

Inheritance of Resistance to *Sclerotinia minor* in Selected Spanish Peanut Crosses¹

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ABSTRACT

TxAG-5, a sclerotinia resistant spanish germplasm line released jointly by the Texas Agricultural Experiment Station, USDA, and the Oklahoma Agricultural Experiment Station, was crossed in reciprocal to two spanish lines, Tx851856 and Sn73-30. Parent, F₁, F₂, BC₁F₁, and F₃ populations were evaluated under high natural inoculum for resistance to *Sclerotinia minor* using a disease rating scale of 1 (no disease) to 5 (severely diseased), and the number of days from first appearance of the fungus until plant death was recorded. F_{2,3} families were compared for disease the following year for genotypic assessment of the F₂ parents. F₁ generation plants of the Sn73-30 cross were susceptible, but F₁ plants from Tx851856 were intermediate. Some TxAG-5 succumbed to the disease. F₂ distributions were continuous. F₂ genotypic frequency distributions based on F₃ and BC₁F₃ families were near continuous. Broad-sense heritability estimates for disease ratings for TxAG5/Tx851856 and TxAG-5/Sn73-30 were 14 and 23%, respectively. Narrow-sense heritabilities based on parent offspring regression of

F₃ families on F₂ plants were 11% for Tx851856/TxAG-5 and 1% for Sn73-30/TxAG-5. Selection for resistance among the F₂ plants to increase the frequency of resistant F₃ families would have been ineffective.

Key Words: *Arachis hypogaea* L., sclerotinia blight, heritability, soilborne pathogen, groundnut

Peanut (*Arachis hypogaea* L.) wilt caused by a *Sclerotinia* sp. has been reported in several peanut producing countries, including Argentina, China, Australia, and Taiwan (10). Porter and Beute (11) identified *S. sclerotiorum* (Lib) de Bary on peanuts in the U.S.A. in Virginia in 1971. Sclerotinia blight, caused by *Sclerotinia minor* Jagger (7), is now a major disease of peanut in Virginia, Oklahoma, Texas, and North Carolina and has been reported in Louisiana (17). An estimated 11% of the total peanut production of North Carolina, Oklahoma, Texas, and Virginia was lost to sclerotinia blight in 1982 (16).

Fungicide control has been of limited effectiveness. The only product fully labelled for control of sclerotinia blight in peanut is iprodione. In Texas, botran (2, 6 dichloro-4-nitroaniline) appears to offer more control of sclerotinia blight than iprodione (8). Porter (9) suggested that fungicides used to control other pathogens might enhance sclerotinia blight. When captafol and chlorothalonil

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were applied at recommended rates for cercospora leafspot control, sclerotinia blight severity increased over that of the untreated control.

Partial resistance to *S. minor* has been reported by several researchers (1, 2, 4, 12). Two virginia-type cultivars have been released on the basis of useful resistance to *S. minor* (5), and the spanish cultivars Toalson and Tamsan 90 have good resistance (1, 14). Useful resistance among runner-type cultivars has not been reported.

Akem (1) evaluated several peanut breeding lines for sclerotinia blight reaction in 1985 and 1986 including entries from the Texas pod rot resistance program. Among the resistant lines was TxAG-5 (15), which had an average maximum disease incidence of 16.3% compared to 98% for Florunner. The purpose for this study was to gain information on the inheritance of the resistance of TxAG-5 to *S. minor* that might be used in breeding for resistance.

Materials and Methods

TxAG-5 (15) was crossed to Tx851856 and Sn73-30, two spanish-type lines. Tx851856 is a very early maturing, small-leaved, bunch peanut obtained in Africa from a reportedly Asian source. Sn73-30 (3), from Senegal, West Africa, is a spanish-type cultivar typical for morphological characteristics, but has fresh seed dormancy.

