EPIDEMIOLOGY AND DISEASE RESISTANCE
OF SCLEROTINIA BLIGHT
IN PEANUT

By

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PREFACE

Detailed and quantitative epidemiological data are essential for the development of effective and economical control programs for diseases caused by *Sclerotinia* species. Data presently available on the epidemiology of white mold of beans and drop of lettuce have contributed significantly to the control of these important diseases. Such data is not complete on Sclerotinia blight of peanut, and will be necessary to develop intergrated disease control strategies. Identifying sources of resistance to *Sclerotinia minor* in peanut germplasm remains one of the most desired means of control of Sclerotinia blight.

In this study, several peanut genotypes were evaluated for reistance to *S. minor* in both the greenhouse and in field plots. Epidemiological parameters for resistance such as sclerotial production and viability, as well as pod yield among different genotypes were evaluated. Seed transmission as a possible means of disease dissemination through contaminated seed lot was also investigated. The infection process of *S. minor* on peanut in the early stages of disease development was studied using a scanning electron microscope, in an attempt to understand more about the host-pathogen interaction.

I am deeply indebted to my major advisor, Dr.

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CHAPTER I

INTRODUCTION

Diseases caused by *Sclerotinia* species in economically important plants occur worldwide and result in considerable damage. Typically they have been unpredictable and difficult to control culturally or chemically. Host resistance to the diseases has been inadequate.

Sclerotinia blight of peanut (*Arachis hypogaea* L.) caused by the soilborne fungus *Sclerotinia minor*, was first observed on peanut plants in Argentina in 1922. It is now present in most peanut-producing countries of the world. It was first observed in the United States in Virginia in 1971. Thereafter it spread to North Carolina, Oklahoma, and Texas, and by 1982, Sclerotinia blight was considered the most important disease of peanut in Virginia and Oklahoma. The disease is serious because of yield reduction, lack of established consistent and economical control practices, problems of identification, and difficulties to detect in early stages of development. Yield losses of 10% are common and in some blighted spots of a severely infected field, as much as 50% of expected pod yield may be left in the soil after harvest. Fungicides have provided only limited control. Other methods of control that have met with varying

degrees of success include cultural practices, and the use of biological control agents.

The use of resistant crop varieties as a disease control means is preferred whenever possible. Very few crops have resistant varieties that can withstand all pests to which they are exposed. Very often what is initially identified as resistance turns out to be tolerance or just an escape means that does not stand under severe disease pressure. The search for resistance to *Sclerotinia minor* in peanut germplasm has been intensified in recent years, and seems like the best approach to solve the problem. So far none of the findings have been overwhelming. Most have made conclusions based mainly on field evaluations and none has included disease progress or the role of inoculum produced as parameters for evaluation of resistance. At present there is no peanut cultivar available to growers that is highly resistant to *S. minor*.

Quick and reliable techniques to evaluate peanut genotypes in the laboratory or greenhouse before extensive field screening are lacking. No detailed studies have been made on the infection process of the pathogen, and the exact role of the host plant in infection and disease development is only speculative. Besides known cultural methods of spread of inoculum, the possible role of seed transmission in disease spread is only suspected.

Evaluating several peanut genotypes for resistance to

S. minor in both greenhouse and field conditions, epidemiological aspects of the disease including possibility of seed transmission and the infection process of the pathogen on peanut stems were major areas of study of this research.

This dissertation is presented in an alternative format that deviates from the traditional organization outlined in the Thesis Writing Manual provided by the Graduate College of Oklahoma State University. The dissertation is composed of four manuscripts (Chapters) written according to the format style established by the American Phytopathological Society (APS) for manuscripts submitted to the professional journals of PHYTOPATHOLOGY or PLANT DISEASE. The final Chapter (Appendix) includes material investigated in the research but not presented in the manuscripts or presented in a summarized form.

Chapter II entitled "A Detached Shoot Technique to Evaluate the Reaction of Peanut Genotypes to *Sclerotinia minor*", describes a technique developed to rapidly assess the reaction of peanut genotypes to *S. minor* under laboratory conditions. This technique is useful for all initial screening of peanut lines before extensive field evaluations. This manuscript will be submitted to either PEANUT SCIENCE or PLANT DISEASE. Chapter III entitled "Evaluation of Peanut Genotypes for Resistance to Sclerotinia Blight in Field Plots", examines the disease progress of Sclerotinia blight

in field plots infested with *Sclerotinia minor*. Based on interval disease incidence readings, and calculated disease progress values, several peanut genotypes are separated into resistant and susceptible categories. Chapter IV entitled "Sclerotial Production and Viability on Peanut Genotypes Planted in *Sclerotinia minor*-infested Field Plots", examines the role of inoculum produced by different genotypes on stems and pods as a criteria for measuring degree of resistance. Viability of the sclerotia produced is also compared among genotypes since it is important for inoculum produced to be viable in order to be effective in causing disease. The effect of the disease on pod yield among genotypes is also determined in this chapter. As with disease progress measurements, this data is used to further separate genotypes into resistant and susceptible categories. Chapter V entitled "Transmission of Sclerotinia blight of Peanut from Infected Seed", examines the role of infected peanut seed from susceptible genotypes in transmitting the disease under greenhouse conditions. Based on the results of this test we should be able to establish if indeed contaminated seed from infected peanut fields, as suspected, has been an important vehicle in moving the pathogen from one peanut growing region to another. The last three manuscripts will be submitted for publication to either PEANUT SCIENCE or PLANT DISEASE. Appendix A entitled "Infection Process of *Sclerotinia minor* on a Resistant and Susceptible Peanut

Cultivar", summarises the procedure and our observations under the scanning electron microscope, of the various modes of infection of *S. minor* on peanut stems. Resistant and susceptible cultivars are compared at different time intervals, after placement of inoculum on the stem samples. Appendices B and C contain a listing of tables presenting detailed data that were useful in making summaries and conclusions presented and discussed in the proceeding chapters.

