Sites of Action of Fungicides in the Control of Citrus Melanose

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ABSTRACT

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Major sites of action against *Diaporthe citri* were determined for basic copper sulfate (BCS), captafol, dithianon, benomyl, and chlorothalonil. Relatively long protectant action on fruit rind was provided by single postbloom sprays of BCS, captafol, and dithianon, but not by chlorothalonil, thus explaining, at least in part, why field control of melanose with chlorothalonil has been variable and often poor. Benomyl provided little protectant action, but, unlike the other fungicides, it inhibited inoculum production on dead twigs. Such antisporulant action probably explains why prebloom benomyl sprays have reduced melanose severity on rind. Redistribution of applied material from sites of application to newly exposed susceptible tissue apparently played a major role in disease control only with captafol. Although captafol did not reduce inoculum production, it did reduce the number of viable conidia that reached the surface of previously unprotected tissue. Captafol deposits on bark were more lethal than were deposits of the other fungicides to conidia suspended in water that contacted briefly a treated bark surface.

Additional key words: *Citrus paradisi*, *Phomopsis citri*.

The major source of inoculum of the citrus melanose fungus, *Diaporthe citri* (Fawc.) Wolf is provided by pycnidia formed on dead twigs in the tree canopy (7, 11). No fruiting bodies are produced on the melanose pustules themselves. Fruit rind remains susceptible to infection for about 12 wk after petal fall (7, 11).

In Florida, copper fungicides and captafol are the only materials currently used in sprays to control melanose (4). The coppers are applied postbloom to protect fruit from infection, but sprays before fruit set have proved ineffective (7, 9, 11). Captafol also is effective when applied postbloom (6, 8, 12), but in Florida it generally is not applied then or after new shoot growth commences because of the risks of phytotoxicity. However, captafol at high rates can be applied safely in February before growth commences to reduce infections of fruit rind (6, 9, 10). Control has been attributed to redistribution of captafol by water from sprayed parts of the canopy onto newly exposed tissue, as originally described in Japan (12). Importance of redistribution in achieving control with captafol has been reported also for apple scab (5) and European apple canker (2).

Other fungicides that have reduced melanose severity when applied postbloom have included dithianon (9, 10, 12), which has been consistently effective, and benomyl (3, 8, 9) and chlorothalonil (3), which have given variable results. Postbloom sprays of benomyl are sometimes as effective as copper fungicides for melanose control (3, 8), but in most Florida tests, benomyl has given relatively poor control of this citrus disease (9). Unlike copper fungicides, however, benomyl has reduced melanose severity on fruit rind when applied prebloom (9). Chlorothalonil gave little or no control of melanose in some Texas (8) and Florida (9) tests, but in one Florida report (3) and in some of the tests reported herein, this material and basic copper sulfate provided comparable control.

The purpose of these studies was to determine (i) the relative abilities of deposits of a copper fungicide, captafol, dithianon, benomyl, and chlorothalonil to provide long-term protection of fruit rind, (ii) whether these fungicides can reduce the amount of viable inoculum reaching the fruit or leaf surface and (iii) whether redistribution of fungicide is a major factor in melanose control.

MATERIALS AND METHODS

Fungicides.—The fungicides used were basic copper sulfate (BCS) (Tribasic copper sulfate, Cu 53%, Cities Service Co., Atlanta, GA 30302); captafol (Difolatan 4F, Chevron Chemical Co., Ortho Division, San Francisco, CA 94104); dithianon (Thynon W75, Thompson-Hayward Chemical Co., Kansas City, KS 66110); benomyl (Benlate 50W, E. I. duPont de Nemours & Co., Wilmington, DE 19898); and chlorothalonil (Bravo 6F, Diamond Shamrock Chemical Co., Cleveland, OH 44114). Concentrations and rates of application are given as the amounts of formulated product suspended in water.

Fungus culture and inoculum production.—Cultures of *Diaporthe citri* were maintained on potato-dextrose agar (PDA). To produce pycnidia, the fungus was grown in culture tubes on sterilized, 1- to 2-yr-old stem portions of 4- to 8-mm diameter, cut from the canopy of grapefruit (*Citrus paradisi* Macf.) trees.

