Field, Microplot, and Greenhouse Evaluations of Resistance to *Sclerotium rolfsii* in Peanut

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ABSTRACT

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Three of 12 virginia peanut (*Arachis hypogaea*) genotypes evaluated in the field for resistance to *Sclerotium rolfsii* had few disease loci and low ratios of dead plants to disease loci. Two of these genotypes (NC 2 and NC Ac 18016) had upright canopies. NC 2 was tall and bushy and NC Ac 18016 was very compact. Another field-resistant genotype (NC Ac 18416) was tall but had a more spreading canopy than NC 2 or NC Ac 18016. NC Ac 18416 also showed partial resistance to infection in the greenhouse. A soil temperature of 28 C and high humidity near the moist soil surface were highly conducive to disease development in the greenhouse. In field microplots, fewer sclerotia were recovered after NC 2 was grown than after NC Ac 18016 and NC Ac 18416 were grown. Differences in results in field, microplot, and greenhouse trials may have been caused by different resistance components in the genotypes evaluated.

Stem rot of peanut (*Arachis hypogaea* L.) is caused by the soilborne fungus *Sclerotium rolfsii* Sacc., which has a very wide host range and worldwide distribution in warm climates (1). The necrotrophic behavior of *S. rolfsii* probably accounts for the universal susceptibilty of cultivated peanut to stem rot (15). Mycelia from germinated sclerotia of *S. rolfsii* usually colonize dead or senescent plant tissues on the soil surface before infecting healthy plants. Colonized food bases supply energy for invasion of living plant organs and bridge distances between germinated sclerotia and host (7). Availability of dead or senescent plant tissue and a warm moist environment near the base of the host plant favor disease development (18).

Low incidence of plant diseases caused by necrotrophic soilborne pathogens has been associated with plant growth habit (6,8,12,17). Open or compact canopies allow greater penetration of sunlight and improved air movement, resulting in a drier, hotter microclimate near the soil surface (6). Plants with an upright growth habit also have fewer stems and senescent leaves touching soil and inoculum. Resistance to *S. rolfsii* in peanut has been associated with growth habit (erect, semierect, or runner) by some investigators (9,14) but not by others (11).

Characterization of components of resistance to *S. rolfsii* in peanut should discriminate between disease escape associated with canopy type (phenological suppression) and resistance associated with structural barriers or active responses of the plant to infection (metabolic resistance). Different mechanisms of suppression of *Sclerotinia sclerotiorum*, a necrotrophic soilborne pathogen, were detected in field and greenhouse evaluations of bean (10,13,17). Phenological suppression of disease was best detected in field plots where differences in canopy structure were most fully expressed (10). Metabolic resistance was also expressed in the field but was confounded with phenological suppression of disease (10). Conversely, screening in highly conducive environments in the greenhouse negated the effects of crop canopy, allowing detection of metabolic resistance only (10,13).

Resistance that limits end-of-season populations of the pathogen is difficult to detect in the field because of uneven distribution of initial inoculum. Field soils in microplots may be infested with known populations of *Sclerotium rolfsii* and allow monitoring of inoculum density-disease relationships on partially resistant genotypes.

The purpose of this study was to evaluate components of partial resistance to *S. rolfsii* in peanut by field, microplot, and greenhouse methods.

MATERIALS AND METHODS

Genotypes evaluated. Twelve entries of virginia-type peanuts were evaluated in field tests in 1984 and 1985 (Table 1). Three entries (NC 6, NC 7, and Florigiant) are standard susceptible genotypes. Five genotypes (entries 3–7) are advanced-generation selections from crosses for resistance to Cylindrocladium black root rot (CBR). Resistance to stem rot has been correlated with CBR resistance in CBR screening trials (5). NC 8C is a commercial cultivar with partial resistance to CBR and slightly greater resistance to stem rot than the standard commercial cultivar, Florigiant (19). Moderate resistance to stem rot was previously reported in NC 2 (9) and NC Ac 18016 (19). NC Ac 18416 and NC Ac 18417 are selections from NC 8C × Florigiant and NC Ac 18016 is a selection from NC 3033 × NC 9088. Several genotypes are small-seeded and low yielding, making them unsuitable for yield trials or production for current North Carolina markets.