CHAPTER II

A DETACHED SHOOT TECHNIQUE TO EVALUATE THE REACTION OF PEANUT GENOTYPES TO *Sclerotinia minor*

ABSTRACT

Fifteen cm long shoot-tips from thirteen peanut genotypes were individually immersed in Hoagland's solution in 1 x 14 cm test tubes, and supported by foam plugs. All leaves were removed leaving about 1 cm of each petiole on the shoot. A 4 mm mycelial plug of *Sclerotinia minor*, taken from the periphery of a 2-day old culture grown on potato dextrose agar containing 100 ug/ml streptomycin sulfate (SPDA), was placed between the stem and a petiole in the middle of the shoot. Tubes with shoots were then placed in a fabricated polyethylene enclosure on a greenhouse bench where the day and night temperatures were 31 2C and 24 2C, respectively. Relative humidity (RH) was maintained at 95 to 100% by lining the bottom of the enclosure with wet burlap. Lesions appeared on shoot tips 3 days after inoculation, and their length was measured at various times. Two weeks after inoculation, tubes were drained, and shoots remained in the chamber at about 60-70% RH to allow sclerotial production. Sclerotia from each shoot were removed and counted, and their viability was determined by germination on SPDA at 25 2C in

darkness. This method is effective in differentiating reaction of peanut genotypes to *S. minor*.

INTRODUCTION

Sclerotinia blight of peanut (*Arachis hypogeae* L.), caused by *Sclerotinia minor* Jagger (10), is a major problem in peanut-producing areas of the United States, especially Virginia (14,17), North Carolina (13), and Oklahoma (20). Symptoms of the disease include flagging, wilting, necrosis of one or more stems (21), and relatively "dry" lesions produced on stems, stalks, branches or twigs with demarcations between healthy and diseased tissue (4,18). Under moist humid conditions white, cottony, fluffy mycelium appear on the base of diseased stems. The pathogen produces numerous sclerotia on the surface and within infected stems, pegs, and roots. Sclerotia can also form between the shell and seed of infected peanut pods. Sclerotinia blight, first observed in Oklahoma in 1972 (22), was widespread in most of the peanut-producing counties of the state by 1983 (23). In 1982, farm income losses in Virginia alone due to the disease was estimated at $8.6 million, and annual disease losses up to 13 % are common in years with favorable disease development (5).

Such losses have resulted in the immediate need for effective, economical strategies for disease management.

The disease, however, has not yet been controlled consistently and economically with fungicides. In addition to economical considerations, repeated application of specific fungicides within a growing season or a succession of growing seasons may select for a fungicide-tolerant strain of S. minor (11,16). Fungicide-tolerant strains of S. minor have not been noted under field conditions, however, in vitro development of resistance to dicarboximide fungicides by S. minor was reported (3,19). Resistant variants of other fungi have developed under field conditions, resulting in loss of disease control (5,15). This could also happen in Sclerotinia species.

Porter et al (13) were the first to screen peanut germplasm for resistance to S. minor, where they showed that the cv. Florigiant was the most tolerant cultivar among 19 genotypes tested, although 100% infection was observed at harvest. Coffelt and Porter (7) reported on the existence of morphological and physiological resistance of peanut genotypes to S. minor under field conditions. Brenneman et al. (6) recently reported on an excised stem technique that could be adapted for rapid evaluation of physiological resistance in peanut genotypes using lesion expansion, fungitoxicity of chemicals, and pathogenicity of isolates of S. minor.

Concentrated efforts are being directed in developing effective techniques to determine reaction and

identify resistance in peanut germplasm to S. minor. This paper reports on a detached shoot technique for preliminary screening of peanut genotypes for their reaction to S. minor under controlled conditions, using rate of lesion expansion, sclerotial production and viability among genotypes. A preliminary report and a brief description of the method has been reported (12).

MATERIALS AND METHODS

Fifteen cm long shoot tips from thirteen peanut genotypes (TX 798736, TX 804475, TX 798731, TX 798683, UF 73-4022, TX 771108, TX 771174, TP 107-3-8, TP 107-11-4, TX 833829, TX 835841, TX 833841, and Florunner), obtained from main stems of 8-week old plants grown in the greenhouse were used in this study. All genotypes were obtained from Dr. Olin Smith, Department of Crop Science, Texas A & M University, College Station, TX 77843. The lines TX 804475, TX 798736, UF-73-4022, TX 798683, and TX 798731 were selected because they exhibited some resistance to S. minor in replicated field plots at Stillwater, OK in 1986 (2). Florunner was selected because of its susceptibility to S. minor, and the other lines were included because of their varying susceptibility to S. minor as observed in field plots. The culture of S. minor used for inoculation was isolated from infected peanut cv. Florunner, and

maintained on potato dextrose agar containing 100 ug/ml of streptomycin sulfate (SPDA) at 25 2C.

All leaves on shoot tips except the primordial leaves were excised leaving about 1 cm of each petiole on the shoot. Individual shoots supported by a foam plug, were immersed in Hoagland's solution (9), in 11x14 cm test tubes. Each shoot was inoculated by placing a 4 mm mycelial plug of *S. minor*, taken from the periphery of a 2-day old culture grown on SPDA, at the axil between the stem and petiole at about mid portion of the shoot. Test tubes with inoculated stems were placed on wooden racks in fabricated clear polyethylene chambers (60 x 60 x 75 cm), the bottom of which was lined with wet burlap, and placed on greenhouse benches. The wet burlap maintained the relative humidity in the chamber between 95 and 100%. Temperatures in the chambers were 25 2C and 29 2C during the night and day, respectively. Ten shoots of each peanut genotype were inoculated with *S. minor* in each test, and shoots inoculated with plain SPDA plugs served as controls.

Lesion Expansion

Lesion lengths (cm) were measured as the distance from the site of inoculation to the farthest macroscopically visible extent of the lesion. This was done daily from day 3 after inoculation through day 7 when some of the shoots were completely colonized with mycelia of *S.*

minor. Mean lesion lengths of each genotype in each test, were calculated as the sum of individual lesion lengths divided by the total number of inoculated shoots whether infected or not. Length of lesions were linearly regressed against time after inoculation to determine the rate of lesion expansion, where the slope of the line represented the rate of lesion expansion (cm/day) on each genotype.

