# Screening Peanuts for Resistance to Sclerotinia Blight

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**ABSTRACT**

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Sclerotinia blight, caused by *Sclerotinia minor*, results in peanut (*Arachis hypogaea*) crop losses of 4–6% annually in Virginia. *Sclerotinia minor* attacks plant parts that are in contact with the soil, causing lesions on stems and branches as well as pod rot. Three field screening tests were conducted in 1977, 1978, and 1979 to identify resistant peanut genotypes. The genotypes Chico, PI 371521, and VA 71-347 exhibited a significantly lower incidence of Sclerotinia blight symptoms in 1977 field screening than Starr, NC 17, Florigiant, and two breeding lines. Chico, NC 3033, VA 71-347, and VGP 1 exhibited significantly fewer Sclerotinia blight symptoms in 1978 field screening than GK 3, Early Bunch, NC 6, Florigiant, and six breeding lines. Under severe disease pressure, VA 71-347 had the highest value per hectare, being significantly higher than GK 3, Early Bunch, and Florigiant. In 1979, Chico, NC 3033, VA 71-347, and VGP 1 exhibited significantly fewer Sclerotinia blight symptoms than 20 other entries, including Florigiant, Tifrun, NC 7, and Early Bunch. VA 71-347 had a significantly higher yield and value per hectare than all other entries in 1979. The influence of plant canopy structure, morphological and physiologic aspects of resistance, use of multiline peanut cultivars to reduce genetic vulnerability, and multiple pest resistance as related to Sclerotinia blight are discussed.

Additional key words: disease resistance, groundnuts, peanut breeding

Sclerotinia blight of peanuts (*Arachis hypogaea* L.), caused by *Sclerotinia minor* Jagger (9), results in annual crop losses of 4–6% in Virginia (15). Sclerotinia blight, first observed in Virginia in 1971, was widespread by 1974 (13,17). *Sclerotinia minor* attacks plant parts that are in contact with the soil, causing lesions on stems and branches as well as pod rot (13).

Porter et al (14) were the first to screen peanut germ plasm for resistance to *S. minor*. They concluded that Florigiant was the most tolerant cultivar grown of 19 genotypes tested, although 100% infection was observed by harvest. Breeding lines with Spanish and Valencia pedigrees were more resistant to Sclerotinia blight than Florigiant (14).

In other crops, luxuriant plant growth has been shown to enhance the severity of *S. sclerotiorum* infection (5,7,11,18). In beans (*Phaseolus vulgaris*), smaller plant types with open canopies are less susceptible than larger plant types with dense canopies (5,7,18). Newton and Sequeira (11) observed that lettuce (*Lactuca sativa*) plant types with a raised growth habit are the most resistant to *S. minor*.

Plants with open canopies have a warmer, drier microclimate within the plant canopy, as a result of more air circulation and light penetration, than those with dense canopies, which have a cooler, wetter microclimate because of less air circulation and light penetration (5,7,11,18). The former microclimatic conditions are less favorable for colonization and infection by *S. minor* than the latter. This type of resistance has been characterized as a morphological escape mechanism rather than a physiologic phenomenon (2,11). Steadman et al (19) obtained the same effect using wider than normal row spacings in plantings of great northern beans. However, the greatest reduction in disease occurred when both wider row spacings and a cultivar with an open canopy were used. Schwartz et al (18) proposed that the most critical determinant of disease severity in this type of resistance was the distribution of leaf area near the soil surface.

Physiologic resistance to *S. sclerotiorum* has also been suggested in beans (1,2,18). Abawi et al (1) proposed that a single dominant gene conditioned resistance in *Phaseolus coccineus*. Field resistance in lettuce also appears to be heritable (11).

*Sclerotinia* spp. can be controlled with various fungicides (1,3,7,12). Porter and Rud (16) have suppressed Sclerotinia blight in peanuts with the use of dinitrophenol herbicides. Coyne et al (7) reported that open-canopy type cultivars of dry beans used with a fungicide provide better control than dense-canopy type cultivars used with a fungicide.