Field tests. Two fields on the Peanut Belt Research Station in Lewiston, NC, were used for screening genotypes for resistance to *S. rolfsii*. Fields were planted to corn the year before use and had a history of stem rot. Entries were planted in four replicate two-row by 6.4-m plots in May 1984 or in 10 replicate two-row by 7.3-m plots in May 1985. Rows were 0.9 m apart, and treatments were arranged in randomized complete block designs. Recommended cultural practices were followed for land preparation and fertilization and for control of weeds and insects. Leaf spot caused by *Cercospora arachidicola* and *Cercosporidium personatum* were controlled by regular foliar application of recommended fungicides. Natural inoculum of *S. rolfsii* in the fields was supplemented by distributing 150 cm^3 (1984) or 60 cm^3 (1985) of dried, colonized oat grains over each plot during the first week of August and again 3 wk later. *S. rolfsii* infection sites (disease loci) were counted on 29 August and 27 September in 1984 and on 28 August, 12 September, and 30 September in 1985. Infections less than 30 cm apart were counted as one disease locus (16); 42 and 48 loci per plot (12.8 and 14.6 m of row) were possible in 1984

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and 1985, respectively. Dead plants were also counted on 12 and 30 September 1985. Canopy height and row width were measured at a representative site within plots on each rating date. End-of-season inoculum density was determined from soil sampled on the final assessment date in 1985. Soil cores (2.5 cm diameter × 15 cm deep) were taken in an alternating pattern along each two-row plot and pooled to obtain a sample of 600–800 g. Soil samples were placed in polyethylene bags and stored in insulated containers for no more than 5 days before assay. A 400-g portion of each soil sample was processed in a semiautomatic elutriator and assayed for viable sclerotia (18). Known numbers (0–8) of *S. rolfsii* sclerotia were added to 20 400-g samples of noninfested soil that were processed similarly as a check of the assay efficiency. Eighty-one percent of sclerotia placed in check samples were recovered, and sclerotial counts had a C.V. of 8.3%.

Microplot test. Ten to 12 seeds of four genotypes (NC Ac 18016, NC Ac 18416, NC 2, and Florigiant) were planted in 0.9-m-diameter microplots (2) located on the Upper Coastal Plain Research Station near Rocky Mount, NC, on 24 May 1985. Plants were later thinned to four per plot. Plots were treated with carbofuran (3.4 kg a.i./ha) and alachlor (3.4 kg a.i./ha) at planting, and chlorothalonil (1.2 kg a.i./ha) was applied to foliage at 2- to 3-wk intervals to control leaf spots. Plots were drench-irrigated as needed to maintain soil moisture at favorable levels. Plots were infested with 6, 25, 100, or 400 culturally produced, nonsterile sclerotia (4) on 3 July. Similar sclerotia were plated on water agar to test viability; 60 of 60 germinated. Four replicates of four peanut genotypes × four inoculum densities were arranged in a randomized complete block design. On 11 October, plants were dug, inverted, and evaluated for damage by *S. rolfsii*. Numbers of stem rot lesions, length of longest lesion, and percent pod rot were recorded for each plot. Soil (600–700 g) was sampled from each plot by pooling several 15-cm-deep × 2.5-cm-diameter cores. Soil samples were elutriated and assayed for viable sclerotia.