Inoculum Production

After the conclusion of lesion measurement, one end of the chamber was opened to lower the relative humudity in the chambers to 60-70 %. Hoagland solution was then drained from test tubes. The tubes with infected shoots on racks were left in the chambers for 2 weeks during which time sclerotia were formed on the surface and in pith cavities of infected stems. Total number of sclerotia per shoot both on the surface and within the pith cavity were estimated 3 weeks after inoculation.

Sclerotial Viability

Sclerotia collected from all genotypes were tested for viability. Sclerotia were washed under running tap water and surface sterilized with an aqueous solution of 0.5% sodium hypochlorite for 3 min. Five samples, each consisting of 10 sclerotia randomly picked from each infected peanut genotype, were plated on SPDA. Plates were incubated

at 25 2C in darkness. The number of germinated sclerotia in each plate was recorded daily from the 2nd day of incubation through the 5th day when most of the plates were filled with mycelial growth of *S. minor* from germinating sclerotia.

RESULTS

Lesion Development

The first noticeable symptoms on infected shoots were watersoaked lesions that started forming at the points of contact of *S. minor* and the stem 2 days following inoculation. Generally, these lesions expanded rapidly in the susceptible lines and completely girdled stems within 72 hrs. Shoots began to wilt following complete stem girdling. Stem girdling was observed on TP 107-3-8, TX 833841, TX 771174, TX 835841, TX 833829, TP 107-11-14 and Florunner. Genotypes that showed moderate susceptibility girdled slowly, starting with one sided infection, and wilting was delayed accordingly. This was observed on TX 771108, TX 798731, TX 798683 and UF 73-4022 (Table 1). Infection did not develop further at points of contact of inoculum and stems on some genotypes. These points were restricted or confined and no further lesion expansion was observed. These reactions were noticed on stems of TX 804475 and TX 798736 (Table 1).

Thus the following range of lesion types were observed

on inoculated shoots. 1) no visible lesions (rarely observed); 2) small scale-like lesions (less than .2 cm in length) generally restricted to point of contact of inoculum and stem; 3) rapidly expanding lesions restricted to one side of the stem; and 4) rapidly expanding lesions that completely girdled stem and expanded to colonize entire shoot.

The average lesion expansion rate on shoot tips was less for genotypes, TX 798683, TX 804475, and TX 798731, all identified as having some resistance to S. minor in field screening tests (2), as compared to the more susceptible genotypes, (TP107-3-8, Florunner, and TX 741174). Other genotypes had varying lesion expansion lengths, corresponding to their varying degrees of resistance to S. minor (Table 2). However, not all genotypes exactly exhibited this relationship, genotype TX 835841 known to be susceptible to S. minor under field conditions (2), had lower lesion expansion on shoots under the controlled conditions of the test. A close relative of this line, TX 833841, had a much higher level of lesion expansion.

The rates of lesion expansion as determined by slopes of regression lines for all the genotypes were compared. There was a significant difference ($P<0.05$) in the mean rates of lesion expansion among the peanut genotypes. (Table 3). Genotypes with the least fraction of stems infected, also had the lowest rates of lesion expansion,

while those with more stems infected had higher rates of lesion expansion (Tables 3,4).

Inoculum Production

Sclerotia were collected from both the surface of stems and inside of pith cavities. Not all infected stems produced sclerotia (Table 4). Among those that did, some produced sclerotia only on the surface of the stems. Genotypes TX 804475 and TX 798683, with lower rates of lesion expansion produced the lowest numbers of sclerotia on /in shoot tips (Tables 3,4). The other genotypes produced varying numbers of sclerotia corresponding to their varying rates of lesion expansion.

Sclerotial Viability

The percent viability of sclerotia collected from the surface and pith cavities of peanut genotypes as determined by germination on SPDA medium ranged from 54% to 74%, with TX 804475 having the least value (54 %), and TP107-3-8 the highest (74 %), (Table 4). Sclerotia collected from the susceptible genotypes TX 835841, Florunner, and TP 107-3-8 were significantly ($P<0.05$) more viable than sclerotia from the other genotypes evaluated (Table 4).

DISCUSSION

The detached shoot technique described in this paper provides a rapid evaluation procedure for preliminary screening of peanut genotypes for resistance to S. minor under greenhouse conditions. Actively growing mycelia from the periphery of S. minor culture plates provided inoculum in its optimum aggressive form to infect peanut stems. The relative high humidity provided by the wet burlaps and high temperatures within the polyethylene enclosures gave the proper conditions for S. minor infection (1).

Induction of lesions on some plants under optimum greenhouse conditions that are not normal in the field could be advantageous in screening genotypes for resistance. Genotypes that exhibit resistance under optimum conditions are likely to have high levels of resistance in field conditions in which their actual fitness under disease pressure is evaluated. Even genotypes classified as moderately resistant under artificial conditions may be of high resistance in the less favorable field conditions. Caution should be exercised in inoculation methods and incubation conditions that could produce high levels of disease in such a way that plants having some resistance are regarded as susceptible.

Most stem inoculations are performed through wounds

(6,8) which assist the pathogen in penetration of the host. Extrapolations of results from such laboratory wound inoculations to field conditions could be misleading. In this research, we were able to induce infection on shoots of the peanut genotypes without wounding.

Careful observations should be made of all possible changes shown on the plant or plant part in response to the presence of the pathogen. Point infections on some of our peanut genotypes suggest a form of hypersensitive response We suspect such a response may be initiated by a reaction of the pathogen to structural components of the cell wall. This still needs to be determined.

Lesion expansion for the genotype TX 835841, a susceptible genotype of S. minor under field conditions was comparable to that of resistant genotypes. We cannot at this point speculate why, except to equate this with the escape mechanism sometimes observed among susceptible plants grown in an area with a high inoculum density of their potential pathogen.

The rate of stem lesion expansion can be used to rank peanut genotypes for resistance to S. minor. The ranking obtained using this technique, however, has not been comparable to that obtained using disease incidence under field conditions (2). The rate of lesion expansion appears to be a simple and effective method of screening peanut genotypes for resistance to S. minor under controlled conditions.