The observation by Lumsden and Dow (10) that the histologic pathology of *S. sclerotiorum* (which infects dry beans) and that of *S. minor* (which infects lettuce and peanuts) are similar led us to initiate a series of experiments in 1977 to apply to peanuts the results observed in control of *Sclerotinia* spp. with other crops. The objective of this study was to test the effect of various peanut plant canopies on severity of Sclerotinia blight.

## MATERIALS AND METHODS

Ten peanut genotypes were screened in the field in 1977 for reaction to *S. minor* (Table 1). Florigiant was included because Porter et al (14) reported it as the most tolerant cultivar and because it is currently grown on more than 90% of the Virginia peanut allotment. NC 17, VA 70-91, VA 71-347, PI 371521 (minileaf mutant), and PI 362130 (narrow-leaf mutant) were evaluated because of their differing plant canopy types. PI 365553 and PI 371961 were included because of their resistance to other soilborne diseases. Starr and Chico were included because Porter et al (14) suggested that Spanish types might be less susceptible to *S. minor*. Three visual estimates of the percentage of plants per plot showing symptoms of Sclerotinia blight were made at intervals during the growing season.

**Table 1.** Percentage of peanut plants infected with Sclerotinia blight on three observation dates in 1977

| | Infected plants per plot (%) | | |
|---|---|---|---|
| Genotype | 10 Aug. | 22 Aug. | 31 Aug. |
| PI 362130 | 21.3 a[z] | 28.7 b | 50.0 a |
| Florigiant | 22.0 a | 30.7 ab | 45.3 ab |
| PI 371961 | 19.7 ab | 42.0 a | 45.0 ab |
| NC 17 | 22.0 a | 38.0 ab | 37.7 bc |
| Starr | 16.3 ab | 16.3 cd | 36.0 bc |
| VA 70-91 | 15.0 ab | 25.7 bc | 28.0 cd |
| PI 365553 | 12.7 b | 36.0 ab | 28.0 cd |
| VA 71-347 | 3.0 c | 10.7 de | 18.0 de |
| PI 371521 | 2.0 c | 3.3 e | 11.3 ef |
| Chico | 1.0 c | 3.0 e | 5.3 f |

[z] Means within a column followed by the same letter are not significantly different at the 5% level according to Duncan's new multiple range test.

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Mention of a specific commercial peanut cultivar does not constitute endorsement by the USDA.

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In 1978, 20 peanut genotypes including four entries from the 1977 test (Florigiant, Chico, VA 71-347, and PI 371521); nine advanced breeding lines (VA 732813, VA 732815, VA 732816, VA 732817, VA 732818, VA 732827, VA 732829, VA 732832, and VA 732834) from reciprocal crosses between Florigiant and Chico; three new cultivars (NC 6, GK 3, and Early Bunch); two germ plasm lines resistant to Cylindrocladium black rot (CBR) caused by *Cylindrocladium crotalariae* (NC 3033 and VGP 1); and two additional breeding lines (VA 740809 and VA 760513) were screened in the field for reaction to *S. minor* (Table 2). Disease severity was determined by making 20 observations per plot, scoring the disease severity from 1 = no disease to 10 = dead plant, and averaging the 20 observations for each plot. Based on the disease ratings, 13 genotypes were selected for which yield (kg/ha), market grade, and value ($/ha) were determined.