Greenhouse test. Four entries (NC Ac 18016, NC Ac 18416, NC 2, and Florigiant) were grown in 15-cm-diameter pots in water bath temperature tanks adjusted to 24 or 28 C. Pots contained a 1:2 mixture (v/v) of greenhouse potting mix (W. R. Grace and Co, Cambridge, MA) and pasteurized sandy loam soil (v/v) plus commercial *Rhizobium* inoculum (cowpea group; Keel Peanut Co., Greenville, NC). Ten sclerotia of *S. rolfsii* were added to pots 8 wk after seeding. After inoculation, pots were either wrapped around the top with two layers of cheesecloth or not wrapped. The cheesecloth was wet constantly by contact with water in temperature tanks, creating a humid environment near the soil surface and plant crowns in wrapped pots. Wrapped or unwrapped pots were either watered every 1–2 days with 500 ml of water to maintain soil moisture or plants were allowed to wilt between waterings. Treatments were arranged in a factorial design. The experimental design was a split plot, with entries, cheesecloth, and watering treatments randomized within three replicate whole plots (temperature). Plants were harvested 30 days after inoculation and lesions were counted. The three longest lesions on each plant were measured and the average lesion length calculated.

RESULTS

Field tests. Genotypes were ranked similarly in 1984 and 1985 despite large differences between the two years in mean numbers of stem rot loci (Table 1). Mean final disease incidence was about 12% in 1984 and 65% in 1985. Differences among genotypes were not significant on the first assessment date in 1984 but were significant ($P \leq 0.01$) for all remaining assessments. Disease incidence increased with time, and performance of genotypes was consistent among assessment dates. NC Ac 18016, NC 2, and NC Ac 18416 had fewer disease loci than the standard cultivar Florigiant in both years (Table 1). Genotypes with fewer disease loci per row-meter also had fewer dead plants per row-meter and a lower ratio of dead plants to disease loci (Table 2). The standard susceptible cultivars NC 6, NC 7, and Florigiant had the highest ratios of dead plants to disease loci. Genotypes also differed in canopy height and width ($P \leq 0.05$, Fig 1). NC 8C and NC Ac 18417 have short, spreading canopies. In contrast, NC 2 has a tall, bushy growth habit. NC Ac 18416 is also taller than the standard Florigiant. Rows of NC Ac 18016 are very narrow, and the canopy never closes. The remaining genotypes have spreading (runner) growth habits similar to Florigiant.

Sclerotial populations varied among

Table 1. Disease incidence on 12 peanut genotypes evaluated for resistance to *Sclerotium rolfsii* in North Carolina in 1984 and 1985 field tests

		Untransformed		Transformed[a]	
Entry	Identity	1984[b]	1985[c]	1984	1985
1	NC Ac 18016	0.02	0.68	0.07	0.80
2	NC 8C	0.29	2.24	0.52	1.49
3	NC Ac 17921 × 17969	0.64	2.38	0.76	1.52
4	NC Ac 17922 × NC 8C	0.33	2.60	0.57	1.61
5	NC Ac 17921 × 18016	0.41	2.51	0.61	1.57
6	NC Ac 18416	0.14	1.20	0.29	1.08
7	NC Ac 18417	0.31	2.43	0.55	1.55
8	NC 2	0.16	1.78	0.38	1.29
9	NC 6	0.64	2.56	0.79	1.56
10	NC 7	0.55	2.54	0.73	1.58
11	Florigiant	0.47	2.28	0.68	1.50
12	NC 9	0.37	2.41	0.60	1.54
Mean		0.36	2.113	0.55	1.43
LSD ($P = 0.05$)		···	···	0.24	0.19

[a] Y = square root (disease loci); statistical analyses performed on transformed data only.
[b] Mean disease loci per row-meter in four replicate two-row × 6.4-m plots on 27 September 1984.
[c] Mean disease loci per row-meter in 10 replicate two-row × 7.3-m plots on 30 September 1985.