Sclerotia collected from some stems of less susceptible genotypes were not fully matured. They had a whitish appearance and were not as dark as fully formed sclerotia on the susceptible genotypes. Low viability counts were shown by sclerotia from these cultivars. It appears viability of sclerotia is affected by the maturity level.

The method described may prove useful in assessing resistance to Sclerotinia blight in peanut genotypes and in screening populations segregating for resistance to the disease in a breeding program. It can be effectively adapted as a useful tool for rapid pre-evaluation of plant genotypes before whole plant evaluations in greenhouse and field. It has several added advantages over evaluation of intact plants. There is economy of labor and an experiment requires only 8-9 wk, 7-8 wk to grow the plants and 1 wk for disease development, and it requires a minimum of laboratory space. Quite a few shoots could be detached from a single plant and evaluated as individual treatments, a savings of plant material. The technique could be adapted for other uses including evaluation for fungicide resistance. It can give reproduceable results within a limited period of time without having to wait on seasonal field evaluations of whole plants.

The technique, however, should not be used as a substitute for field evaluations because of certain ramifications. No exact correlation has yet been established

between reaction of *S. minor* in the laboratory using the technique and disease resistance in the field. The genotype TX 804475, for example, had no disease in the field in 1986 (2), yet some of its shoots were fully susceptible to *S. minor* in this test. The genotype TX 835841, a highly susceptible genotype to *S. minor* under field conditions, showed some resistance to *S. minor* using this technique. We feel that a correlation is possible if greenhouse growing conditions can be established that closely mimic conditions in the field. Even if this is not possible, a genotype in which a lesion cannot develop by shoot inoculation would seem likely to be highly resistant to the disease under field conditions.

Our results showed that the genotypes TX 771174, TP 107-3-8, TP 107-11-4, TX 833829, TX 833841, TX 771108, UF 73-4022 and cv. Florunner, are very susceptible to *S. minor*, while the genotypes TX 798736, TX 804475, TX 798731, TX 798683 and TX 835841 have some resistance to *S. minor*, if we consider all the parameters evaluated. These results correlate fairly well with field results for most of the genotypes as already pointed out.

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Table 1. Reaction of peanut genotypes to *S. minor* three days after inoculation[1)]

Genotypes with[2)]		
Rapid stem girdle	One sided infection	Point infection
TP 107-3-8	TX 771108	TX 804475
TX 833841	TX 798731	TX 798736
TX 771174	TX 798683	-
TX 835841	UF 73-4022	-
TX 833829	-	-
TP 107-11-14	-	-
Florunner	-	-

1) Inoculation was accomplished by placing a plug of actively growing mycelia of *S. minor* on the leaf axil of detached shoot. Shoots were incubated in boxes under high relative humidity.

2) More than 50% of inoculated shoots in each of the categories showed the typical response of the group.

Table 2. Average lesion length (cm) per shoot tip of peanut genotypes in a 7-day period following inoculation with *S. minor*.

Genotype	Days after inoculation 3	4	5	6	7
TX 798683	.21	.58	1.18	1.75	2.23
TX 804475	.23	.60	1.25	1.70	2.25
TX 798731	.13	.60	1.35	1.98	2.85
TX 798736	.34	1.16	2.08	2.75	3.45
UF 73-4022	.34	1.16	2.05	3.20	4.30
TX 771174	.63	1.78	3.20	4.78	5.58
TX 771108	.50	1.05	2.13	3.30	4.33
TP 107-3-8	.83	2.18	3.68	5.03	6.15
TP 107-3-4	.48	1.23	2.28	3.60	4.60
TX 833829	.40	1.33	2.60	3.95	5.08
TX 835841	.57	.85	1.73	2.83	3.87
TX 833841	.88	1.58	2.90	3.88	4.90
Florunner	.58	1.60	2.83	4.38	5.85
LSD (.05)	.37	.81	1.24	1.12	1.42

a) Averages were calculated from 2 separate tests, each using 10 shoot tips per genotype.

Table 3 . Infection of peanut shoots and rate of lesion expansion (cm/day) after inoculation with *Sclerotinia minor*.

Genotype	Fraction of shoots		Rate of lesion expansion (cm/day) [a]		
	Infected [b]	W/sclerotia	Test 1	Test 2	Mean [c]
TX 798736	0.6	0.5	0.72	0.93	0.83
TX 804475	0.5	0.2	0.64	0.38	0.51
TX 798731	0.5	0.4	0.83	0.53	0.68
TX 798683	0.5	0.5	0.45	0.59	0.52
UF 73-4022	0.8	0.7	0.96	1.03	0.99
TX 771174	0.9	0.8	1.32	1.25	1.28
TX 771108	0.6	0.5	1.07	0.78	0.92
TP 107-3-8	0.8	0.8	1.42	1.27	1.34
TP 107-11-4	0.7	0.7	0.97	1.16	1.06
TX 833829	0.8	0.7	1.33	1.06	1.19
TX 835841	0.6	0.4	0.91	0.70	0.80
TX 833841	0.6	0.5	1.30	0.71	1.00
Florunner	0.9	0.9	1.32	1.34	1.33
LSD (.05)	-	-	0.47	0.51	0.49

a) Length of lesions (cm) was measured at 3, 4, 5, 6, and 7 days after inoculation.

b) Infection was determined by the formation of measurable lesions. Shoots with point infections were not considered infected.

c) Calculated from a total of 20 shoot tips in 2 tests with 10 shoots in each.

Linear Regression Model Equation:

$$Y = \text{Intercept} + \text{Slope} \cdot X$$

Table 4 . Production and viability of sclerotia of *Sclerotinia minor* on infected peanut shoot tips.

Genotype	Avg sclerotia/ stem surface [a]	Avg sclerotia/ pith cavity	Percent germination [b]
TX 798736	12	8	56
TX 804475	13	2	54
TX 798731	12	8	60
TX 798683	16	0	62
UF 73-4022	14	11	64
TX 771174	23	11	68
TX 771108	21	13	60
TP 107-3-8	16	11	74
TP 107-11-4	13	8	56
TX 833829	24	0	55
TX 835841	18	8	70
TX 833841	22	14	68
Florunner	25	17	71
LSD (.05)	8	4	11

a) Average number of sclerotia per infected shoot tip, determined from a total of 20 shoot tips in 2 tests with 10 shoots in each.

b) Sclerotia were plated on potato dextrose agar medium in 5 replications of 10 sclerotia per plate. Germination counts were made 3 days after incubation at 25 2C in darkness.

