatment applications is shown in Table 4. Sprayed fruit were significantly wetter than unsprayed, but there was no consistent

pattern across the treatments in the different trials. Efficacy of the treatments did not appear to be related to the extent of fruit wetness.

TABLE 2: Percent control ($\pm$ SE) of botrytis storage rots of a dicarboximide application and a water spray compared with an unsprayed control (=0). Numbers in brackets are mean standard errors for five replicates. The probability that a treatment differs significantly from an unsprayed control is indicated.

Treatment	Trial 1		Trial 2		Trial 3	
unsprayed	0	-	0	-	0	-
water	46 (19)	P= 0.04	32 (16)	P=0.06	25 (8)	P=0.05
dicarboximide	36 (14)	P= 0.03	-36 (46)	P=0.45	-1 (11)	P=0.89

TABLE 3: Incidence (%) of *B. cinerea* on necrotic leaf discs immediately prior to and 4 h after treatment application for Trials 1 and 3. Numbers in brackets are mean standard errors (n¹=250).

Treatment	Trial 1		Trial 3	
	Before	After	Before	After
unsprayed	30 (6)	28 (5)	42 (7)	44 (9)
water	43 (8)	36 (9)	39 (6)	35 (5)
dicarboximide	32 (11)	23 (11)	36 (4)	38 (4)

¹ n = number of leaf discs/ treatment

TABLE 4: Fruit wetness ($\pm$ mean standard error) expressed as a percentage of “dry” fruit weight after 24 h. All fruit were collected and weighed at the time of harvest. Numbers in brackets are mean standard errors (n¹=20).

Treatment	Trial 1	Trial 2	Trial 3
unsprayed	0.31 (0.01)	0.33 (0.01)	0.45 (0.01)
water	0.39 (0.02)	0.69 (0.05)	0.84 (0.05)
dicarboximide	0.48 (0.03)	0.65 (0.04)	0.63 (0.03)

¹ n = number of fruit/treatment.

DISCUSSION

The results show that dicarboximide treatment failed to significantly reduce storage rot incidence in two of the three trials. In Trial 1 a moderate reduction in rots was achieved despite the high incidence of dicarboximide resistant strains of *B. cinerea* on this property. However, the reduction in rots achieved did not significantly differ from that of the water spray. Pyke *et al.* (1994) reported nil control of botrytis stem-end rots with pre-harvest dicarboximide applications.

In the absence of dicarboximide resistant strains of *B. cinerea*, the pre-harvest spray has two main effects: 1) to reduce the incidence of primary storage rots and 2) to reduce losses from “nesting” which is the development of secondary rots caused by mycelial spread from fruit to fruit. In the case of “nesting”, the spray acts as a prophylactic, and good control can be achieved in the absence of resistant strains. However, Pennycook (1989) has found that dicarboximide-resistant strains of *B. cinerea* are able to infect dicarboximide treated fruit, indicating that nesting can occur where the primary rots have developed from resistant inoculum. In these trials, to prevent “nesting”, infected fruit were removed from the trays as rot symptoms

appeared. The results presented here are, therefore, a reflection of the efficacy of the pre-harvest spray in reducing primary rots.

The mechanism by which pre-harvest sprays achieve a reduction in primary storage rots is unknown. The major pathway for infection of the picking wound is inoculum (spore) transfer from the surface of adjacent fruit during post-harvest handling (Manning and Pak 1993). The mechanism of action of the pre-harvest spray cannot, therefore, be prophylactic as infection occurs after harvest through the picking wound, which at the time of spray application is protected by the pedicel. However, application of a dicarboximide to the picking wound either directly (Pennycook 1989) or as a dip (Pennycook 1989; Pyke *et al.* 1994) provides effective control of dicarboximide-sensitive *B. cinerea*. In this case the fungicide acts as an eradicant at the site of infection.

Given that the primary source of inoculum for fruit infection is necrotic leaf tissue colonised by *B. cinerea* (Manning and Pak 1993), it could be assumed that the mode of action of the pre-harvest spray is a reduction of the amount of viable inoculum in the field. However, in these trials the incidence of viable inoculum in the field was not significantly affected by a pre-harvest dicarboximide application.

A possible mechanism of action of the pre-harvest fungicide spray is to reduce the transfer of inoculum to the picking wound during the harvest process. Spores may be immobilised on the surface of wet fruit, inhibiting the transfer onto the picking wound. Such a mechanism would explain the results obtained from the water spray and would also explain why, in at least one trial, the fungicide application had moderate efficacy despite the high incidence of dicarboximide-resistant strains.

The wetting effect is dependent both upon drying conditions and upon the water retention characteristics of the fruit surface. In these trials a significant wetting effect from the water treatment continued for least 4 hours. Label recommendations for dicarboximide sprays indicate a withholding period of 24 hours, which in most cases would be sufficient to ameliorate the wetting effect. This may explain the lack of efficacy of pre-harvest sprays reported by Pyke *et al.* (1994) who maintained the 24 hour withholding period.

Further support for wetting as the possible mode of action of the pre-harvest fungicide spray comes from the data of Pennycook and Manning (in Brook 1990) which indicated that fruit picked during, and immediately after, rain had less rots in storage than fruit picked in dry weather. However, wetting can be eliminated as the mechanism operating in earlier fungicide trials (Pennycook 1984) in which harvest was delayed for 3 days (the withholding period then in force) after the pre-harvest dicarboximide application. The efficacy demonstrated in those trials (with 100% dicarboximide-sensitive *B. cinerea* populations) must be attributed to a fungicidal mechanism.

Treatment efficacy can be demonstrated only in trials which have a sufficient level of disease incidence to discriminate between treatments. The low level of disease incidence in Trial 2 (Table 1) has contributed to the apparent lack of treatment efficacy (Table 2). In kiwifruit a major factor in reducing storage rot incidence is the "curing" which occurs when harvested fruit is held for a period of time before being coolstored (Pennycook and Manning 1992). In these trials, curing was minimised and this resulted in high expression of the storage rot potential. Curing may be another explanation for the lack of efficacy obtained by Pyke *et al.* (1994) from their pre-harvest fungicide applications.

These trials are continuing in the 1995 season to understand the effect of fruit wetness at harvest on *B. cinerea* infection of kiwifruit.

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