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## CONTROL OF GLIOCLADIUM IN CHAMAEDOREA PALMS<sup>1,2</sup>

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**Abstract.** The fungus, *Gliocladium vermoeseni* (Biourge) Thom. causes leaf and stem necrosis of all commercially produced *Chamaedorea* species. Optimum growth of *G. vermoeseni* on potato dextrose agar (PDA) was at 24-27 C. Growth inhibition at 30 C or greater suggests that high temperatures may account for decline in severity of the disease during summer months in Florida. Fungus growth was completely inhibited by 1.0 ppm or greater of benomyl in PDA. Foliar sprays of benomyl, benomyl + mancozeb, and chlorothalonil were used at 0.6g, 1.35g, and 1.34g a.i./liter, respectively. All stems of *C. cataractarum*, *C. elegans*, and *C. seifrizii* developed symptoms when needle-punctured prior to inoculation, but in unwounded, inoculated stems of these palm species symptoms developed in 0/4, 0/8, and 2/4 stems, respectively. Disease was more severe in stems having chlorotic to green leaves removed than in stems having senescent, dry leaves removed prior to inoculation.

Leaf and stem necrosis of *Chamaedorea* palms is a common disorder in Florida. Following infection of sheaths (petiole bases), leaves of affected plants become chlorotic and eventually die. Lower leaves on a plant are usually affected first. When the dead or yellow leaves are removed, the palm stems appear barren and quality is reduced.

The disease is caused by the fungus, *Gliocladium vermoeseni* (Biourge) Thom., (2, 5). This fungus is a serious problem in California where it is involved in the death of

street plantings of Phoenix palms (3). The disease in *Chamaedorea* palms in Florida was described by J. E. Reynolds (5). Only palms wounded prior to inoculation became infected in his tests, but others (4) found wounds were not necessary for infection. The palms Reynolds found susceptible were *C. elegans*, *C. erumpens*, *C. seifrizii*, *C. tenella* and *Chrysalidocarpus lutescens*. Eleven other palm species of 10 genera were resistant to the fungus.

No experimental data on control of the fungus in nurseries has been reported previously. This study was performed with the objective of developing information for control of *G. vermoeseni* in Florida nurseries.

### Methods and Materials

The fungus culture (no. 79-58) used for in vitro and inoculation experiments was isolated from diseased tissue of *C. seifrizii* and then maintained on PDA (potato dextrose agar). For growth studies, 7 mm diameter cores from 72 hr old cultures were transferred to the center of fresh 9 cm diameter PDA plates. Benomyl was incorporated into molten PDA before pouring plates. Final concentrations of benomyl were 0, 0.1, 1, 10, 100, 500, and 1000 ppm. The fungus was incubated on the benomyl-PDA plates at 27 C. Five replications were used for each treatment in all growth studies and radial growth was measured at 96 hr incubation at designated temperatures. Each experiment was performed at least twice.

Inoculum for experiments on *C. seifrizii*, *C. elegans*, and *C. cataractarum* was prepared from cultures grown on V-8 juice agar by the method used by Reynolds (5). Wounds in palms were made by puncturing the outer 2 petiole bases 5 times using a sterilized needle, and inoculum was applied with a sterile cotton swab. Inoculated sites on plants and appropriate controls were enclosed in plastic for 3-10 days following inoculation. Symptom development was observed for 21 days. The influence of needle-wounding on disease development was evaluated in 4-11 month old palms while removal of senescent vs. green leaves was compared in plants approximately 2.5 years old.

Fungicides were evaluated in a commercial nursery on 6 month old *Chamaedorea seifrizii* growing in 14-inch diameter pots. A benomyl soil drench (0.6g a.i./liter, 0.5 liters/pot) at 30-day intervals; benomyl, mancozeb, benomyl + mancozeb, and chlorothalonil sprayed at 7-day intervals; and benomyl + mancozeb sprayed at 14-day intervals were applied during May 25 to July 13, 1979. Concentrations of benomyl, mancozeb and chlorothalonil sprayed alone or combined were 0.6g, 1.35g, and 1.34g a.i./liter, respectively.

Senescent and diseased leaves were removed from stems in treatments A through G and treatment I (Table 2) be-

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<sup>2</sup>Mention of fungicide names in this study does not imply endorsement or registration for use in *Chamaedorea* palms. For English conversions of metric units, see the table provided at the beginning of these proceedings.

fore beginning fungicide treatments. After the first evaluation senescent and diseased leaves were removed as before and the fungicide treatments continued. Plants were arranged in a randomized complete block design with 10 replications and 5 pots per replication. Only 6 replications per treatment were available at the second evaluation. The plants were evaluated by determining the percent of stems having petiole bases colonized by the fungus as evidenced by presence of conidia. Evaluations were made on June 18 and July 27, 1979.