For some studies, the fungus was grown on 150-mm-

long autoclaved grapefruit stems in 1,000-ml Erlenmeyer flasks. In other studies, when it was desired to emulate more closely the physical condition of a natural dead bark substrate, the fungus was grown on stems sterilized with propylene oxide gas. In the latter case, living stems were cut into 75-mm lengths and dried in the greenhouse for 2 wk. Six stems were placed in each 9-cm-diameter petri dish and the dishes were stacked, with lids slightly raised, in the upper compartment of a desiccator. Propylene oxide (4 ml/liter of desiccator volume) was poured into the lower compartment and the desiccator was sealed for 16 hr. After the petri dishes were removed from the desiccator and the contents aired for 24 hr by keeping the lids slightly raised, 15 ml of sterile distilled water were poured into each dish to moisten the stems.

The autoclaved green stems or propylene oxide-sterilized dried stems inoculated with mycelium from PDA cultures began to produce mature pycnidia within 2-3 wk. After pycnidia development was adequate, the stems were placed between two layers of 6-mm (mesh size) hardware cloth and exposed outdoors on a wooden bench under conditions of natural wetting and drying.

Protectant action of fungicides on fruit surface.—The protectant action of the fungicides against fruit infection was studied by spraying grapefruit trees with low natural disease potential and by placing inoculum later in the tree canopy. Sprays were applied by handgun at 28 kg/cm^2 to single-tree plots replicated five times in a randomized block design. After the spray residue had dried, three hardware-cloth carriers, each containing 12, 150-mm-long pycnidia-bearing stems, were placed at two sites on each tree above groups of young fruit. One to 2 mo later, fruit located beneath the introduced inoculum were picked and examined for melanose. Numbers of fruit thus examined averaged 35 per tree in 1972 and 47 per tree in 1974.

This procedure was insufficient to separate a purely protectant action of a fungicide from a possible lethal action on released conidia following contact with fungicide deposits on leaves and bark en route to the rind surface. Therefore, in 1975 and 1976 another method was used to study the relative protectant action more precisely. In this study, grapefruit trees with a low natural melanose potential were sprayed postbloom with the test fungicides using four trees per treatment in 1975 and two trees per treatment in 1976. Periodically after spraying, samples of fruit (18 to 30 per replication depending on size and the number required to fill the basin), with stem attached and leaves removed, were cut from the tree and placed on 3-cm high, 12-mm (mesh size) hardware-cloth platforms in plastic wash basins (26 cm × 32 cm × 14 cm deep). The stems of the fruit were inserted through the hardware cloth and the fruit were placed close together to prevent shaking and change in their orientation. The basins were returned to the laboratory 1 to 2 hr after picking and deionized water was added to the level of the platform. Care was taken not to touch the surface of the rind at any time following fungicide application.

The rind was inoculated with suspensions of conidia prepared by stirring spore tendrils (exuded from pycnidia-bearing stems maintained over water in a closed chamber for 4 days) into a 1% solution of filtered orange juice in distilled water. Orange juice was used because the conidia germinated better in it than in distilled water, but only a low concentration was required; greater concentrations (> 5% orange juice) inhibited appressorium development and thereby reduced the infectivity of the inoculum. Conidia sank rapidly and adhered to the bottom of the container and frequent dislodgement and stirring with a small paintbrush was necessary to maintain a uniform suspension. Drops (15 μliter) of suspension adjusted to 5×10^5 conidia/ml were applied with a syringe to two sites, approximately 10 mm apart, near the uppermost point of each fruit. To increase the relative humidity rapidly, a fine spray of water was directed (but without causing run off) onto the fruit just prior to sealing each basin with a polyethylene sheet. The seals were removed after 2 days and the rind was examined for melanose symptoms after 7 days. The fruit was regarded as lacking an effective fungitoxic residue even if disease symptoms developed at only one of the two inoculation sites.