CHAPTER III

EVALUATION OF PEANUT GENOTYPES FOR RESISTANCE TO SCLEROTINIA BLIGHT IN FIELD PLOTS.

ABSTRACT

Reaction of 19 cultivated peanut genotypes to *Sclerotinia minor* was evaluated in small field plots at Stillwater, Oklahoma in 1986, 1987, and 1988. Entries were arranged in a completely randomized block design in field plots with four replications. For the three years of evaluation, average maximum disease incidence (percent) for the most resistant genotypes TX 804475, Toalson, TX 798731, TX 798683, and TX 798736 were 1.6, 3.9, 6.7 9.1 and 11.6, respectively; while the most susceptible genotypes, Florunner, Okrun and OK FH-15 had disease incidence values of 93.4, 93.5 and 91, respectively. Incidence of *S. minor* infection was recorded throughout the growing season, and data were logistically transformed to determine disease progress. Average disease progress values (r) were .006, .002, .003, .004 and .006, for the resistant genotypes Toalson, TX 804475, TX 798731, TX 798683 and TX 798736, respectively; while Florunner and its three hybrids (OK-FH 13, 15, and Okrun) had r values of 0.13, 0.12, 0.12 and 0.14, respectively. Other genotypes had varying degrees of resistance. Generally, genotypes with

an erect bunch growth habit tended to be more resistant to *S. minor* than those with a prostrate growth habit. These results were fairly in agreement with greenhouse tests using a detached shoot technique to evaluate the genotype reaction to *S. minor*.

INTRODUCTION

Sclerotinia blight of peanut (*Arachis hypogaea* L.), caused by *Sclerotinia minor* Jagger (13), was first observed in Virginia in 1971 (17), and in Oklahoma in 1972 (24). Since then it has become an important peanut disease in Virginia, North Carolina and Oklahoma. Sclerotinia blight was widespread in Oklahoma by 1983, it was observed in 12 of the 23 peanut-producing counties in the state (25).

Sclerotinia blight is a serious disease because of yield reduction and lack of established control practices. *S. minor* attacks plant parts that are in contact with the soil, causing lesions on stems and branches as well as pod rot (17). Lesions on branches are light tan with distinct demarcations between diseased and healthy tissue. Lesions turn dark brown followed by severe shredding of tissue and eventually the infected plant dies.

Porter et al (18) screened peanut cultivars in the field for resistance to *S. minor*, and of the 19 cultivars screened, Florigiant was the most tolerant cultivar, even

though 100 % infection was observed by harvest. Breeding lines with Spanish and Valencia pedigrees were more resistant to Sclerotinia blight than Florigiant (18). Coffelt and Porter (9) conducted 3 field screening tests where they identified resistant genotypes to *S. minor* based on morphological or physiological characteristics. Among 20 genotypes evaluated, 4 exhibited significantly fewer Sclerotinia blight symptoms than the other entries. In other crops, luxuriant plant growth has been shown to enhance the severity of *Sclerotinia* species infection (5,8,16). Resistance to *Sclerotium rolfsii* in peanut has been correlated with growth habit by some investigators (7), but not by others (11). Open canopies allow better penetration of sunlight and better circulation of air, inhibiting infections and colonization of food bases before infection (23). In beans (*Phaseolus vulgaris* L.) smaller plant types with open canopies are less susceptible to white mold, than larger plant types with dense canopies (5,8). Lettuce (*Lactuca sativa* L.) plant types with a raised growth habit are the most resistant to *S. minor* (16). Excessive plant growth and dense foliage favors reduced air circulation, promotes higher humidities, prolongs dew periods, and allows cooler soil surface temperatures (18). Prime requisites for infection of other crops by *Sclerotinia* spp. include high humidity (12, 15), moderate temperatures of 16-27 C (6,12) and heavy dew (10).

Sclerotinia spp. can be controlled by various fungicides (3,8,19). Porter and Rud (20) suppressed Sclerotinia blight in peanut with the use of dinitrophenol herbicides. Brenneman (4) reported on the possible development of resistance of *S. minor* to some fungicides. Current knowledge of sources and stability of resistance in peanut to *S. minor* is limited because few cultivars have been evaluated.

The objectives of this field study were 1) to evaluate the reaction of selected cultivated peanut genotypes to *S. minor*, and 2) to determine Sclerotinia blight disease progress on the cultivated lines. Preliminary results of this study have been reported (1).

MATERIALS AND METHODS

Nineteen peanut genotypes were evaluated in small field plots in 1986 for reaction to *S. minor* (Table 1). The genotypes TX 804475, TX 798683, and TX 798731 were included in the test because they had been observed to exhibit resistance to *S. minor* in a detached shoot technique used to evaluate peanut genotypes for initial reaction to *S. minor* (14). Florunner was included because it is known to be susceptible to *S. minor*, while Toalson, a Spanish cultivar was included because of its reported resistance to other soilborne pathogens (22). Other selections were

included because of their varying reactions to *S. minor*. The same lines were evaluated in 1987 and 1988, as in 1986 to ascertain their reaction to *S. minor*.

The field site choosen at the Oklahoma Experiment Station research farm in Stillwater, was artificially infested with *S. minor* in 1981, and had been planted to peanuts continuously. The soil had a sclerotial density of about 3-5 sclerotia per 100 g of soil. The soil was a sandy loam type that is typically used for peanut production.

A randomized complete block design with four replicates was used during each of the 3 years of this study. Each block consisted of 19 rows 4.55 m long and 0.91 m apart. Blocks were separated by 1.5 m alleys. To ensure a good stand, seeding rates were doubled at planting and thinned after germination to obtain plants spaced at about 0.3 m apart. Planting was done each year towards the end of May, and harvesting towards the end of October, allowing an average of about 150 growing days. Recommended standard production practices for fertilizer, herbicide and irrigation were followed every year in all tests. No disease control measures were employed for soilborne or airborne diseases.

