The Effect of Peanut Leafspot Fungicides on the Nontarget Pathogen, Sclerotium rolfsii

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ABSTRACT

White mold damage (caused by *Sclerotium rolfsii*) was visually assessed in Florunner peanut plots sprayed with fungicides for control of *Cercospora* spp. during two growing seasons. Although the fungicides gave similar leafspot control, unsprayed plots had consistently lower levels of white mold; those sprayed with benomyl consistently had the highest, and other fungicides were intermediate. Regression analysis revealed that both *S. rolfsii* and *Cercospora* were causing yield loss, but that losses due to each were not related.

Additional key words: Trichoderma, disease interaction.

In vitro tests indicated that the treatment responses could be attributed to a direct effect of the fungicides on *S. rolfsii,* or indirectly by affecting *Trichoderma viride,* a natural antagonist to *S. rolfsii.* Highest field levels of *S. rolfsii* were found for those fungicides with no toxicity to *S. rolfsii,* but which were toxic to *T. viride.* Leaf loss caused by high levels of *Cercospora,* or by mechanical removal of leaves, resulted in lowest levels of *S. rolfsii.*

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Since 1970, peanut farmers in the Southeastern USA have become increasingly aware of losses to white mold (Southern stem blight) disease caused by *Sclerotium rolfsii* Sacc. During this period, 80-90% of the farmers adopted the systemic fungicide benomyl for season-long control of peanut leafspots caused by *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. & Curt.) Deight. Benomyl afforded almost complete control of peanut leafspot when applied at 14-day intervals at 0.39 kg per hectare (ha) throughout the season. Maintenance of a virtually intact peanut canopy made at least three major changes to the ecology of soil-borne fungi: (i) few leaves were lost to the soil surface to serve as organic food sources for *S. rolfsii;* (ii) fungicides were prevented from reaching the soil by the "umbrella effect" of the intact canopy; (iii) an altered sub-canopy environment was created which may be stimulatory to soil-borne fungi. Previous workers (1, 2, 7) indicated that *S. rolfsii* is more severe when defoliation provides an organic food base. Those studies were made when

TABLE 1. Percent infection of Florunner peanuts by *Cercospora* and *Cercosporidium* following treatment with foliar fungicides

Treatment	Infection (%)				Defoliation mean[a] (%)
	1972	1973a	1973b	Mean[a]	
Untreated control	87.1	48.3	80.7	70.0	37.4
Benomyl	8.2	11.2	13.0	11.0	5.9
Chlorothalonil	9.5	7.9	8.3	8.4	5.9
CuOH + S	28.1	8.8	4.8	12.1	6.4
LSD ($P = 0.05$)	7.3	8.2	10.4	5.0	12.7
LSD ($P = 0.01$)	10.3	11.0	14.3	6.6	18.5

[a]Mean weighted for the number of replications in each experiment.

TABLE 2. Yield [kg/hectare (ha)] and dollar-value per metric ton of peanuts treated with fungicides for leafspot control

Treatment	Yield (kg per ha)				Mean value per metric ton[a] ($)
	1972	1973a	1973b	Mean[a]	
Untreated control	3,450	2,480	2,675	2,765	336.92
Benomyl	4,050	2,540	3,285	3,109	311.99
Chlorothalonil	4,190	3,320	3,840	3,705	315.93
CuOH + S	3,895	2,890	3,685	3,356	321.75
Thiophanate methyl	4,005	2,545	2,865	2,995	307.78
LSD ($P = 0.05$)	601	365	581	265	24.24
LSD ($P = 0.01$)	842	491	799	352	...

[a]Mean weighted for number of replications in each experiment.

TABLE 3. Number of dead plants per 30 m of row showing signs of *Sclerotium rolfsii* in peanut field plots following application of *Cercospora* leafspot fungicides

Treatment	Dead plants per 30 m row			
	1972	1973a	1973b	Mean[a]
Untreated control	2.17	1.88	1.80	1.92
Benomyl	3.75	6.12	4.00	4.94
Chlorothalonil	2.83	3.88	3.40	3.49
CuOH + S	3.17	2.00	2.60	2.45
Thiophanate methyl	1.50	5.12	1.80	3.29
LSD ($P = 0.05$)	2.68	3.23	2.10	1.46
LSD ($P = 0.01$)	3.76	3.47	2.90	1.94

[a]Mean weighted for number of replications in each experiment.

effective fungicides for leafspot control were not available, and were of necessity performed under high levels of defoliation. Increased levels of *S. rolfsii* damage in fields with excellent leafspot control indicate that something other than a food base of leaf litter is involved in white mold severity.

The present study was made to determine: (i) the significance of defoliation on severity of white mold; (ii) the importance of canopy in shielding the soil from foliar fungicides, and the contribution of sub-canopy environment to disease severity; and (iii) the relationship between the incidence of *S. rolfsii* and the toxicity of leafspot fungicides to this pathogen and the natural antagonist *Trichoderma viride* (Pers. ex. Fr.).