Field Evaluation of Parent, F₁, F₂, and BC₁F₁ Generations

TxAG-5 was crossed in reciprocal to Tx851856 and Sn73-30 to produce F₁, F₂, and BC₁F₁ progeny. Hybrid seeds were harvested on 31 May, 1988 and selfed seed were harvested on 9 June. The seed were imbibed, treated with ethylene gas to break fresh seed dormancy and planted in a *S. minor* infested field near Stephenville, TX on 24 June in a completely randomized experimental design with two replications. Recommended production practices, except for disease control and irrigation schedule, were followed for each test. Fields were irrigated frequently to keep the soil surface at humidity levels conducive for *S. minor* development. Percent emergence was recorded one week after planting. At the first appearance of mycelia on the soil surface, data collection was initiated as follows: date of first visible contact of the fungus with the plant, date of wilting and death due to *S. minor*, a disease rating, and pod yield in grams per plant. Disease symptoms were recorded three times a week beginning 31 August until 7 October, at which date the seeds borne on branches which had been damaged by *S. minor* were beginning to sprout. To save as much seed as possible, the tap roots were severed and the plant harvested individually by hand on 7, 8, and 10 October. Plants were forced air dried at 33C, rated for disease injury, and the pods were removed by hand and weighed. Disease ratings were made based on the number of branches wilted or necrotic because of *S. minor* attack on a scale as follows:

RATING	SYMPTOMS
1	No symptoms
2	Wilting or necrosis of 1 lateral branch
3	Wilting or necrosis of 2 or more lateral branches
4	Wilting or necrosis of mainstem; 2 or more lateral branches living
5	Wilting or necrosis of mainstem; no more than one lateral branch living

Duncan's multiple range test was used to separate means for all variables. The Chi-square test was used to evaluate segregation ratios.

F_{2,3} and BC₁F₁ Generation Test - 1988

Genotypic evaluations were made using F₂ derived F₃ families from crosses of TxAG-5 to Tx851856 and Sn73-30. A completely randomized experimental design was used. Twenty-one F_{2,3} and BC₁F_{2,3} seed were planted 23 cm apart in single row plots 4.6 m long adjacent to the previously discussed test. Data recorded included first wilting due to *S. minor* on a plant basis and a disease rating. Beginning 31 August, the number of plants wilted due to *S. minor* was counted three times a week until 10 October. On 10 October and 9 November all 4247 plants were rated using the previously described disease rating system.

The number of plants wilted each day was used to calculate an area under the disease progress curve (AUDPC) for each F_{2,3} and BC₁F_{2,3} plant row as follows:

$$(DAP2-DAP1) \times [Rating\ 1 + (Rating\ 2 - Rating\ 1) / 2]$$

where DAP = Days after planting

Rating = the number of plants wilted

A similar procedure was used for the successive ratings, and the values were summed for each F₂ row. Thus, AUDPC was a cumulative number for each plant-row resulting from comparisons among rating dates. The F₂ plants were classified genotypically for AUDPC on the basis of the fit of their F_{2,3} or BC₁F_{2,3} AUDPC values. F₂ plants were arbitrarily designated as resistant, susceptible, and segregating using parental AUDPC values as guides. The frequency of plant rows within each category was estimated and the goodness of fit tested by Chi-square.

F_{2,3} Generation Test - 1989

Seed from resistant and susceptible F₂ plants that yielded 20 g or more in 1988 were planted in the same *S. minor* infested field near Stephenville in 1989. Plants with a disease rating of 1 or 2 were defined as resistant and those with a disease rating greater than two were defined as susceptible. Twenty-one seed from each F₂ plant were spaced 15.2 cm apart in single-row plots 3.04 m long using a randomized experimental design with two replications. Data collected included a disease rating based on the number of plants wilted and necrotic due to *S. minor* on a scale as follows: 1=no disease, 10=all plants within a plot dead.

Heritability Estimation

Estimates of broadsense heritability (H) were calculated for the variables DTOW and DRATE using the formula cited by Fehr (6). Heritability was calculated using the susceptible parent and F₁ progeny to estimate environmental variance because of a large variation in DTOW for TxAG-5. Heritability in the narrow sense was calculated by parent-offspring regression of F₂ plants grown in 1988 and F_{2,3} families grown in 1989. Adjustments were made in the estimation of h² as described by Smith and Kinman (13).

Results and Discussion

Emergence was good in 1988 (93% average,) and plant development was normal. Germinated sclerotia were first observed on the soil surface on 31 August. *Sclerotinia minor* was abundant and decisively the predominant pathogen in the field, although a trace of other plant pathogens were present. A few plants were killed by *Aspergillus niger* van Tiegh and were deleted from the analysis. Climatological data and first appearance of mycelia were considered in the choice of 29 August as a reference date for assumed initiation of fungal activity, and this date was used in data summarization to compare plant response to the fungus. The number of days after 29 August until mycelia were seen in contact with the plant (DTOM) was not different among entries and generations (Table 1). The inoculum load was heavy and well

Table 1. Number of plants tested, and average number of days until *S. minor* was in contact with plants (DTOM), number of days until plants wilted (DTOW), disease rating, and pod weight for Tx851856, TxAG-5, and progenies.