**Table 2.** Sclerotinia blight disease ratings, yields, and values for selected peanut genotypes evaluated in 1978

| Genotype | Disease rating[x] | Yield (kg/ha) | Value ($/ha) |
|---|---|---|---|
| VA 732834 | 6.5 a[y] | ...[z] | ... |
| VA 740809 | 5.9 ab | ... | ... |
| VA 732827 | 5.6 ab | ... | ... |
| VA 732829 | 5.5 ab | ... | ... |
| VA 732832 | 5.4 abc | ... | ... |
| Florigiant | 4.8 bcd | 2,483 c | 1,058 d |
| NC 6 | 4.1 cde | 3,446 a | 1,589 ab |
| Early Bunch | 4.1 cde | 2,768 bc | 1,324 bc |
| VA 760513 | 3.8 def | 2,632 c | 899 de |
| GK 3 | 3.2 efg | 3,147 ab | 1,324 bc |
| VA 732815 | 3.0 efgh | 3,175 ab | 1,310 c |
| VA 732817 | 3.0 efgh | 3,486 a | 1,470 abc |
| VA 732818 | 3.0 efgh | 3,362 a | 1,379 bc |
| VA 732816 | 2.6 fghi | 3,202 ab | 1,334 bc |
| PI 371521 | 2.5 fghi | ... | ... |
| VA 732813 | 2.4 ghij | 3,255 ab | 1,347 bc |
| VGP 1 | 1.6 hij | 2,388 c | 983 de |
| VA 71-347 | 1.6 ij | 3,635 a | 1,668 a |
| NC 3033 | 1.4 ij | 1,939 d | 783 e |
| Chico | 1.1 j | ... | ... |

[x] 1 = no disease and 10 = dead plant.
[y] Means within a column followed by the same letter are not significantly different at the 5% level according to Duncan's new multiple range test.
[z] Not determined.

In 1979, 24 peanut genotypes were screened for reaction to *S. minor* (Table 3). In addition to Florigiant, Early Bunch, VGP 1, NC 3033, VA 71-347, and Chico from the 1978 test, the seven component lines of Florigiant (B, C, E, F, G, H, and I), two new cultivars (Tifrun and NC 7), and nine breeding lines (VA 751011, VA 751012, VA 751013, VA 751014, VA 751021, VA 760503, VA 760504, VA 760505, and VA 760510) were included in the field test. Disease severity was determined using a scale of 1 = no disease to 5 = dead plant. Yield (kg/ha), market grade, and value ($/ha) were determined for all entries except Chico.

A randomized complete block design with four replicates was used in all field experiments except those in 1979, when three replicates were used. Tests were conducted in peanut fields having a history of severe Sclerotinia blight. Standard production practices for fertilizer, herbicide, fungicide, and insecticide application were used in all tests. No chemicals were applied for control of Sclerotinia blight. All plots were 1.8 × 6.1 m using standard 91-cm rows and received 107,000 seeds per hectare. Data were analyzed by analysis of variance and Duncan's new multiple range test.

**Table 3.** Sclerotinia blight disease ratings, yields, and values for peanut genotypes evaluated in 1979

| Genotype | Disease rating[x] | Yield (kg/ha) | Value ($/ha) |
|---|---|---|---|
| Florigiant | 3.7 a[y] | 1,953 cdef | 892 defg |
| Florigiant-B | 3.6 a | 2,143 bcde | 1,053 bcde |
| Florigiant-C | 3.2 a | 2,048 bcdef | 927 bcdefg |
| Florigiant-E | 3.5 a | 2,184 bcde | 1,043 bcde |
| Florigiant-F | 3.3 a | 1,845 cdef | 838 efg |
| Florigiant-G | 3.3 a | 1,953 cdef | 934 bcdefg |
| Florigiant-H | 3.3 a | 2,116 bcdef | 1,001 bcdef |
| Florigiant-I | 3.3 a | 1,939 cdef | 904 bcdefg |
| VA 751011 | 3.6 a | 1,953 cdef | 914 bcdefg |
| VA 751012 | 3.5 a | 2,211 bcde | 1,008 bcdef |
| VA 751013 | 4.0 a | 1,519 f | 657 g |
| VA 751014 | 3.0 a | 2,591 b | 1,201 bc |
| VA 751021 | 3.1 a | 1,939 cdef | 914 bcdefg |
| VA 760503 | 3.0 a | 1,966 cdef | 895 cdefg |
| VA 760504 | 3.4 a | 2,075 bcdef | 988 bcdef |
| VA 760505 | 3.2 a | 2,346 bc | 1,139 bcde |
| VA 760510 | 3.4 a | 2,157 bcde | 1,023 bcde |
| VGP 1 | 1.7 b | 1,736 def | 712 fg |
| VA 71-347 | 1.8 b | 3,133 a | 1,475 a |
| Tifrun | 3.5 a | 2,279 bcd | 983 bcdef |
| NC 3033 | 1.9 b | 2,252 bcd | 969 bcdef |
| NC 7 | 3.4 a | 2,360 bc | 1,184 bcd |
| Early Bunch | 3.2 a | 2,388 bc | 1,203 b |
| Chico | 1.2 b | 1,614 ef | ...[z] |