The percentage of plants infected was determined by presence of visible above ground symptoms. A plant having evidence of disease, however slight or severe was considered infected and flagged each time disease incidence readings were taken at intervals during the growing season. Six

readings were taken throughout the growing season in 1986, 1987 and 1988. At the end of each growing season plants were individually hand-dug, separated and harvested according to visual maturity estimates.

Total possible infections were calculated using the formula for simple interest disease:

$$\log_e \frac{1}{1-X_t}$$

where X_t equals the proportion of infected plants in each plot row at a particular disease incidence rating date. Values of total possible infection were linearly regressed overtime to obtain rate of disease progress r (26). The Duncan multiple range test was used to separate between means of % disease incidence and means of disease progress among genotypes.

RESULTS

In 1986, *S. minor* infected all peanut genotypes evaluated in the field except Toalson and TX 804475, and maximum disease incidence ranged from 0 to 100 % (Table 1). Above average rainfall and lower than normal night temperatures in August and September were favorable for early appearance of Sclerotinia blight and faster disease progression. Eight genotypes (TX 835841, TX 771174, OK FH-13,

Okrun, OK FH-15, Florunner, TX 833841, and TP 107-3-8 had maximum DI values above 80 %. Disease progress values for these susceptible genotypes were high; and ranging from 0.04 to 0.173 (Table 2). Disease progress values were correspondingly low for the resistant genotypes where values were less than 0.01 (Table 2).

In 1987, as in 1986, all genotypes were infected to some degree by S. minor in the field, except Toalson and TX 804475. Four genotypes (TX 798731, TX 798736, TX 798683, and UF 73-4022) had significantly less Sclerotinia blight than all the other genotypes (Table 1). Except for three genotypes (TX 833829, TX 835841, and TP 107-3-8), disease progress values in 1987 were generally lower than those of 1986, on the same genotypes. That year also had a late disease outburst where the first disease symptoms were observed on Sept. 6th, compared to August 22nd when first disease symptoms were observed in 1986. Environmental conditions were also less suitable for the disease than in 1986. Even with this difference in environmental conditions, the general trend in both DI and disease progress values was very similar (Tables 1& 2).

In 1988, all genotypes without exception, were infected by S. minor (Table 1). This was the first time in three years of study that Toalson and TX 804475 became infected during the season. Maximum DI for Toalson was even more than for three other genotypes (TX 798683, TX

798731 and UF 73-4022) that had exhibited some disease in previous years. TX 804475 still had the lowest maximum DI value of all the genotypes. Among the genotypes previously classified as susceptible, the trend was similar to previous years. Florunner and lines derived from it were among the most susceptible (Table 1). Disease progress values among the susceptible genotypes, were consistent with past years as well, (Table 2). As in 1987, there was a late disease development. The first disease symptoms were detected on Sept. 5th 1988.

Generally, genotypes with runner and prostrate growth habits were more susceptible than those with an upright (bunch) growth habit in all 3 years of this study (Table 3). In all 3 years in which disease was monitored throughout the growing season, Sclerotinia blight generally increased in severity with time for all the susceptible genotypes.

DISCUSSION

The best time to make observations and recordings of disease on infected plants was in the morning hours. This was when crown regions and points of initial contact and infection on branches of susceptible plants had the cottony or fluffy appearance characteristic of *S. minor* under moist conditions. Observations made after midday were likely to be less accurate because the mycelial growth was not obvious

following the penetration of sunlight through leaf canopy, and the evaporation of the morning dew. Readings at this time of day were likely to be based mainly on wilting symptoms which occured only on the more severely infected plants. Irregularly shaped sclerotia could only be detected on the surface of infected branches when the plants were completely infected and near dead.

Resistant plants either actively restricted lesion development after infection, or conditions that inhibited initial infection inhibited further lesion development. Restricted lesion development has been observed on genotype shoots inoculated using the detached shoot technique (14). Some genotypes showed a stable and high level of resistance under field conditions. Toalson, TX 804475, TX 798731, TX 798736, and TX 798683 were the only genotypes that had little or no disease in all 3 trials. The high level of resistance evident in Toalson and TX 804475 represent new sources of disease resistance and indicate that breeding for Sclerotinia blight resistance in peanut is a viable alternative control strategy. This has been noted also in studies using other cultivars (9,18).

Two modes of infection were observed on susceptible plants. Infection initiated from branches at points of contact with the soil surface were less dramatic and generally did not result in entire plant death. Only infected branches died. Infections initiated from the crown

region towards the branches was more dramatic. Under high relative humidity, especially in the morning periods, such infected plants looked green and healthy. As the day progressed and the sun penetrated into the plant canopy, sudden wilting and death of the entire plant occurred. This is because transpiration rate of plants increased with increased atmospheric temperature, while fungal mycelia invaded the vascular bundles and greatly inhibited water conduction.

The five most resistant genotypes, (Toalson, TX 804475, TX 798731, TX 798683, and TX 798736) identified in 1986, 87 and 88 (Table 1), have more upright plant canopy structure than the dense spreading type of most of the other genotypes tested. Limbs of these genotypes have limited contact with the soil and inoculum, and development of a favorable microclimate is extremely restricted because a close canopy rarely develops. Morphologic resistance in such bunch-type peanuts with an upright growth habit may be related to the number, size and distribution of leaves within the canopy as has been reported for resistance to white mold in beans caused by *S. sclerotiorum* (23). Based on results in other crops (2,9), it has been suggested that resistance in these erect types of genotypes may be more of a morphological escape mechanism rather than physiologic resistance. Genotypes which exhibit low disease incidence or severity in field evaluations should be tested in the growth chamber

to determine if disease escape or physiologic resistance is present. The almost 100% reduction in Sclerotinia blight for TX 804475, for example, compared with Florunner (Table 1), may be physiologic. In early tests using the detached shoot technique (14), TX 804475 was less susceptible to *S. minor* than Florunner and other genotypes with a similar growth habit. In these tests, when the 2 genotypes were artificially inoculated with *S. minor* and compared, the average lesion length on infected shoots of TX 804475 was 22 mm compared to 59 mm on infected shoots of Florunner, seven days after inoculation (14). Schwartz et al (23) reported similar lesion lengths on resistant (10-40 mm) and susceptible (30-100 mm) dry bean genotypes in a growth chamber test. Field trials and greenhouse evaluations were not correlated for all genotypes. This lack of correlation between field and greenhouse studies is not uncommon (21). Small differences in resistance to infection can be hidden by variation in environmental or biological factors that can be induced by differences in genotype architecture (21). The identification of genotype characteristics that affect the responses of peanut genotypes to disease under field conditions increases the difficulty of identifying sources of field resistance to this pathogen using only greenhouse screening techniques. We believe there is some physiologic resistance involved in our genotypes, in addition to morphologic escape possible under field conditions. Resistance to *S. minor* in genotypes