	Number of Plants	DTOM*	DTOW*	Disease Rating**	Pods g/plant
Parents					
Tx851856	20	5.8 a*	13.5 ab	3.1 ab	39 c
TxAG-5	23	4.4 a	20.0 a	3.4 ab	48 bc
Susceptible Check					
Starr	21	3.6 a	11.1 b	3.6 b	36 c
F₁					
Tx851856/TxAG-5	34	4.4 a	16.3 ab	3.3 ab	61 a
TxAG-5/Tx851856	25	3.6 a	15.6 ab	3.5 b	47 bc
F₂					
Tx851856/TxAG-5	106	5.2 a	15.5 ab	3.1 ab	42 bc
BC₁F₁					
Tx851856/Tx851856/TxAG-5	20	4.7 a	18.4 a	2.8 a	53 ab
TxAG-5//Tx851856/TxAG-5	25	5.5 a	18.7 a	3.3 ab	54 ab

* Means within columns followed by the same letter are not different, P=0.05 Duncan's Multiple Range Test.

+ Number of days after 29 August, 1988.

- 1=no disease symptom; 5=severe injury.

distributed as determined by the DTOM data and the sclerotia count in soil samples; approximately 19 per 100 g of soil. It is probable that plants that were not visibly in contact with the fungus were challenged by subsurface or hidden fungal propagules.

Wilting was apparent in Starr, a known susceptible cultivar, earlier than in TxAG-5 and occurred 6.5 days earlier in Tx851856 than in TxAG-5, but that difference was not significant ($P=0.05$) (Table 1). The mean number of days to wilt (DTOW) for the F_1 and F_2 generations were near the mid-parent, and the DTOW for both backcross populations were between the F_1 and TxAG-5.

The Tx851856/TxAG-5 F_1 mean pod yield (61.3 g per plant) was higher than both parents. The pod weight of the reciprocal F_1 was between the parents as was the mean pod weight of the F_2 . The average pod weight of both backcross generations was larger than the parental lines.

Differences in the generation mean disease ratings were small for the TxAG-5/Tx851856 population. The disease rating (DRATE) for the BC_1F_1 generation was lower than for the F_1 , but not different from the other entries. The DRATE of the parent F_1 and F_2 populations were approximately equal.

Tx851856 and TxAG-5 reacted similarly, with low disease ratings, slow plant death, and high average pod yields per plant. Low disease ratings, a large number of days before wilting, and pod yields greater than both parents were recorded for the progeny of TxAG-5 and Tx851856. The F_1 progeny reactions were similar to their parents with higher yields. Variability for days to wilt (DTOW) was greater for the F_2 progeny than for Tx851856 and TxAG-5. This suggests that Tx851856 and TxAG-5 have components of resistance that are different from each other, for if the mode of resistance is the same, the distribution of values for DTOW of the F_2 progeny would have followed the distributions in the parents.

In crosses involving Sn73-30 and TxAG-5, the average DTOW of TxAG-5 was almost twice that for Sn73-30 (Table 2). The DTOW for the F_1 's were similar to Sn73-30, while

Table 2. Number of plants tested, and average number of days until *S. minor* was in contact with plants (DTOM), number of days until plants wilted (DTOW), disease rating, and pod weight for Sn73-30, TxAG-5 and progenies.

	Number of Plants	DTOM*	DTOW [†]	Disease Rating [~]	Pods g/plant
Parents					
SN73-30	18	6.5 a*	11.4 c	3.8 ab	11 e
TxAG-5	24	4.7 ab	21.4 a	3.1 a	50 ab
Susceptible Check					
Starr	22	4.1 ab	13.8 bc	3.8 ab	36 cd
F_1					
Sn73-30/TxAG-5	36	3.7 b	10.1 c	4.1 b	45 bc
TxAG-5/Sn73-30	35	5.2 ab	14.3 bc	3.8 ab	59 a
F_2					
Sn73-30/TxAG-5	150	5.0 ab	15.8 abc	3.7 ab	31 d
BC_1F_1					
Sn73-30//TxAG-5/Sn73-30	11	5.4 ab	9.8 c	4.1 b	32 d
TxAG-5//TxAG-5/Sn73-30	19	4.5 ab	18.3 ab	3.7 ab	56 ab

* Means within columns followed by the same letter are not different, $P=0.05$ Duncan's Multiple Range Test.

