Causes and Control of Cantaloupe Postharvest Wastage in Australia

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ABSTRACT

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Wastage of cantaloupe melons (*Cucumis melo* var. *reticulatus*) by postharvest diseases in Australia is caused by species of *Fusarium, Geotrichum, Rhizopus, Cladosporium,* and *Alternaria.* Agar plates amended with fungicides were inoculated with isolates of each of these organisms. Benomyl, guazatine, imazalil, fenapanil (α-butyl-α-phenyl-1*H*-imidazole-1-propanenitrile), sodium-*o*-phenylphenate, TD 5056 (2-methylsulphanil-6-nitrobenzothiazole), thiabendazole, thiram, and tridemorph reduced the growth of two or more of the test organisms by at least 90% relative to controls. Captan, dichloran, and fenaminosulf were less inhibitory but affected all or most of the test organisms. In tests with wound-inoculated fruit, benomyl controlled *F. solani* only. Guazatine reduced wastage due to *G. candidum, Alternaria* sp., *F. solani,* and *R. oryzae.* Guazatine and benomyl have potential use for the control of cantaloupe postharvest wastage.

Mature cantaloupe melons (*Cucumis melo* L. var. *reticulatus* Naud.) are an extremely perishable commodity that suffers postharvest wastage from a disease and blemish complex incited by soilborne fungi (2,15). Wastage can be severe under Australian conditions of hot, wet weather at harvest time and limited use of refrigeration. Recommendations for minimizing postharvest disease in melons stress the importance of good handling procedures, including avoidance of wounding and temperature management (9).

Postharvest dip treatments for both cantaloupe and honeydew melons have also been studied, and some beneficial responses have been obtained from copper sulphate paste applied to the stem scar (10); dips containing sodium borate (1), chlorine (1,7), or sodium-*o*-phenylphenate (6,7); hot water dips at 57–63 C (6); and nitrogen trichloride fumigation (1). *Fusarium* decay of cantaloupes was controlled by dips containing thiabendazole, captan, and maneb, whereas dips of hot water (57 C), chlorine, and ziram gave only moderate control (14). Captan dips heated to 57 C reduced total wastage in cantaloupes affected with disease and blemish caused by *Fusarium, Rhizopus,* and *Alternaria* and gave better results than cold captan dips or hot water dips (57 C) (12,13). Hot benomyl dips (57 C) reduced stem-scar and rind moulds on cantaloupes (8).

In this paper we describe the postharvest disease complex of cantaloupes, which is of economic concern in Australia, and we report in vitro and in vivo tests on the susceptibilities of cantaloupe wastage organisms to fungicides. A number of fungicides are identified that have potential use in the control of melon wastage, and the effects of some hot water treatments are also examined.

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MATERIALS AND METHODS

Disease survey. Diseased cantaloupes were taken from consignments arriving at the Sydney Farm Produce Markets during 1976–1980. Mature cantaloupes harvested in the main producing areas of New South Wales were stored in the laboratory at 25 C and examined for disease. Superficial fungal growth and excised pieces of diseased tissue were plated onto potato-dextrose agar (PDA) with and without added lactic acid. The isolates were identified and tested for pathogenicity by wound-inoculation of surface-sterilized melons with a spore suspension. Symptoms were observed, and the fungi were reisolated. Isolates of the major wastage organisms found were deposited in the Department of Agriculture Herbarium, Rydalmere. The organisms chosen for detailed study were *Fusarium solani* (Mart.) Sacc. (DAR 26277), *Geotrichum candidum* Link ex Pers. emend. Carmichael (DAR 26283), *Rhizopus oryzae* Went & Prinsen Geerlings (DAR 26282), *Cladosporium cladosporioides* (Fresen.) de Vries (DAR 26280), and *Alternaria* sp. (DAR 26281).