TX 804475 and TX 798731, for example, appeared to be independent of canopy morphology. Comparative evaluations of all reported sources of resistance would be valuable in identifying the most resistant cultivars for use in plant breeding programs. Further studies are required on the nature of resistance to *S. minor* in peanut germplasm, and the relative importance of physiological resistance and disease escape mechanisms under field conditions. Development of peanut cultivars with resistance to *S. minor* appears feasible and may represent an effective and economic strategy for disease control in areas where this disease is prevalent, and practical means of control have not been developed.

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Table 1. Maximum Sclerotinia blight incidence in cultivatec peanut genotypes in field plots in 3 years.

Genotype	% Disease Incidence[a]		
	1986	1987	1988
TX 804475	0 c	0 e	5 d
Toalson	0 c	0 e	12 d
TX 798731	10 c	2 e	8 d
TX 798683	7 c	5 e	5 d
TX 798736	16 c	5 e	14 d
UF 73-4022	24 bc	9 e	8 d
TX 771108	48 bc	24 de	39 d
TAMNUT 74	42 bc	32 de	47 cd
Sn 55-437	21 bc	43 de	20 d
TX 833829	40 bc	56 de	42 cd
TX 835841	100 a	61 bcd	68 bcd
TX 771174	100 a	69 cde	68 bcd
Sn 73-33	76 b	74 abc	51 cd
OK FH-13	100 a	76 ab	81 ab
Okrun	100 a	86 a	95 a
TX 833841	98 a	86 a	53 cd
Florunner	98 a	92 a	90 a
OK FH-15	100 a	92 a	81 ab
TP 107-3-8	82 a	95 a	87 a

[a] Based on last readings taken at end of growing season

[b] Means followed by the same letter (within block) are not significantly different (P = 0.05) according to Duncan's multiple range test.

Table 2. Sclerotinia blight Disease Progress in small field plots in a 3 year period

Genotype	Disease Progress (r)[a]		
	1986	1987	1988
TX 804475	0 c	0 d	.002 d
Toalson	0 c	0 d	.008 d
TX 798731	.003 c	0 d	.005 d
TX 798683	.006 c	.002 d	.003 d
TX 798736	.006 c	.002 d	.009 d
UF 73-4022	.009 c	.004 d	.005 d
TX 771108	.024 c	.006 d	.022 d
TAMNUT 74	.019 c	.012 d	.035 cd
Sn 55-437	.006 c	.020 d	.014 d
TX 833829	.046 c	.021 d	.031 d
TX 835841	.124 ab	.049 bcd	.066 bcd
TX 771174	.159 a	.029 cd	.065 bcd
OK FH-13	.173 a	.048 bcd	.134 ab
Sn 73-33	.050 c	.031 cd	.046 bcd
Okrun	.167 a	.101 a	.198 a
OK FH-15	.160 a	.043 cds	.133 ab
TX 833841	.135 ab	.074 abc	.060 bcd
TP 107-3-8	.099 b	.093 ab	.149 a
Florunner	.138 ab	.078 ab	.203 a

a) Calculated by regressing disease incidence data, $\log_e \frac{1}{1-Xt}$, over time.

b) Means within a column followed by the same letter are not significantly different (P=.05) according to DMRT.

TABLE 3: Reaction of *Sclerotinia minor* to peanut genotypes with different growth habits.

Genotype	Growth Habit	Avg Max DI [b]	Reaction [a]
TX 804475	Bunch	2	Resistant
TX 798731	Bunch	7	Resistant
TX 798683	Bunch	6	Resistant
TX 798736	Bunch	12	Resistant
Toalson	Bunch	4	Resistant
Florunner	Prostrate	93	Susceptible
TX 833841	Prostrate	79	Susceptible
TX 835841	Prostrate	76	Susceptible
TP 107-3-8	Prostrate	88	Susceptible
LSD (0.05)	-	20	-

a) Based on average data over three growing seasons

b) Maximum disease incidences were read on 9/24, 10/11 and 9/22 in 1986, 1987, and 1988, respectively.

CHAPTER IV

SCLEROTIAL PRODUCTION AND VIABILITY ON PEANUT GENOTYPES PLANTED IN *SCLEROTINIA MINOR*-INFESTED FIELD PLOTS.

ABSTRACT

Nineteen peanut genotypes were evaluated in field plots at Stillwater, Oklahoma for reaction to *S. minor* in 1986, 1987, and 1988. After digging, plants were separated into infected and healthy groups, and stored on greenhouse benches to dry. Number of pods per plant were taken from randomly selected plants in each category to determine the effect of *S. minor* on pod yield of the lines. Sclerotia were collected from randomly selected stem segments and pods of susceptible lines and evaluated for viability. Most genotypes that had sclerotia on pods also had sclerotia on stems. Of 10 susceptible genotypes, more sclerotia were formed in/on stems than in/on pods. Sclerotia collected from stems were significantly more viable than those from pods in all of the susceptible genotypes. Highest sclerotial viability was 82% from stems of cv. Tamnut 74, and 60% for pods of Florunner. Lowest was 29% from stems and 13% from pods on genotypes TX 771174 and TX 835841, respectively. No sclerotia were produced on resistant genotypes.