Effect of fungicides on inoculum supply.—The first tests were made on naturally colonized dead twigs collected from the canopy of old grapefruit trees that had a history of severe melanose. Ten, 150-mm-long twigs were placed in each hardware-cloth carrier, and 15 carriers were dipped in each fungicide suspension for 30 sec. Carriers were suspended from each of five 1.2-m-high wooden tripods spaced 3.5 m apart in a randomized block design in an area isolated from woody vegetation. Two potted greenhouse-grown trap plants, each with a

TABLE 1. Effectiveness of fungicides for protecting grapefruit rind from attack by conidia of *Diaporthe citri* (which can cause melanose) following their release by rain from pycnidia-bearing twigs placed in the tree canopy after spraying

Fungicide and rate of application	Melanose-free fruit (%)[x]	
	1972[y]	1974[z]
Basic copper sulfate 1.8 g/liter (1.5 lb/100 gal)	68.5 a	77.6 a
Dithianon 1.2 g/liter (1.0 lb/100 gal)	69.1 a	...
Benomyl 0.6 g/liter (0.5 lb/100 gal)	38.8 b	50.6 b
Chlorothalonil 1.7 ml/liter (0.17 gal/100 gal)	34.3 b	...
Chlorothalonil 2.5 ml/liter (0.25 gal/100 gal)	...	85.5 a
Control (unsprayed)	25.1 b	31.7 b

[x]Values followed by different letters are significantly different ($P = 0.05$) using Duncan's multiple range test.

[y]Trees sprayed 1 May: infection periods on 7, 16, 18, 20, 29, and 30 May, but no further infection prior to disease recording on 12 June.

[z]Trees sprayed 15 May: infection periods on 16 May, 2, 4, 5, 10, and 11 June, but no further infection prior to disease recording on 25 June.

minimum of five young susceptible shoots, were placed under each tripod to await rainfall-induced conidia dispersal and infection. After 2-3 wk, the plants were returned to the greenhouse for symptom development and diseased and healthy new shoots were recorded after another 2 wk.

To determine whether a fungicide actually inhibited development of pycnidia or conidia, the following procedure was followed. Colonies of *D. citri* were established on 75-mm long stems, that had been sterilized with propylene oxide. After the mycelium had completely covered the stem, four stems were placed in each hardware-cloth carrier. These were held outdoors, either until the first pycnidia began to appear or until abundant mature pycnidia had formed. The carriers (five per treatment) were immersed for 30 sec in a suspension of fungicide, after which they were placed on hardware-cloth platforms in wooden boxes outdoors.

Direct counting of pycnidia to determine the effect of fungicides on their development was impossible because many were too deeply embedded in the bark substrate to be clearly visible. For this reason, and also because it was necessary to establish whether previously formed pycnidia still contained viable contents, the inoculum potential was determined by counting the number of tendrils exuded from a 2-mm-wide transect along each stem, following exposure to near 100% relative humidity for 4 days in a closed chamber.

Even if a fungicide deposit has no direct effect on inoculum production, it might still exert some lethal effect if aqueous suspensions of conidia flowed over treated bark during the water-induced dissemination. To test for such action, 75-mm lengths of 4- to 6-mm-diameter dead grapefruit twigs were placed in hardware cloth carriers (four twigs per carrier and four carriers per treatment), immersed for 1 min in the fungicide suspension, and allowed to drip dry. After various periods of natural weathering, the twigs from each carrier were floated for 2 min on 15 ml of conidia suspension in 5% orange juice in 9-cm diameter petri dishes. Conidia soon became affixed to the petri dish bottom. After 16 hr, most of the supernatant liquid was decanted to facilitate microscopic examination of the conidia at the bottom of the petri dish and germination counts were made. Conidia were considered germinated if germ tubes exceeded three times the original conidia lengths.

TABLE 2. Reduction in inoculum potential of *Diaporthe citri* after immersing pycnidia-containing grapefruit twigs in fungicide suspensions; as measured by the amount of melanose that developed on batches of trap plants exposed beneath the treated twigs for two consecutive periods

	Healthy shoots (%) on exposed trap plants			
	1972[a]		1973[b]	
	Exposure period		Exposure period	
Fungicide and concentration of dip	18 July to 7 August	7 August to 25 August	29 June to 17 July	17 July to 6 August
Basic copper sulfate, 1.8 g/liter	66.6	54.6	35.3	...
Captafol, 2.5 ml/liter	100.0*[c]	52.4	77.0*	84.7*
Dithianon, 1.2 g/liter	80.7	42.4	23.0	...
Benomyl, 0.6 g/liter	91.5*	62.0	54.4*	67.8
Chlorothalonil, 1.7 ml/liter	43.6	31.2	29.1	...
Control (water only)	68.1	53.2	42.2	54.4

[a]Twigs treated and suspended over trap plants on 11 July. Infection periods occurred on 18, 19, 31 July, and 9 and 16 August. Total rainfall 11 July to 7 August = 141 mm and 11 July to 25 August = 334 mm.