MATERIALS AND METHODS.—During the 1972 and 1973 seasons, plots sprayed with fungicides for control of peanut leafspot were evaluated for white mold severity and leafspot damage. The 1972 test included the following fungicides and rates applied on a 14-day schedule: benomyl 50% WP, 0.39 kg/ha; thiophanate methyl 70% WP, 0.52 kg/ha; copper hydroxide + sulfur 27 + 15.5% flowable 4.7 liter/ha; chlorothalonil 75% flowable 1.7 liter/ha, and a nontreated control. Plots were 7.3×45.8 m, with eight single-rows. Fungicides were applied with conventional ground, airplane and low-volume ground sprayer. Each application method × fungicide was replicated four times. Two tests were conducted in 1973. The first test (1973a) was the same as the 1972 test, except that treatments were replicated five times, were sprayed with a conventional ground sprayer, and plot length was 15.2 m. The second test conducted in 1973 (1973b) was replicated eight times and contained all fungicidal treatments applied by a conventional sprayer to 15.2 m plots. Two additional treatments were added: a standard benomyl program was sprayed 55 days after planting with 0.52 kg/ha of 85% succinic acid 2,2-dimethyl-hydrazide (a hormone affecting internode length), and 0.26 kg/ha at 14-day intervals thereafter for the next 8-weeks; a second benomyl treatment was "clipped" 90 days after planting with a rotary mower to remove nonbearing vertical runners. The clipping was timed for a period of low photosynthetic (leaf) demand (3) and was adjusted to give maximum exposure of the soil in the crown region without damaging the bearing runners.

Leafspot incidence (*Cercospora* + *Cercosporidium*) was determined 14 days before harvest by removing 10 vertical runners at random from each plot and determining infection using the following criteria: (i) total leaflets = number of leaf nodes × 4; (ii) percent defoliation = number of leaflets lost ÷ total leaflets × 100; (iii) total leaflets infected = number of leaflets lost + number of leaflets infected; and (iv) percent infection = leaflets infected ÷ total leaflets × 100. This method assumes that defoliation occurs because of previous leafspot infection. *S. rolfsii* damage was determined by counting the number of dead plants in the two center rows of each plot showing

TABLE 4. Comparison of disease occurrence in peanut field plots with growth of *Sclerotium rolfsii* and *Trichoderma viride* on fungicide-amended potato-dextrose agar (PDA)

Fungicide treatment	Rate (μg/ml)	No. dead plants	Growth[a]	
			S. rolfsii	*T. viride*
PDA control	...	1.92	15.3	20.5
Benomyl	0.5	4.94	16.2	4.5
	5.0	...	14.8	0.0
Chlorothalonil	0.5	3.49	9.0	12.0
	5.0	...	4.0	4.8
CuOH + S	50.0	2.45	15.8	11.8
	250.0	...	13.0	3.8
Thiophanate methyl	0.5	3.29	16.2	20.0
	5.0	...	16.8	5.5

[a]Radial growth in mm (longest axis + shortest axis) ÷ 2 on PDA culture plate; average of five replications.

signs of the fungus (4). Counts were made 10 days before these same two rows were dug for yield. Harvested peanuts were graded for quality (value per metric ton) according to current Federal-State Inspection Service standards (8).

Fungicides were incorporated into potato-dextrose agar (PDA, Difco) at concentrations of 0.5 and 5.0 μg/ml, except copper hydroxide + sulfur at 50 and 250 μg/liter (based on CuOH). Twenty ml of each medium and a no-fungicide control were poured into 90-mm diameter petri dishes. Each treatment was represented by 10 replications, five of which were inoculated with one disk (7-mm diameter) of *T. viride* and five with *S. rolfsii*. Inoculum disks were removed from the periphery of 48-hour-old PDA-grown cultures. Radial growth of the fungi was measured 36 hours after inoculation.

Regression equations relating peanut yields to percent leafspot infection, percent defoliation and to white mold damage for plots receiving fungicides were calculated. Regressions between white mold damage and leafspot infection and defoliation were also computed (6).

RESULTS.—Field tests revealed only minor differences in peanut leafspot control among fungicide treatments (Table 1). Multi-year analyses indicated that these small differences in leafspot control reflected disproportionate differences in peanut yield and quality (Table 2). Numbers of dead plants killed by *S. rolfsii* were significantly different ($P = 0.01$) between treatments (Table 3).

Laboratory studies indicated that the foliar fungicides differed greatly in effects on *S. rolfsii* and its antagonist *T. viride* (Table 4). In agar medium, benomyl was the only fungicide which displayed little or no effect on the pathogen, but toxicity to the antagonist. The fungi showed intermediate responses to other fungicides.