In vitro plate tests. Plates of PDA were amended with commercial fungicide formulations (1,000 mg a.i./L) and inoculated with mycelium (5 mm^2) cut from the margin of a 7-day-old culture of the test organism on PDA. Three replicate plates per treatment were incubated at 20 C, and the ratios of colony diameters on amended and control plates were expressed as percentages. The fungicides used were benomyl, dichloran, captan, thiram, maneb, fenapanil (α-butyl-α-phenyl-1*H*-imidazole-1-propanenitrile), imazalil, tridemorph, guazatine, quintozene, etridiazol (5-ethoxy-3-trichloromethyl-1,2,4-thiadiazole), 2-aminobutane, TD 5056 (2-methylsulfanil-6-nitrobenzothiazole), thiabendazole, copper oxychloride, calcium polysulfide, dithianon, fenaminosulf, sodium-*o*-phenylphenate, dodine, and thiophanate methyl.

In vivo tests with wound-inoculation. Seven-day-old colonies of test organism on PDA were wetted with a 0.01% solution of Agral 60, a nonionic wetter, and spores were detached by gentle brushing. The suspension was filtered through glass wool and total spore count determined with a hemacytometer.

Cantaloupes, cv. PMR 45, were harvested from commercial crops at "full-slip" maturity and "eastern choice" ripeness (11) (ie, abscission crack between fruit and pedicel, fruit greenish yellow to light yellow). Punctures 4 ± 1 mm deep were made with nail tips at the stem end and at three points around the equator of each melon. Aliquots of spore suspension (40 µl) containing 10^5 spores were injected into each puncture by syringe, and the melons were incubated with the punctures open to the air.

Trial 1. Melons inoculated with *F. solani, G. candidum*, and *R. oryzae* were incubated for 24 hr at ambient temperature (20–35 C) and humidity. The melons were then immersed for 1 min in dips containing benomyl, captan, dichloran, fenaminosulf, guazatine, or water alone (1,000 mg a.i./L + 0.01% Agral 60). For hot water treatment, 50 L of water containing 0.01% Agral 60 was heated to 55 C, and melons were dipped for 1 min. Each treatment was applied to four replicate units made up of 15 melons each.

The melons were drained, air-dried, packed in fiberboard cartons, and transported 700 km to the laboratory. Wastage was assessed after storage at 25 C and ambient humidity for 8 days after harvest. Each lesion at an inoculation site was counted and its diameter measured on a transverse section cut through each puncture. The organism present in each lesion was determined from internal and external symptoms and confirmed by examination of a number of lesions by microscopy and reisolation. Less than 5% of inoculation sites were infected with contaminating organisms.

Trial 2. Melons were inoculated with *G. candidum* and incubated for 4 hr at ambient temperature (20–35 C) and humidity. The melons were then immersed for 1 min in dips containing guazatine, tridemorph, TD 5056, or water alone (1,000 mg a.i./L + 0.01% Agral 60). Each treatment was applied to four replicate units made up of 15 melons each. The treated melons were packed, transported, and assessed as in trial 1.

Trial 3. Melons were inoculated with *G. candidum* and incubated at 20 C and ambient humidity for 0, 4, 8, 16, 24, and 48 hr. After each incubation time, five melons were immersed for 1 min in a guazatine solution (500 mg/L), and five melons were similarly treated with water alone. Agral 60 (0.01%) was added to both dips. Lesion diameter was measured after storage at 20 C and ambient humidity for 5 days after inoculation.

In vivo tests with surface inoculation. Seven-day-old cultures of *G. candidum* on PDA were cut into slabs (5 mm^2). Slabs were inserted in the abscission cracks of PMR 45 melons attached to the vine or were applied to the stem scars of harvested, full-slip PMR 45 melons. Pads of moist cotton wool were wrapped around abscission cracks or pressed onto stem scars, then sealed with masking tape. This technique partially simulated natural infection of the stem scar. The provision of PDA substrate ensured a high incidence of infection. At 24 or 48 hr after inoculation, the melons were dipped for 1 min in guazatine (500 mg/L) or water alone or for 3 min in hot water (55 C). Agral 60 (0.01%) was added to all dips. Each treatment was applied to four replicate units made up of 15 melons each. The treated melons were packed, transported, and assessed as in trial 1.