[b]Twigs treated and suspended over trap plants on 22 June. Infection periods occurred on 3, 5, 8, 9, 17, 29 July, and 5 August. Total rainfall 22 June to 17 July = 134 mm and 22 June to 6 August = 337 mm.

[c]Asterisk (*) = significantly different from control ($P = 0.05$).

TABLE 3. Effect of fungicide dips on the potential supply of conidia of *Diaporthe citri* from propylene-oxide-sterilized and artificially-colonized grapefruit stems

	Number of spore tendrils exuded per cm^2 after holding twigs in damp chamber for 4 days	
	State of pycnidia development at time of treatment	
Fungicide and concentration of dip	Few and all immature[a]	Abundant and many mature[b]
Basic copper sulfate, 1.8 g/liter	50.0	101.6
Captafol, 2.5 ml/liter	49.7	92.6
Dithianon, 1.2 g/liter	45.7	79.0
Benomyl, 0.6 g/liter	8.8*[c]	13.6*
Chlorothalonil, 1.7 ml/liter	55.2	84.2
Control (water only)	42.4	98.3

[a]Period of exposure outdoors after treatment was 13 days and rainfall during this period was 87.5 mm.

[b]Period of exposure outdoors after treatment was 4 days and rainfall during this period was 45.5 mm.

[c]Asterisk (*) = significantly different from control ($P = 0.05$).

Fungicide redistribution.—To determine whether fungicide deposits were redistributed in fungitoxic amounts to protect nearby unsprayed shoots, sprays were applied to densely foliated 1.5-m-high rough lemon *(Citrus jambhiri* Lush.) trees that would hold an unusually large amount of residue per unit volume of canopy. After the spray deposit had dried, artificially infected pycnidia-bearing twigs in hardware-cloth carriers were wedged into the canopy and potted grapefruit plants with susceptible shoots were placed beneath them. Following a rain-induced infection period, the plants were returned to the greenhouse for development of melanose and a second batch of trap plants was placed under the trees to detect any later redistribution.

Tests also were made to determine whether sufficient fungicide could be redistributed onto fruit that had set after spraying. Grapefruit trees were sprayed either in February before new growth commenced or postbloom. Fruit with stems attached were brought to the laboratory on hardware-cloth platforms in wash basins and inoculated with drops of conidia suspension as previously described.

Rating procedures.—With melanose, the dividing line between diseased and healthy tissue is ill-defined, particularly when the tissue is nearing a resistance stage of development. In compiling the results, an arbitrary division was adopted. Fruit and shoots with < 50 small pustules, none of which exceeded 0.5-mm diameter, were classified as healthy.

Determination of natural infection periods.—The probable occurrence of infection periods was based on periodic observations of fruit and young leaves for melanose symptoms and on the climatic criteria for infection used in previous studies (9). These criteria were based on data concerning minimum wetting requirements for infection at different temperatures.