Table 2. Number of dead plants per row-meter and ratio of dead plants to disease loci in 12 peanut genotypes screened for resistance to *Sclerotium rolfsii* in field plots in 1985[a]

		Untransformed		Transformed	
Entry	Identity	Dead	Ratio	Dead[b]	Ratio[c]
1	NC Ac 18016	0.02	0.03	0.06	0.08
2	NC 8C	0.29	0.12	0.46	0.30
3	NC Ac 17921 × 17969	0.38	0.15	0.52	0.34
4	NC Ac 17922 × NC 8C	0.40	0.16	0.58	0.37
5	NC Ac 17921 × 18016	0.40	0.16	0.59	0.39
6	NC Ac 18416	0.03	0.02	0.07	0.05
7	NC Ac 18417	0.32	0.13	0.54	0.36
8	NC 2	0.05	0.02	0.12	0.08
9	NC 6	0.66	0.22	0.71	0.43
10	NC 7	0.75	0.27	0.79	0.51
11	Florigiant	0.50	0.20	0.64	0.43
12	NC 9	0.38	0.15	0.55	0.36
Mean		0.35	0.13	0.47	0.31
LSD ($P = 0.05$)		···	···	0.21	0.14

[a] Numbers represent means of 10 replicate two-row × 7.3-m plots. Statistical analyses performed only on transformed data.
[b] Y = square root (number of dead plants per meter).
[c] Y = arc sine [square root (dead plants per plot/disease loci per plot)].

replicates ($P \leq 0.01$); genotype effects were less consistent ($P = 0.07$), with lowest inoculum densities recovered from plots planted with NC 8C (four sclerotia per kilogram of soil). Highest inoculum densities were recovered from plots planted with NC 7 and averaged 26 sclerotia per kilogram of soil.

Microplot test. Fewest sclerotia were recovered from soil in plots planted with NC 2 (Table 3), and final inoculum densities were greatest in plots planted with NC Ac 18016 or NC Ac 18416. The four genotypes did not differ significantly ($P = 0.05$) in lesion numbers, lengths, or pod rot. Plants had an average of six lesions, with the longest lesion averaging 90 mm, and a mean pod rot rating of 22%. The greatest numbers of stem lesions developed in plots initially infested with 100 sclerotia and mean longest lesions developed in plots infested with 25 sclerotia (Table 4). Plots infested with 100 or 400 sclerotia had the highest final inoculum densities (Table 4). Effects of initial inoculum density on lesion numbers, lesion lengths, and final inoculum density were highly significant ($P \leq 0.01$). All disease variables responded to initial inoculum density independently of genotype.

Greenhouse test. At soil temperatures of 24 and 28 C, 33 and 67% of plants were infected, respectively. At 28 C, 76% of plants in pots wrapped with wet cheesecloth had lesions, compared with 41% in pots that were not wrapped. Mean lesion lengths in wrapped and unwrapped pots at 28 C were 39 and 15 mm, respectively. At 28 C, 70% of plants in pots watered every 1–2 days had lesions, and mean length of longest lesions was 34 mm. Mean lesion length on plants that wilted before watering was 20 mm, and 48% of plants had lesions. All differences among cheesecloth and watering treatments were significant ($P \leq 0.05$). In the warmer soil, more lesions developed on NC 2 and NC Ac 18016 than on NC Ac 18416 or Florigiant (Table 5). Effects of cheesecloth treatment, watering frequency, and genotype at 28 C were additive.