+ Number of days after 29 August, 1988.

~ 1=no disease symptom; 5=severe injury.

the F_2 mean was near the mid-parent. There was a difference ($P=0.05$) in the DTOW for the two backcross populations.

Sn73-30 produced the lowest pod yield, 11.5 g per plant, which was significantly ($p=0.05$) lower than TxAG-5. The mean pod yield of the F_1 , F_2 and backcross generations were intermediate or equal to the average of the best parent. Sn73-30 reacted very similar to Starr, a known susceptible cultivar, with a high DRATE, a low DTOW value, and the lowest average pod yield per plant.

$F_{2,3}$ and $BC_1F_{2,3}$ Generations

The lowest (213.8) and the highest (522.7) AUDPC values were for TxAG-5 and Sn73-30, respectively (Table 3). The AUDPC of Starr was intermediate but closer to Sn73-30. The mean AUDPC values for the $F_{2,3}$ populations were not significantly ($P=0.05$) different from their parents. The values for $BC_1F_{2,3}$ generations were intermediate between Sn73-30 and TxAG-5, and not different from TxAG-5. The distributions of these two populations were continuous (Fig. 1 & 2).

Table 3. Areas under disease progress curves (AUDPC) for Parents, susceptible check, $F_{2,3}$ and $BC_1F_{2,3}$ populations.

	Number of Rows ⁺	Mean
Parents		
Tx851856	4	258 a*
Sn73-30	5	523 c
TxAG-5	8	214 a
Susceptible Check		
Starr	8	418 b
$F_{2,3}$		
Tx851856/TxAG-5	60	290 a
TxAG-5/Tx851856	60	263 a
$BC_1F_{2,3}$		
TxAG-5//Sn73-30/TxAG-5	40	293 a
TxAG-5//TxAG-5/Sn73-30	40	288 a

* Means within columns followed by the same letter are not different, $P=0.05$ Duncan's Multiple Range Test.

+ Number of plant rows evaluated.

The AUDPC values for the $F_{2,3}$ families ranged from less than 50 to near 600. AUDPC values of 250 or less were considered representative of non-segregating plant rows on the basis of the AUDPC value of TxAG-5. Sn 73-30 was considered susceptible because of the large average AUDPC value, larger than that of Starr. Plant rows with AUDPC values of 401 or greater were considered non-segregating and susceptible. Plant rows with values between 251 and 400 were considered segregating. No meaningful ratios of $F_{2,3}$ plant rows were found using the AUDPC values, and no objective basis for definition of classes was found for testing with mono- or digenic F_2 genotypic models. Trigenic models were not tested because population sizes were too small. The AUDPC value of Tx851856 was within the intermediate category.

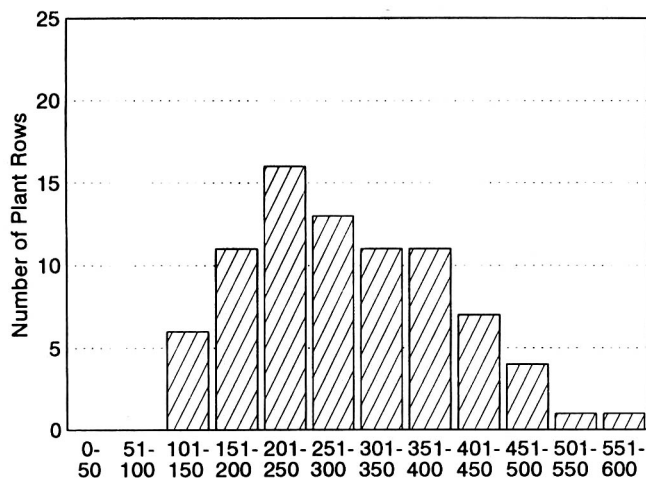


Fig. 1. Frequency distribution of area under disease progress curve (AUDPC) values for Tx851856/TxAG-5 and TxAG-5/Tx851856 F_{2,3} plant rows.