The incidence of infection was invariably greater from postharvest inoculation, where incubation took place in a saturated atmosphere at about the optimal temperature (30 C) for growth of *G. candidum*. When inoculation occurred before harvest, the cotton wool pads dried out rapidly under shade temperatures greater than 40 C, which are above optimal for *G. candidum*.

Statistical analysis. Results were analyzed by analysis of variance, using the arc sine transformation for percentage data. Mean separation was by the Waller-Duncan k-ratio LSD test (3), using the k = 100 level.

RESULTS AND DISCUSSION

Causes of postharvest disease. *G. candidum, R. oryzae, C. cladosporioides*, at least four species of *Fusarium*, and one species of *Alternaria* were readily isolated from diseased cantaloupes. Many isolates (80%) of *Fusarium* sp. and all isolates of *G. candidum* and *R. oryzae* tested were proven pathogenic by Koch's postulates. Species of *Alternaria* and *Cladosporium* were usually isolated from superficial mould on the rind or stem scar, but none of these isolates was pathogenic. Isolates of both *Alternaria* and *Fusarium* species were occasionally obtained from the same internal lesion, but only *Fusarium* sp. was pathogenic. Despite their lack of pathogenicity, the *Alternaria* and *Cladosporium* species isolated caused an unsightly blemish on the rind and stem scar of the fruit.

Pathogenicity to cantaloupes of *R. oryzae* (5); *F. solani, A. tenuis, A. brassicae microspora, C. cucumerinum* (15); and *G. candidum* (2) has been reported. We have not yet isolated *C. cucumerinum* from cantaloupes nor characterized the species of *Alternaria* that grow on the rind and stem scar.

Activity of fungicides in vitro. Benomyl, guazatine, imazalil, fenapanil,

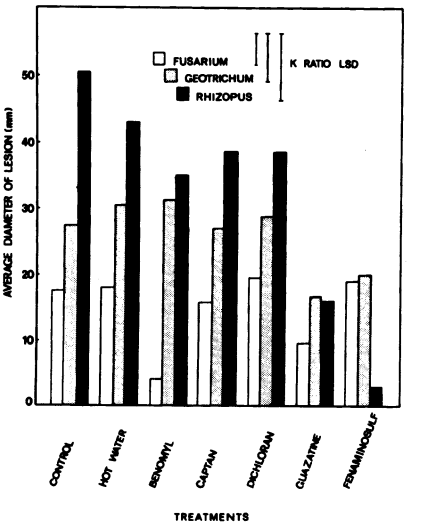

Fig. 1. Effects of fungicide (1,000 mg a.i./L) and hot water (55 C, 1 min) dips on lesion size in cantaloupes wound-inoculated with *F. solani, G. candidum*, and *R. oryzae*.

Table 1. Effect of fungicides on growth of cantaloupe wastage organisms in vitro

Fungicide[a]	Growth (%) compared with control				
	Fusarium solani	*Geotrichum candidum*	*Rhizopus oryzae*	*Alternaria* sp.	*Cladosporium cladosporioides*
2-Aminobutane	100	96	100	48	84
Benomyl	0	94	100	90	0
Captan	10	15	100	32	14
Copper oxychloride	100	90	100	97	96
Dichloran	21	25	34	14	20
Fenaminosulf	100	46[b]	8[b]	64[b]	68[b]
Dithianon	37	49	100	61	43
Dodine	68	51	100	29	28
Guazatine	20	10	100	5	18
Imazalil	0	64	100	0	16
Fenapanil	0	80	100	0	0
Calcium polysulfide	90	92	100	60	82
Maneb	63	37	100	39	20
TD 5056	8	0	14	11	7
Quintozene	50	89	75	22	49
Sodium-*o*-phenylphenate	0	17	100	0	0
Thiabendazole	0	79	100	46	0
Etridiazol	68	69	100	88	90
Thiophanate methyl	0	78	100	88	13
Thiram	0	23	100	8	15
Tridemorph	17	0	100	4	15

[a] 1,000 mg a.i./L.
[b] Growth very sparse.