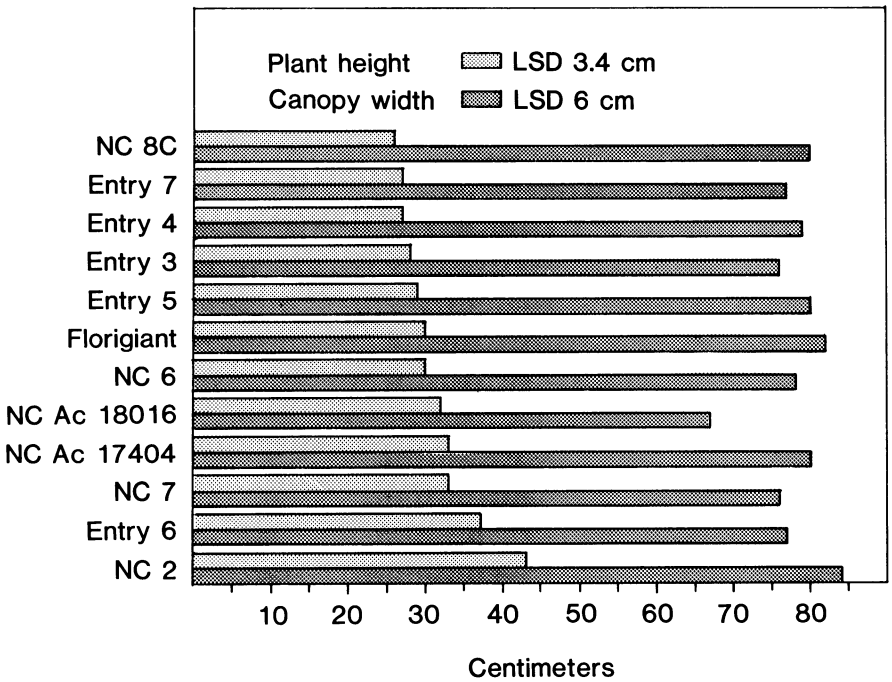

Fig. 1. Mean plant heights and row widths of 12 peanut genotypes evaluated for resistance to *Sclerotium rolfsii* in North Carolina in 1984 and 1985. Entry 3 = NC Ac 17921 × 17969, entry 4 = NC Ac 17922 × NC 8C entry 5 = NC Ac 17921 × 18016, entry 6 = NC Ac 18416, and entry 7 = NC Ac 18417.

Table 3. Inoculum densities of *Sclerotium rolfsii* on 11 October 1985 in microplots planted with four peanut genotypes[a]

Entry	Identity	Sclerotia/kg soil	Transformed[b]
1	NC Ac 18016	9	7.7
6	NC Ac 18416	8	7.7
8	NC 2	3	3.2
11	Florigiant	5	5.7
Mean		6	6.0
LSD ($P = 0.05$)		...	3.1

[a] Means of four replications and initial inoculum density treatments of 6, 25, 100, or 400 sclerotia per plot; 400 g of soil was assayed from each microplot.
[b] Y = square root (sclerotia/kg soil). Statistical analysis performed only on transformed data.

DISCUSSION

Characterization of components of partial resistance to *Sclerotium rolfsii* could improve efforts to select peanut genotypes that suppress stem rot. Field trials alone do not allow distinctions between phenological and metabolic components of resistance, but comparisons of field, microplot, and greenhouse trials may help to identify different resistance components.

Metabolic resistance to *S. rolfsii* can be selected only if environment is equally conducive to disease under all canopy types, thus eliminating the effects of crop phenology on disease suppression. Warm, moist soils and high humidities near the soil surface were most conducive to disease development in the greenhouse, allowing detection of metabolic resistance in NC Ac 18416. In the greenhouse, NC Ac 18016 and NC 2 were less resistant to *S. rolfsii* than NC Ac 18416 or Florigiant. In the field, however, NC 2 and NC Ac 18016 suppressed disease development equally as well as NC Ac 18416 and better than Florigiant. These comparisons suggest that disease suppression in NC 2 and NC Ac 18016 was caused primarily by crop phenology.

NC 2 and NC Ac 18016 had growth habits that differed from each other and from the spreading habits of the susceptible genotypes. The narrow canopy of NC Ac 18016 was unique and created a hot, dry microenvironment unfavorable for infection. NC 2 was more similar to the ideotype of *Sclerotinia*-resistant bean (17), with a large but porous canopy. Resistricted limb contact in both upright genotypes may have inhibited limb-to-limb spread of disease within and between plants, thus limiting both disease increase and plant mortality. On the other hand, high ratios of dead plants to disease loci on Florigiant were attributed to a closed canopy and close limb-to-limb contact, which favored disease increase, lesion expansion, and high mortality.