[x] 1 = no disease and 5 = dead plant.
[y] Means within a column followed by the same letter are not significantly different at the 5% level according to Duncan's new multiple range test.
[z] Not determined.

## RESULTS

In 1977, three genotypes (VA 71-347, PI 371521, and Chico) had significantly ($P = 0.05$) less Sclerotinia blight than five other genotypes tested, including Florigiant and NC 17, two commercial cultivars (Table 1). Sclerotinia blight generally increased in severity with time for all genotypes.

In 1978, 11 genotypes had significantly ($P = 0.05$) less Sclerotinia blight than Florigiant. Four of these genotypes (Chico, NC 3033, VA 71-347, and VGP 1) had significantly ($P = 0.05$) less Sclerotinia blight than GK 3, the least infected commercial cultivar (Table 2). Five of these genotypes (VA 732813, VA 732815, VA 732816, VA 732817, and VA 732818) were from the cross Chico (female) × Florigiant (male) and had significantly ($P = 0.05$) less Sclerotinia blight than four genotypes (VA 732827, VA 732829, VA 732832, and VA 732834) from the reciprocal cross, as well as Florigiant. VA 71-347 had significantly ($P = 0.05$) higher yield and value per hectare than the other genotypes evaluated, except NC 6 and VA 732817.

In 1979, four lines identified as resistant in 1978 (NC 3033, VA 71-347, VGP 1, and Chico) had significantly ($P = 0.05$) less Sclerotinia blight than all other genotypes, and no significant differences existed among the other 20 genotypes (Table 3). Florigiant did not differ significantly for disease rating, yield, and value per hectare from the seven sister lines that are composited to form the commercial cultivar. VA 71-347 had a significantly ($P = 0.05$) higher yield and value per hectare than the other genotypes tested (Table 3).

## DISCUSSION

The three resistant genotypes (Chico, VA 71-347, and PI 371521) identified in 1977 (Table 1) have different plant canopy structures than the dense, spreading-type canopy of most current Virginia-type cultivars. Chico (Spanish type) has a small upright growth habit. VA 71-347 (large-seeded Virginia type) has an open, upright type of canopy that closely resembles the ideotype proposed by Coyne et al (7) for resistant dry bean cultivars. PI 371521 (small-seeded Virginia type) is a semi-upright, minileaf mutant, having marked reduction in leaflet size compared with other genotypes. Based on results in other crops (5,7,11,18), the resistance in these genotypes is thought to be a morphological escape mechanism rather than physiologic resistance.

The significant differences between the advanced breeding lines from reciprocal crosses between Chico and Florigiant in 1978 (Table 2) strongly suggest that a cytoplasmic factor may be involved in determining at least partial resistance to Sclerotinia blight. This also leads to the conclusion that the resistance of Chico is at least partially physiologic in nature and not caused solely by the small upright growth habit, because all breeding lines from the reciprocal crosses are similar to Florigiant in growth habit. The 78% reduction in Sclerotinia blight for Chico compared with Florigiant (Tables 1–3) is probably the result of a combination of physiologic and morphological factors. The cytoplasmic factor involved may have a pleiotropic effect because reciprocal differences were also noted between these lines for their reaction to Cercospora leaf spot (Coffelt and Porter, *unpublished*). Additional work is being conducted to answer these questions.