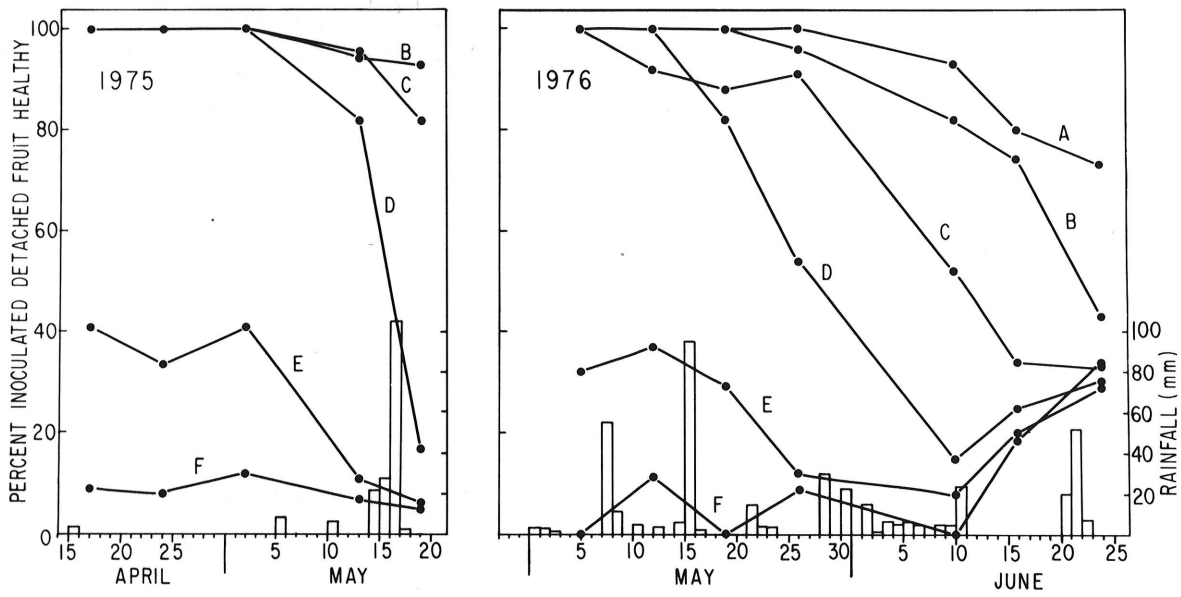

Fig. 1. Relative protection against rind infection by *Diaporthe citri* provided by fungicides applied postbloom to grapefruit trees; based on the number of artificially inoculated, detached fruit that remained healthy. Curves represent data for: A = captafol 2.5 ml/liter, B = dithianon 1.2 g/liter, C = basic copper sulfate 1.8 g/liter, D = chlorothalonil 1.7 ml/liter, E = benomyl 0.6 g/liter, F = not sprayed. Dates of spraying: 14 April 1975 and 27 April 1976.

TABLE 4. Relative effects of fungicide residues on the bark of dead grapefruit twigs on conidia survival after temporary contact of conidia suspension with the treated surface

Fungicide and concentration of suspension in which twigs were dipped	Germination of conidia 16 hr after fungicide-treated dead twigs were floated on a conidia suspension for 2 min (%)			
	No. days between twig treatment and test			
	3	10	21	28
Basic copper sulfate, 1.8 g/liter	93	86	...	...
Captafol, 2.5 ml/liter	0	0	58	90
Dithianon, 1.2 g/liter	71	81	...	...
Benomyl, 0.6 g/liter	96	88	...	...
Chlorothalonil, 1.7 ml/liter	1	73	98	...
Control (water only)	90	83	95	91
Rainfall between treatment and test (mm)	45	55	113	197

RESULTS

Protectant activity of fungicides on fruit rind.—In the 1972 and 1974 field tests (Table 1), in which naturally occurring inoculum of *D. citri* was placed in the tree canopy after spraying, benomyl failed to reduce the amount of melanose significantly. Dithianon and BCS were effective in both years, but chlorothalonil reduced disease severity significantly only in 1974. The control provided by chlorothalonil in the 1974 test may have been attributable in part to the higher rate applied. Another, and perhaps more important, reason was that in 1974 a major infection period occurred only 1 day after spraying and then not again until June, when pustule numbers and size would have been less because of greater resistance of the rind. In contrast, in the 1972 test there were six infection periods in May, with only one occurring within 2 wk after treatment.

The results from inoculation tests on detached fruit (Fig. 1) indicated more precisely the relative rind-protecting ability of the different materials. Unlike the other materials, benomyl had little protectant action even during the first 1-2 wk after treatment. Microscopic examinations of tangential sections of inoculated rind revealed no germination of conidia on fruit sprayed 5 days previously with BCS, captafol, dithianon, or chlorothalonil. However, on fruit sprayed with benomyl, 98% of conidia had swollen and 37% had produced germ tubes after 16 hr. On unsprayed rind, 91% of conidia had produced germ tubes after this time.

The rind-protecting capabilities of chlorothalonil decreased greatly after 3-4 wk of weathering (Fig. 1), whereas BCS, dithianon and captafol continued to provide good protection for another 2-3 wk, even under the conditions of higher and more frequent rainfall experienced in 1976.

The apparent increase in number of inoculation failures during June 1976 (Fig. 1) on fruit that were not sprayed, or sprayed with only the less effective materials, was due to the increased resistance of the rind to melanose with age.