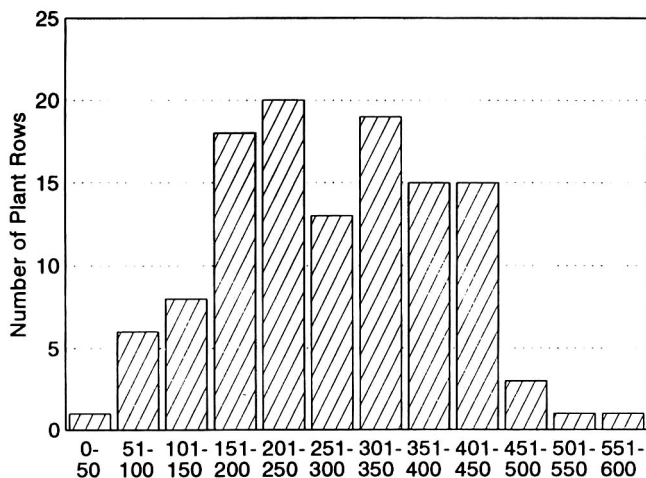


Fig. 2. Frequency distribution of area under disease progress curve (AUDPC) values for TxAG-5/Sn73-30/TxAg-5 and TxAG-5/TxAg-5/Sn73-30 BC₁F_{2,3} plant rows.

Heritability

Disease symptoms on some TxAG-5 plants occurred rather rapidly while other plants withstood the disease for the duration of the study; hence, this parent was omitted in heritability calculations. Broadsense heritability estimates for DTOW were 41.5% and 50.3% for the Tx851856/TxAG-5 and Sn73-30/TxAG-5 crosses, respectively. Heritability estimates for disease rating were 14.0% for the Tx851856/TxAG-5 cross and 23.0% for the Sn73-30/TxAG-5 cross. Narrow-sense heritabilities for disease rating were calculated to be 11% for Tx851856/TxAG-5 and 1% for Sn73-30/TxAG-5.

Estimates of the heritability of the variables for the two crosses were intermediate to low. The estimates for both traits, DTOW and DRATE, were lower for the cross (Tx851856/TxAG-5 and its reciprocal than for Sn73-30/TxAG-5 and its reciprocal. Low heritability estimates suggest either the involvement of several genes for a trait or large environmental variance.

The variable DTOW appeared to be an objective measure of vascular blockage related to infection. Since all plants were labelled at first appearance of wilt, data collection was

rapid and easy. A presumption with this variable is that the rate of growth into and blockage or destruction of the vascular system is an accurate reflection of biological resistance to the fungus. The DTOW means of the parents (Tx851856, Sn73-30, and TxAG-5) were different and seemed coincident with this assumption. However, the assumption might be an over-simplification of the disease development process which resulted in variability that hindered the discrimination of reactions.

Differences among entries were obvious for pod weights but chi-squares were not calculated because of continuous F₂ distributions and the multigenic nature of yield. Differences were expected and seen within the Sn73-30 and TxAG-5 populations. The F₁ and BC₁F₁ reciprocal populations of Sn73-30 and TxAG-5 were different from each other. Pod yields were highest when TxAG-5 was the maternal parent. A yield difference was found, also, between reciprocal Tx851856 and TxAG-5 F₁ progeny, with the highest yield occurring when Tx851856 was maternal parent. This might suggest a maternal or cytoplasmic effect but such was not apparent in the backcross populations.

Few differences among entries occurred for DRATE. The F₂ genotypic distributions were continuous and distributions of the parent plant values prohibited a delineation of classes within the F₂ populations. The DRATE variable might have been more powerful if the disease rating had been taken on fresh plants in the field instead of dried plants.

The AUDPC values suggested Sn73-30 and Starr differ in susceptibility. The other variables measured, except pod weight, did not provide such a clear difference between the susceptible entries. The mean AUDPC of the plant rows distinguished between the susceptible and resistant entries, but not among possible resistant entries. Differences among TxAG-5 and Tx851856 and progenies may have been easier to delineate if more parent rows could have been assayed.

The continuous F₂ distributions for the different variables might have resulted because: a) the level of inoculum might have been so high that partial resistance was overcome or masked, b) the parents might not have been totally homogeneous for factors affecting disease reaction, or c) inheritance of resistance to sclerotinia blight might be controlled by several genes. Additional research on parents and progeny will be required to make that definition. However, resistance of TxAG-5 to sclerotinia blight, affirmed in this study, is heritable and probably controlled multigenically. Utilization of this germplasm line in hybridization and backcrossing followed by family selection should be a viable approach for transferring the resistance to useful breeding lines and cultivars.

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