The 59% reduction in Sclerotinia blight for VA 71-347 compared with Florigiant (Tables 1–3) may also be partially physiologic. In a mist chamber test, when Florigiant and VA 71-347 were artificially inoculated with 39 different isolates of *S. minor*, the average lesion length on infected branches of VA 71-347 was 45.5 mm compared with 68.0 mm on infected branches of Florigiant (Porter and Coffelt, *unpublished*). Schwartz et al (18) reported similar lesion lengths on resistant (10–40 mm) and susceptible (30–100 mm) dry bean genotypes in a growth chamber test. The reaction of the two genotypes to the 39 isolates was variable. This indicates that races or strains of *S. minor* may develop or already be present. This could complicate field screening for resistance as well as greenhouse screening.

Another important factor to consider in greenhouse screening for morphological resistance is that Schwartz et al (18) reported that resistant cultivars can limit fungal development during the day and that the fungus may be unable to overcome this limitation during the night when conditions become favorable for infection. Therefore, greenhouse screening to select resistant lines should utilize environmental conditions comparable to field conditions.

Further, upright growth habit per se may not lead to resistance. Several bunch genotypes, such as NC 17, Starr, VA 70-91, Early Bunch, VA 751021, VA 760503, VA 760504, VA 760505, and VA 760510, were as susceptible as Florigiant (Tables 1–3). This is in agreement with the work by Coyne et al (7) and Schwartz et al (18) in dry beans. The distribution of leaf area near the soil surface, plant canopy structure, and plant canopy density associated with the growth habit all interact to determine whether the microclimate created within the canopy is more or less favorable for colonization and infection (7,18).

Multiple pest resistance may also be present in some peanut genotypes. NC 3033 and VGP 1, which were released as being resistant to CBR (4,6), have shown high levels of field resistance to *S. minor* (Tables 2 and 3). This is important for peanut breeders because both diseases can limit peanut production. More work is needed to determine whether the Sclerotinia blight resistance of NC 3033 and VGP 1 is morphological or physiologic.

Results from 1978 (Table 2) and 1979 (Table 3) indicate that there were no significant differences among the sister lines from the cross Chico × Florigiant, the sister lines from the reciprocal cross, the component lines of Florigiant, or between the composite Florigiant and the component lines for resistance to Sclerotinia blight. The theory that a composite, such as Florigiant, offers a wider genetic base lowering genetic vulnerability and thus providing more resistance to a specific disease than a single line (8) apparently was not valid for the sister lines studied from these three crosses for field reaction to Sclerotinia blight. This is in contrast to results obtained in screening for resistance to CBR, in which sister lines from the Florigiant × Chico cross and two other crosses differed significantly in susceptibility (Coffelt and Garren, *unpublished*). However, the results agree with those found for the sister lines from the Chico × Florigiant cross, which did not differ significantly for reaction to CBR. These results point out the importance of knowing the reaction of sister lines to several pathogens if the objective of blending sister lines together to form a cultivar is to broaden the genetic base for resistance to specific pathogens. Blending sister lines per se does not guarantee a broader genetic base to potential pathogens.

The similarity between the results obtained in our studies with peanuts and those reported for dry beans (1,5,7,18) supports the findings of Lumsden and Dow (10) that the histopathology of *S. minor* and that of *S. sclerotiorum* are strikingly similar. Thus, it appears that methods of control developed for one crop should be able to be adapted for use in control of *Sclerotinia* spp. on the other crop with a high degree of success assured.