## Results and Discussion

Radial growth of the fungus was greatest at 24-27 C (Table 1). Temperatures of 18 C and 30 C strongly suppressed growth and no growth occurred at 33 C. Radial growth of the fungus was 44 mm on both benomyl-free PDA and PDA amended with benomyl at 0.1 ppm. The fungus did not grow where benomyl concentrations were 1.0 ppm or greater.

Table 1. Mean radial growth of *Gliocladium vermoeseni* on PDA after 96 hr at selected temperatures.

	Temperature (°C)				
	18	24	27	30	33
Radial growth (mm):	19.8	38.1	38.3	17.8	0

At the first foliar spray evaluation, applications of benomyl, benomyl + mancozeb, or chlorothalonil at 7-day intervals and benomyl + mancozeb at 14-day intervals each reduced disease incidence significantly (Table 2). Benomyl drenches, sprays of mancozeb alone, and alternating benomyl and mancozeb sprays did not provide good disease control. Removal of diseased and senescent leaves before beginning the experiment did not significantly reduce disease incidence in the nonfungicide treatment I.

Table 2. The percent of *Chamaedorea* palm stems diseased by *Gliocladium vermoeseni*.

Treatment	Mean percent	
	6/18/79	7/27/79
A. Benomyl spray @ 7-day interval	8.52a <sup>z</sup>	3.93a
B. Benomyl + mancozeb spray @ 14-day interval	9.36a	0.00a
C. Benomyl + mancozeb spray @ 7-day interval	9.79a	2.50a
D. Chlorothalonil spray @ 7-day interval	11.50ab	2.60a
E. Alternating benomyl and mancozeb spray @ 7-day interval	14.62abc	3.62a
F. Benomyl drench @ 30 day interval	17.01abc	3.70a
G. Mancozeb spray @ 7-day interval	20.44 bc	4.25a
H. Nonremoval of lower leaves & benomyl + mancozeb spray at 7-day intervals	22.07 bc	6.08a
I. No fungicide	24.49 cd	6.27a
J. Nonremoval of lower leaves & no fungicides	33.22 d	33.95 b

<sup>z</sup>Means not followed by a common letter are significantly different from each other by Duncan's Multiple Range Test ( $P \leq 0.05$ ).

At the second evaluation disease incidence in all fungicide treatments, A-H, and the no fungicide + leaf removal treatment I was significantly lower than in the no fungicide + no leaf removal treatment, J. Treatments I and J are particularly interesting. The very low level of disease in I at the second evaluation was not due to fungicide applications and the plants retaining all leaves, J, exhibited the same level of disease as at the first evaluation. These data

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suggest that the disease was not active during that period. Interviews with nurserymen have indicated that a decline in incidence of this disease occurs during the summer months. The mean high temperatures for June through September in the Palm Beach to Miami area range from 31-32 C (1) which is above the temperature that induces strong growth inhibition of *G. vermoeseni* in vitro (Table 1). High summer temperatures in Florida may inhibit activity of *G. vermoeseni* and be responsible for the reduced incidence during summer months.

Reynolds' procedure (5) of wounding and not wounding stems with a needle prior to inoculation was utilized to inoculate 3 species of *Chamaedorea*. Four of 4 *C. catartarum*, 8/8 *C. elegans*, and 4/4 *C. seifrizii* developed symptoms after wounding and inoculating. However, 0/4, 0/8, and 2/4 unwounded palms of the same species, respectively, developed symptoms after inoculation.

The influence of removal of senescent, dry petiole bases (sheaths) compared to removal of chlorotic to green sheaths on disease development in *C. seifrizii* is summarized in Table 3. The indicated leaves were removed prior to inoculation. Mechanical damage was readily apparent on most of the uninoculated stems from which green sheaths were removed but not on stems from which senescent sheaths were removed. Symptoms of disease were most abundant and severe in stems from which green sheaths had been removed prior to inoculation. Some stems with senescent sheaths removed also developed symptoms. A benomyl spray applied 30 minutes before inoculation completely protected stems where senescent sheaths were removed but did not protect stems where green sheaths were removed.

Table 3. Stems showing symptoms 3 weeks following petiole base removal and inoculation with *Gliocladium vermoeseni*.

Treatment	Stems with symptoms Petiole type removed	
	Brown	Green
Not inoculated	0/5	1/5 (necrosis)
Inoculated	3/5	5/5 (necrosis, ooze, spores)
Inoculated after benomyl spray	0/5	3/5 (necrosis, ooze)

Results of our studies indicate that wounds on stems of *Chamaedorea* spp. are good infection sites for *G. vermoeseni*. Nursery operations that cause wounds should be avoided or modified to minimize the amount of injury to palms. Of particular concern is the removal of chlorotic and senescent leaves during periods when the fungus is active. Restricting leaf removal to periods when temperatures exceed 30 C should minimize the chance of infection. If leaf removal is performed during periods favorable to the fungus, a fungicide application immediately after leaf removal may be warranted to minimize infections.

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