Effect of fungicides on inoculum supply.—Results from tests in which pycnidia-bearing twigs were immersed in fungicide suspensions and placed over trap plants (Table 2) indicated that only benomyl and captafol reduced the amount of viable inoculum that reached the susceptible shoots.

It was impossible, from these results alone, to conclude whether such a reduction was achieved by: (i) direct action of fungicide against the inoculum at source by preventing maturation of pycnidia or by affecting the viability of the conidia within the pycnidia or (ii) loss of viability of conidia between the time they were exuded from the pycnidia and the time they reached susceptible tissue.

The results of another study (Table 3) indicated that benomyl could significantly reduce the production of conidia by pycnidia that developed on artificially colonized dead stems. This reduction was evident whether the stems were treated before or after copious pycnidia had developed. In contrast, the effect of captafol in reducing the inoculum potential apparently was not

TABLE 5. Melanose severity on container-grown trap plants following temporary exposure beneath fungicide-sprayed rough lemon canopies to which inoculum of *Diaporthe citri* had been introduced after spraying

Fungicide and rate of application	Melanose-free shoots (%)[x]	
	Exposure period	
	13 August to 16 August[y]	16 August to 25 August[z]
Basic copper sulfate, 1.8 g/liter	60.5 b	29.0 b
Captafol, 2.5 ml/liter	94.5 a	73.6 a
Dithianon, 1.2 g/liter	60.0 b	26.9 b
Control (unsprayed)	52.0 b	20.9 b

[x]Values followed by different letters are significantly different ($P = 0.05$) using Duncan's multiple range test.
[y]One rainday only with 41 mm rain.
[z]Five raindays totaling 48 mm rain.

TABLE 6. Protectant action against *Diaporthe citri* on grapefruit rind provided by redistributed captafol from a dormant spray and by deposits of basic copper sulfate and dithianon from postbloom sprays

Fungicide and rate of application	Detached fruit remaining melanose-free following inoculation with *D. citri* (%)[x]	
	1975[y]	1976[z]
Captafol, 10 ml/liter (1.0 gal/100 gal)	93.3 a	48.4 b
Captafol, 5 ml/liter (0.5 gal/100 gal)	68.7 b	27.7 c
Basic copper sulfate, 1.8 g/liter (1.5 lb/100 gal)	88.5 a	93.6 a
Dithianon, 1.2 g/liter (1.0 lb/100 gal)	100.0 a	...
Control (not sprayed)	12.5 c	0.0 d

[x]Values followed by different letters are significantly different ($P = 0.05$) using Duncan's multiple range test.
[y]Dormant sprays applied 21 February, postbloom sprays applied 9 May, and fruit inoculated on 27 May. Bloom peak: late March.
[z]Dormant sprays applied 24 February, postbloom spray applied 28 April, and fruit inoculated on 21 May. Bloom erratic: early to late March.

caused by a reduction in inoculum production, but by some action on the conidia after their release.

After conidia masses had oozed out of the pycnidia in the presence of free water, they were observed to flow over nearby bark before dispersal by water splash, drip, or flow action. Thus, many conidia, or the water in which they were suspended, tended to contact any fungicide previously deposited on the bark surface. The results of in vitro tests (Table 4) in which portions of naturally weathered, fungicide-treated dead twigs were floated for 2 min on a suspension of viable conidia, showed that brief contact with captafol deposits killed more conidia than similar contact with deposits of dithianon or chlorothalonil. Temporary contact of suspended conidia with twigs sprayed only 3 days previously with BCS or benomyl caused no loss of conidia viability.

Redistribution of fungicides.—The study (Table 5) in which inoculum was added to a tree canopy after the spray deposits had dried, indicated that captafol, but not BCS or dithianon, could reduce infection of young shoots on trap plants exposed beneath and out of contact with the sprayed canopy. Evidently, redistribution of captafol in fungitoxic amounts occurred simultaneously with conidia dispersal during the one rain shower to which the first batch of plants was exposed. This showed that redistribution of captafol onto newly exposed tissue does not necessarily have to occur in advance of an infection period to control melanose.

Other studies confirmed that redistribution of captafol prior to an infection period also could have an impact on melanose control. In the 1975 test (Table 6), sufficient captafol had been redistributed from bark and old leaves sprayed before growth commenced to provide rind protection similar to that obtained with the single postbloom spray of BCS or dithianon. In the 1976 test, redistribution of captafol onto fruit again was evident, but apparently to a lesser extent than in the previous year.