In summary, we found morphological and physiologic resistance to Sclerotinia blight to be present in peanuts. The physiologic resistance appeared to be at least partially controlled by a cytoplasmic factor, although this requires further investigation. In addition to providing a less desirable microclimate for Sclerotinia blight development, plants with morphological resistance should provide a canopy type that allows for more effective control by fungicides. Chico, VGP 1, and NC 3033 should serve as valuable sources of resistance in breeding for resistance to Sclerotinia blight, whereas VA 71-347 offers possibilities for use as a resistant cultivar.

## LITERATURE CITED


1. Abawi, G. S., Provvidenti, R., Crosier, D. C., and Hunter, J. E. 1978. Inheritance of resistance to white mold disease in *Phaseolus coccineus*. J. Hered. 69:200-202.
2. Anderson, F. N., Steadman, J. R., Coyne, D. P., and Schwartz, H. G. 1974. Tolerance to white mold in *Phaseolus vulgaris* dry edible bean types. Plant Dis. Rep. 58:782-784.
3. Beute, M. K., Porter, D. M., and Hadley, B. A. 1975. Sclerotinia blight of peanut in North Carolina and Virginia and its chemical control. Plant Dis. Rep. 59:697-701.
4. Beute, M. K., Wynne, J. C., and Emery, D. A. 1976. Registration of NC 3033 peanut germplasm. Crop Sci. 16:887.
5. Blad, B. L., Steadman, J. R., and Weiss, A. 1978. Canopy structure and irrigation influence white mold disease and microclimate of dry edible beans. Phytopathology 68:1431-1437.
6. Coffelt, T. A. 1980. Registration of VGP 1 peanut germplasm. Crop Sci. 20:419.
7. Coyne, D. P., Steadman, J. R., and Anderson, F. N. 1974. Effect of modified plant architecture of great northern dry bean varieties (*Phaseolus vulgaris*) on white mold severity, and components of yield. Plant Dis. Rep. 58:379-382.
8. Hammons, R. O. 1974. Genetic vulnerability in peanuts: A second look. Proc. Am. Peanut Res. Educ. Assoc. 6:17-20.
9. Kohn, L. M. 1979. A monographic revision of the genus *Sclerotinia*. Mycotaxon. 9:365-444.
10. Lumsden, R. O., and Dow, R. L. 1973. Histopathology of *Sclerotinia sclerotiorum* infection of bean. Phytopathology 63:708-715.
11. Newton, H. C., and Sequeira, L. 1972. Possible sources of resistance in lettuce to *Sclerotinia sclerotiorum*. Plant Dis. Rep. 56:875-878.
12. Porter, D. M. 1980. Control of Sclerotinia blight of peanuts with procymidone. Plant Dis. 64:865-867.
13. Porter, D. M., and Beute, M. K. 1974. Sclerotinia blight of peanuts. Phytopathology 64:263-264.
14. Porter, D. M., Beute, M. K., and Wynne, J. C. 1975. Resistance of peanut germplasm to *Sclerotinia sclerotiorum*. Peanut Sci. 2:78-80.
15. Porter, D. M., Powell, N. L., and Cobb, P. R. 1977. Severity of Sclerotinia blight of peanuts as detected by infrared aerial photography. Peanut Sci. 4:75-77.
16. Porter, D. M., and Rud, O. E. 1980. Suppression of Sclerotinia blight of peanuts with dinitrophenol herbicides. Phytopathology 70:720-722.
17. Powell, N. L., Porter, D. M., and Pettry, D. E. 1976. Use of aerial photography to detect diseases in peanut fields. I. Sclerotinia blight. Peanut Sci. 3:21-24.
18. Schwartz, H. F., Steadman, J. R., and Coyne, D. P. 1978. Influence of *Phaseolus vulgaris* blossoming characteristics and canopy structure upon reaction to *Sclerotinia sclerotiorum*. Phytopathology 68:465-470.
19. Steadman, J. R., Coyne, D. P., and Cook, G. E. 1973. Reduction of severity of white mold disease on great northern beans by wider row spacing and determinate plant growth habit. Plant Dis. Rep. 57:1070-1071.