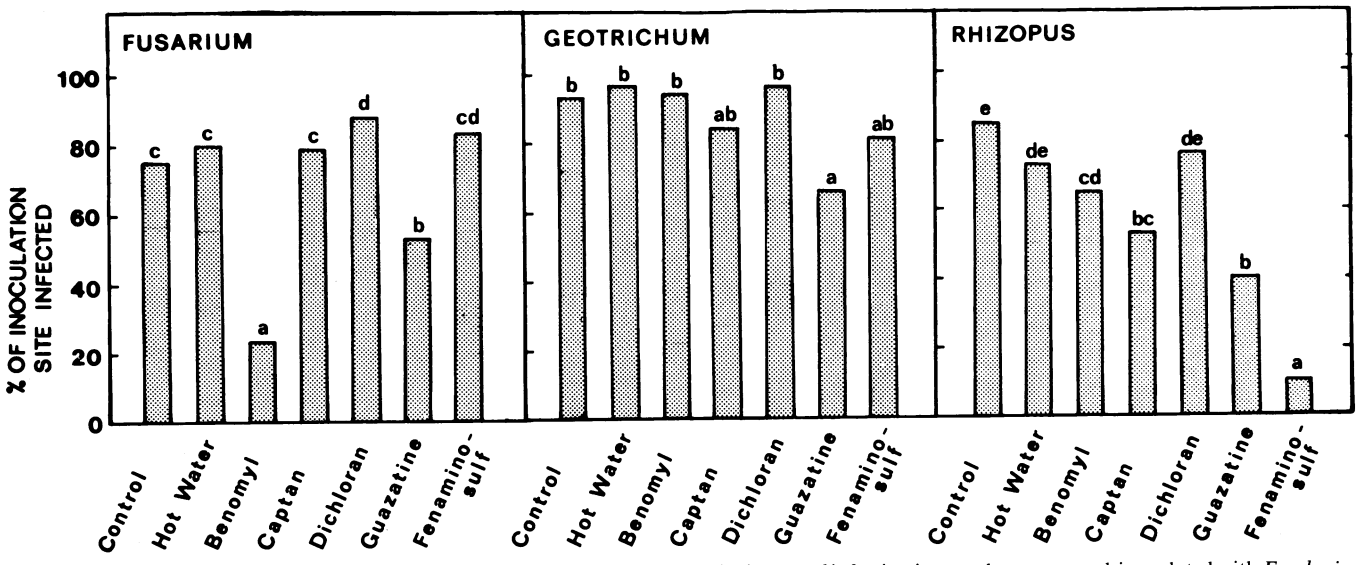

Fig. 2. Effects of fungicide (1,000 mg a.i./ L) and hot water (55 C, 1 min) dips on incidence of infection in cantaloupes wound-inoculated with *F. solani*, *G. candidum*, and *R. oryzae*. (Different letters denote significant differences.)

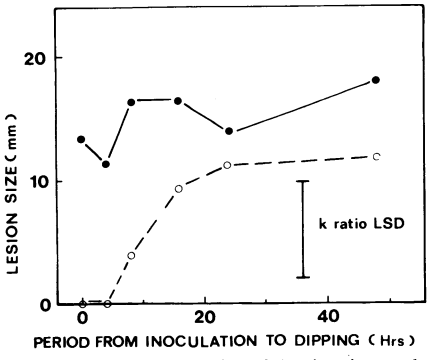

Fig. 3. Effect on lesion size of the time interval between wound-inoculation with *Geotrichum candidum* and application of guazatine at 500 mg a.i./ L (o), or water (•).

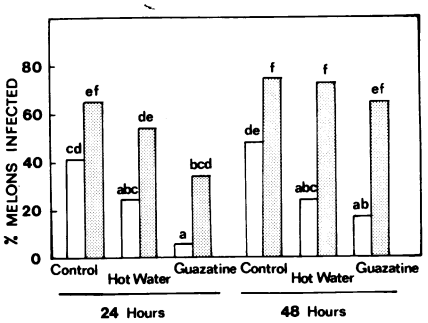

Fig. 4. Effect on infection incidence of the time interval between surface-inoculation with *Geotrichum candidum* before (open bar) or after (dotted bar) harvest and application of guazatine (500 mg a.i./ L) or hot water (55 C, 3 min). (Different letters denote significant differences.)