All genotypes evaluated in this study were derived from virginia-type peanuts. Comparisons of peanut genotypes across botanical groups may account for some conflicting reports on relative susceptibilities of plants with upright or spreading growth habits (9,11,14). Among botanical groups of peanuts, valencia types (which are upright) are least resistant to *S. rolfsii*, spanish types (also upright) are more resistant, and virginia types (which can be upright or spreading) are most resistant (15). Symptoms are also easier to detect on upright plants under some growing conditions (11). The relative importance of plant type in determining incidence of *S. rolfsii* when peanut genotypes from different botanical groups are compared

needs to be investigated.

Clustered spatial patterns of natural inoculum in soil and seasonal variations in stem rot occurrence have hampered efforts to identify resistance to *S. rolfsii* in the field. Because populations of sclerotia are variable and unpredictable, natural inoculum should be supplemented even in fields with a history of disease. The relatively small amounts of fungus added in these tests were sufficient to ensure that some inoculum was present in all plots. Colonized oat grains were applied twice late in the growing season to increase the probability that conditions would be conducive for infection at inoculation; infections originating from colonized oat grains were commonly observed. Timing of inoculation and incorporation of a food base (oat grains) in the inoculum probably caused disease more efficiently than methods used in previous studies, where mycelium or sclerotia were mixed in soil at planting (3,14). Oat grain inoculum required relatively little time or space for preparation.

Production of sclerotia on infected plants and maintenance of *Sclerotium rolfsii* populations in soil do not affect disease within a growing season because spread is limited to plant-to-plant contact. The wide host range and persistence of the fungus in soil (1) increase the importance of end-of-season inoculum densities in resistance breeding, however. Microplot results indicated that sclerotial populations were not restricted in plots planted with NC Ac 18016 or NC Ac 18416, even though these genotypes suppress disease development in the field. Apparent suppression of inoculum production or survival in plots planted with NC 2 should be investigated for incorporation in resistance breeding or management schemes.

Partial resistance (including phenological suppression) to *S. rolfsii* was expressed by the same three genotypes under moderate as well as very high disease pressure in the field and under varying soil moistures (B. B. Shew, *unpublished*), suggesting that disease suppression in these genotypes is stable and could be enhanced by selection in field trials. Time of disease assessment was not critical, but multiple assessments may be useful in predicting yield losses once resistance components have been incorporated into agronomically acceptable genotypes. Promising genotypes should be evaluated in field, microplot, and greenhouse environments to identify and characterize components of resistance.

Of the genotypes we evaluated, NC Ac 18416 was most desirable because it expressed partial resistance in the field and greenhouse. Combining phenological suppression and metabolic resistance is a promising approach to breeding agronomic cultivars for high partial resistance to *S. rolfsii*.

Table 4. Effects of initial inoculum density of *Sclerotium rolfsii* on peanuts grown in microplots in Rocky Mount, NC, in 1985[a]

Number of added sclerotia/plot	Lesion number	Lesion length[b] (cm)	Final number of sclerotia/kg soil[c]
6	13	49	2
25	23	126	3
100	30	93	9
400	25	90	11
$P > F$	<0.01	<0.01	<0.01

[a] Means of four replications of four peanut genotypes planted in 0.9-m-diameter microplots. Each plot contained four plants.
[b] Length of longest lesion in each plot.
[c] Viable sclerotia of *S. rolfsii* recovered from plots in October 1985.

Table 5. Lesion development on four peanut genotypes inoculated with *Sclerotium rolfsii* in the greenhouse

	Untransformed		Transformed[a]	
Genotypes	Lesions (no.)	Length (cm)	Lesions (no.)	Length (cm)
NC Ac 18016	2[b]	34	1.22	4.63
NC Ac 18416	1	26	0.77	3.31
NC 2	2	32	1.23	4.60
Florigiant	1	17	0.57	2.60
LSD ($P = 0.05$)	...	...	0.45	1.75

[a] Number of lesions per plant and mean length of three longest lesions were transformed to square roots. Statistical analysis was performed only on transformed data.
[b] Values represent means of three replications, two watering, and two cheesecloth treatments at soil temperatures of 28 C.

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