Clipping or hormonal treatment of benomyl-treated plots reduced *S. rolfsii* damage (Table 5). Severity of *S. rolfsii* infection was significantly affected by the type of spray equipment used to deliver the test fungicide (Table 6).

DISCUSSION.—A preliminary report in 1973 by Smith (5) indicated that peanut leafspot fungicides did not control *Sclerotium rolfsii* in peanut fields. This study supports these observations and points out major differences between the fungicide used and the frequency of *S. rolfsii* damage. Regression analyses of field data

TABLE 5. The effect of foliage clipping benomyl and a growth-regulating hormone on dead plants (*Sclerotium rolfsii*) and yield in peanut field plots

Treatment	Dead plants (No. per 30 m of row)	Yield (kg/ha)
Benomyl	7.2	2540
Benomyl-hormone	5.7	2246
Benomyl-clipped	3.8	2438
LSD ($P = 0.05$)	3.23	NS
LSD ($P = 0.01$)	3.47	NS

TABLE 6. The relationship of peanut plants killed by *Sclerotium rolfsii* to the method of application of foliar fungicides

Application method	Dead plants (no. per hectare)
Conventional ground	392 A[a]
Airplane	536 AB
Low-volume, ground	639 B

[a]Means followed by the same letter are not significantly different ($P = 0.05$) using Duncan's multiple range test.

from fungicide-treated plots revealed that percent defoliation and peanut leafspot infection were unrelated to *S. rolfsii* damage. For peanut yields in the range of the three experiments, a 1% increase in leafspot infection decreased yield 12.9 ± 4.9 kg/ha; an increase in *S. rolfsii* damage of one dead plant per 30 m of row, decreased yield 63.6 ± 14.4 kg/ha, independent of leafspot infection. Both regressions were linear.

The importance of leaf retention in increasing *S. rolfsii* damage can be determined by comparing the untreated control plot to those receiving fungicide treatments. In all cases the control, which had a high level of leafspot infection (defoliation), had the least *S. rolfsii* damage. This observation is reinforced by comparing benomyl plots to benomyl plots that had been clipped (mechanically defoliated) or hormone-treated (plant size reduced without defoliation). The clipped plots receiving the same amount of fungicide as the benomyl plots had significantly less ($P = 0.01$) *S. rolfsii* damage. *S. rolfsii* damage was reduced to a level similar to the control by clipping fungicide-treated plots. All experiments reported

here were conducted during dry growing conditions (30 days or more of drought after blooming). In our opinion, under dry conditions an intact canopy creates and maintains a humid atmosphere which is more conducive to fungal growth and pathogenicity; the more defoliated control plots would be subject to greater fluctuations in soil moisture and would not have enough leaves to maintain a humid atmosphere. This would be obviated in peanuts grown under irrigation or during wet seasons.

Differences between fungicide plots in levels of damage from *S. rolfsii* can be related to two distinct biological activities (Table 4): effects on the pathogen, and/or its natural antagonist *Trichoderma* spp. Benomyl-treated plots had the most severe incidence of *S. rolfsii* disease. Benomyl exhibited no in vitro effect on *S. rolfsii,* while exhibiting a toxic effect on *Trichoderma.* The related benzimidazole, thiophanate methyl, was equally innocuous to *S. rolfsii* but displayed only mild toxicity to *Trichoderma.* The significant difference ($P=0.05$) in field levels of *S. rolfsii* between these two treatments indicates a probable role for *Trichoderma* in reducing *S. rolfsii* damage under natural conditions. Fungicides with a direct toxic effect to *S. rolfsii* in the laboratory (e.g., chlorothalonil), did not show the field reduction of incidence in *S. rolfsii* that might have been inferred from laboratory data. Chlorothalonil should have had the lowest damage because it was the most toxic fungicide to *S. rolfsii* and least toxic to *Trichoderma.* The fact that it did not perform as expected may indicate the importance of a complete canopy as well as point to other soil ecological factors that may play a role in *S. rolfsii* severity. In addition, data not presented here indicates that chlorothalonil is an excellent contact fungicide, but is inactive once it contacts the soil.

Table 2 indicates that peanuts from plots sprayed with leafspot fungicides had less value per ton than the control. Since value is a quality determination based on fungal damage, maturity, etc., a probable increase in other soil-borne fungal diseases is indicated for fungicide-treated peanuts.

Importance of the umbrella effect in shielding the sub-canopy soil was illustrated in comparisons of spray equipment. The conventional ground sprayer with three nozzles per row operating at 4.2 kg/cm^2, discharges directly into the canopy. The airplane and low-volume ground sprayer lack this propulsive force and rely on gravity settling the fungicide onto the peanut plant. The lower frequency of *S. rolfsii* damage on conventional plots (Table 6) is interpreted as a reflection of the amount of product penetrating to the soil surface.

These data indicate that fungicides should be evaluated for effects on nontarget organisms as well as the target pathogen before disease control recommendations are made.

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