TD 5056, sodium-*o*-phenylphenate, thiabendazole, thiram, and tridemorph reduced the growth of two or more of the test organisms by at least 90% relative to controls (Table 1). Fenaminosulf caused abnormal growth of all organisms except *F. solani* and also severely inhibited *R. oryzae*. Colonies lacking aerial structures and fruiting bodies spread diffusely across fenaminosulf-amended plates. Similarities in the spectrum of fungicidal activity of chemically similar compounds are evident in Table 1 (eg, when comparing results for benomyl, thiabendazole, and thiophanate methyl or for imazalil and fenapanil).

Activity of fungicides in vivo. Lesions caused by *F. solani* were reduced significantly in both size (Fig. 1) and number (Fig. 2) by treatment with either benomyl or guazatine. The size and incidence of *G. candidum* lesions were significantly reduced by guazatine treatment (Figs. 1 and 2), although control was poor. In a further comparison with other fungicides that controlled *G. candidum* in vitro, guazatine was the best treatment and gave excellent disease control. With 4 and 14% of the wound-inoculated sites infected, guazatine and tridemorph differed significantly from each other and from the control (98% infected) in their ability to control *G. candidum*. TD 5056 (also 98% infected) was no better than the control.

Both the size and incidence of *R. oryzae* lesions were significantly reduced by treatment with fenaminosulf, guazatine, captan, and benomyl (Figs. 1 and 2). Severe surface mycelial growth resulting from field-inoculation of *Alternaria* sp. and *Cladosporium* sp. was observed in control fruit (cold water dip), but melons treated with hot water, guazatine, or fenaminosulf were almost free from surface fungi.

When in vitro and in vivo results are compared, our results agree with other findings that postharvest chemical treatments that prevent decay of inoculated fruit may at best be only fungistatic under similar conditions in vitro (4). For example, guazatine did not control *R. oryzae* in vitro but was effective in vivo. Dichloran controlled *R. oryzae* and *G. candidum* on plates only.

Hot water treatment did not control any of the wound-inoculated pathogens. Experiments in which the depth of puncture and the temperature and duration of treatment were studied (*results not shown*) confirmed that noninjurious heat treatments cannot control infections incited by the wound-inoculation technique used.

Effect of time between inoculation and treatment on efficacy of guazatine. Guazatine applied at 0 or 4 hr after wound-inoculation with *G. candidum* gave almost complete control of disease, but efficacy of the treatment decreased rapidly when it was applied later than 4–8 hr after inoculation (Fig. 3). This result explains the poor control obtained with guazatine treatment when the delay was 24 hr (Figs. 1 and 2) and the good control obtained in further comparisons when the delay was only 4 hr. The time between inoculation and application is, therefore, critical in determining the efficacy of guazatine treatment for control of *G. candidum*.

The incidence of decay due to *G. candidum* was reduced when melons were treated with guazatine or hot water at either 24 or 48 hr after inoculation of the abscission crack in the field (Fig. 4). Results obtained with melons surface-inoculated at the stem scar after harvest were less satisfactory, although the proportion of melons infected was significantly reduced from about 60% in controls to 30% by treatment with guazatine at 24 hr (Fig. 4).

Useful reductions in cantaloupe wastage by the benzimidazole derivatives thiabendazole and benomyl have been reported (8,14). However, we have shown that these compounds quite specifically control Fusarium rot, whereas guazatine gives some control of all wastage organisms in vivo (Figs. 1 and 2). The 4–8 hr time period within which guazatine must be applied to give maximum control of wound-inoculated *G. candidum* (Fig. 3)

cannot be related directly to infections incited by natural field-inoculation. When using surface-inoculated melons, a delay of at least 24 hr is permissible (Fig. 4). Preliminary results show that postharvest guazatine treatment can control Geotrichum rot arising from natural field-inoculation. Work is in progress to determine the best way of utilizing the fungicides benomyl and guazatine in reducing market wastage of cantaloupes.

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