DISCUSSION

The results support the long-held contention (7, 9, 11) that copper fungicides are capable of preventing melanose only when applied to the surface of the tissue to be protected. No evidence was obtained that BCS could either reduce the inoculum supply or be redistributed sufficiently to reduce infection at locations away from sites of application.

The major site of action of captafol, if it could be applied safely postbloom, would probably be on the surface of the fruit to which it had been applied. In the present Florida practice of applying captafol before growth commences, the usefulness of the material depends on its redistribution and probably also on some action against conidia as they are washed over the treated bark surface.

Redistribution of captafol was shown to occur even where there was no bridge of leaf or stem material between sprayed parts of the canopy and newly exposed tissue. This agrees with observations by Yamada et al. (12), that when captafol-impregnated hemp was placed above a tree, melanose was controlled on the tree canopy beneath.

Citrus fruit often hang below the supporting limbs and twigs. Therefore, another way in which captafol might be redistributed to fruit in fungitoxic amounts could be by transportation in water that flows over treated bark, down the limbs and twigs onto fruit borne at their extremities.

Dithianon and chlorothalonil evidently acted against *D. citri* mostly as protectants. The superiority of dithianon over chlorothalonil in some tests reported herein and elsewhere (9) could probably be attributed to longer residual activity.

The relatively poor control of melanose provided by benomyl contrasts with the outstanding control that preharvest sprays of this material have given of Phomopsis stem-end rot of mature fruit (1), also caused by *D. citri*. Germ tube production on a benomyl-treated rind surface was inhibited to only a minor extent and many inoculated fruit developed melanose (Fig. 1). Thus, the main effect of benomyl in reducing melanose severity (3, 8, 9) is probably through a reduction in inoculum supply. The results of previous field tests (9) in which comparable control of melanose was obtained, regardless of whether a single spray of benomyl was applied in February (prebloom) or April-May (postbloom), support this contention.

LITERATURE CITED

1. BROWN, G. E. 1968. Experimental fungicides applied preharvest for control of postharvest decay in Florida citrus fruit. Plant Dis. Rep. 52:844-847.
2. DUBIN, H. J., and H. ENGLISH. 1974. Factors affecting control of European apple canker by Difolatan and basic copper sulfate. Phytopathology 64:300-306.
3. FISHER, F. E. 1972. Chemical control of melanose on citrus in Florida. Phytopathology 62:757 (Abstr.).
4. FLORIDA COOPERATIVE EXTENSION SERVICE. 1977. Florida citrus spray and dust schedule. Circ. 393-C, Univ. Fla., Gainesville. 12 p.
5. GILPATRICK, J. D., M. SZKOLNIK, and S. D. GIBBS. 1971. A single high-rate application of Difolatan for the control of apple scab. Phytopathology 61:893 (Abstr.).
6. MOHEREK, E. A. 1970. Disease control in Florida citrus with Difolatan fungicide. Proc. Fla. State Hortic. Soc. 83:59-65.
7. RUEHLE, G. D., and W. A. KUNTZ. 1940. Melanose of citrus and its commercial control. Fla. Agric. Exp. Stn. Bull. 349. 54 p.
8. TIMMER, L. W. 1974. Evaluation of fungicides and application times for control of melanose on Texas grapefruit. Plant Dis. Rep. 58:504-506.
9. WHITESIDE, J. O. 1975. Evaluation of fungicides for the control of melanose on grapefruit in Florida. Plant Dis. Rep. 59:656-660.
10. WHITESIDE, J. O. 1977. *In* Fungicide and Nematicide Tests: Results of 1976. Vol. 32. Am. Phytopathol. Soc., St. Paul, Minnesota. 286 p.
11. WINSTON, J. R., J. J. BOWMAN, and W. J. BACH. 1927. Citrus melanose and its control. U. S. Dep. Agric. Bull. 1474. 62 p.
12. YAMADA, S., H. TANAKA, M. KOIZUMI, and S. YAMAMOTO. 1966. Studies on rationalization and labor saving of fungicide applications for citrus disease control (English summary). Pages 75-104 *in* Bull. Hortic. Res. Stn. (Minist. Agric. For.) Ser. B (Okitsu) Japn. No. 5. 206 p.