INTRODUCTION

Epidemics of Sclerotinia blight of peanut (*Arachis hypogaea* L.) caused by *Sclerotinia minor* have resulted in significant yield losses in Virginia and Oklahoma, where an estimated 3 to 5 % of the crop is lost due to this disease each year (17).

White, cottony, fluffy mycelium appear on the base of diseased stems. Infected branches become chlorotic and eventually die. The pathogen produces numerous sclerotia on the surface and within infected stems, pegs, and roots. Sclerotia also can form between the shell and seed of infected peanut pods .

S. minor overwinters by producing small (0.5-2.0 mm), black, irregularly-shaped sclerotia that persist in soil for long periods of time even under adverse conditions. Sclerotia germinate eruptively (11) by producing a plug of vegetative mycelium through the rind (16). The inoculum density of germinable sclerotia in soil and prevalent conditions that favor sclerotial germination influence the incidence of Sclerotinia blight. Germinable sclerotia have been found in soil throughout the plow layer (the top 20 cm) of fields having a previous history of Sclerotinia blight even after a field has not been planted to peanuts for 4 yr. One sclerotium per 100 g of soil is sufficient to cause severe

infection under favorable conditions for disease development (19). Sclerotinia blight is most severe when cool and moist weather conditions prevail (1), or irrigation is excessive during the growing season, and especially near harvest time (12). A single eruptively germinating sclerotium can infect and kill a plant without prior colonization of a senesence food base (11). Studies on survival of sclerotia of *Sclerotinia* spp. have been carried out under a wide range of soil conditions. Results have been variable, ranging from less than 1 month to 11 years (2, 15).

Davis (8) was of the opinion that sclerotia of *S. sclerotiorum* near the soil surface do not remain viable for more than 1 year. Young and Morris (24), however, reported that at least 4 years rotation was needed before sunflower can be grown on a field with a history of Sclerotinia wilt. In the case of bean white mold, Starr et al (22) suggested 3 to 5 year period of nonhost crops. Based on information provided by farmers in New Jersey, *S. minor* on lettuce can survive for 4-5 years (3). Exact information on length of survival of sclerotia in peanut fields is not available.

Intense efforts have been directed recently at screening peanut genotypes for resistance to *S. minor* (4, 6, 18). Most have based their evaluations on disease incidence and crop yield, and none has considered sclerotial production or viability among the genotypes evaluated as a parameter to consider in selecting genotypes for resistance to *S. minor*.

The objectives of this research were to determine the amount of sclerotia of S. minor produced on and in infected pods and stems of peanut genotypes and to determine differences in viability of sclerotia from pods and stems of the genotypes. The effect of the disease on pod yield of peanut was also evaluated. A preliminary report of this research has been reported (5).

MATERIALS AND METHODS

Peanut genotypes and establishment of field plots.

In 1986, 1987, and 1988, nineteen peanut genotypes (Table 1), most selected because of their varying degrees of reaction to S. minor under controlled conditions using a detached shoot technique (13), were evaluated in field plots for yield under disease pressure, sclerotial production, and sclerotial viability. The field plots were infested with S. minor, in 1981 and planted to peanut annually. These plots had an inoculum density of 3-5 sclerotia per 100 g of soil at the start of each growing season. Plots were arranged in a randomized complete block design with 4 replications. Blocks consisted of 19 rows, each 4.55 m long and 0.91 m apart. Blocks were separated by 1.5 m alleys. Peanut was planted each year towards the end of May, and harvested towards the end of October, allowing an average of about 150 growing days per season. For each year, during the growing

season, plants in each row were marked with surveyor flags at the first date when typical symptoms of *S. minor* were observed and continued until near harvest. At the end of each growing season, plants were hand-dug and separated into infected and noninfected groups in each row. Plants were then bagged in burlap sacs immediately after digging and stored on greenhouse benches where temperatures averaged about 23 C by night, and 28 C by day. In all years, plants were stored to dry for a period of about 3 months.

Pod Yield

Twenty four indivudual plants flagged during the growing season as infected by *S. minor*, were randomly selected from each of the susceptible genotypes. Another 24 were selected similarly from the uninfected plants of the genotypes. A lesser number of plants from other genotypes were randomly selected for the same purpose. Pods were collected from each selected plant, counted and weighed. Average yield values for infected and noninfected plants of each genotype were estimated using the pod count value of each of the plants.

Inoculum Production

Ten pods were randomly picked from each of a minimum of six and a maximum of 24 infected plants depending on the number of infected plants in a particular genotype, to

evaluate sclerotial production. Sclerotia were examined on the surface of pods and counted. Pods were individually hand-cracked and the number of sclerotia inside the pod counted. Sclerotia collected from both outside and inside the pods of each genotype were pooled.

Twenty four 10 cm segments from infected stems, about 10 cm away from the crown, were randomly collected and examined for sclerotial production both outside and inside the stem tissues. Sclerotia were counted both from the surface and inside pith cavity where present, and collected. As with sclerotia from pods, sclerotia from stems of each genotype were pooled.

Sclerotial Viability

Sclerotia collected from pods and stems of infected plants of each genotype were tested for viability. Sclerotia were washed under running tap water and surface disinfected with an aqueous solution of 0.5 % sodium hypochlorite for 3 min, and then rinsed in sterile distilled water. A total of 100 sclerotia from the pods and 100 from the stems of each susceptible genotype were randomly picked, and plated in groups of 10 sclerotia per 9 cm petri plate containing 10 ml of potato dextrose agar with 100 ug strepmycin sulfate / ml (SPDA). Plates were incubated in darkness at 25 2C Number of germinating sclerotia in each plate was recorded after 5 days of incubation.

Statistical Analysis

Data were analyzed using standard analysis of variance procedures.

RESULTS

Pod Yield

Sclerotinia blight was very severe in the plots in 1986. Several genotypes had 100% infection and there were no healthy plants with which pod yield of infected plants could be compared (Table 1). In peanut genotypes where all plants were not infected, there was a significant difference in average number of pods produced on healthy and infected plants in all genotypes compared (Fig 1). This relationship was also true based on the average pod weight per plant among genotypes, where pods from healthy plants weighed significantly more than those from infected plants (Table 1). Comparing pod number and pod weight per plant among genotypes, there was no significant difference between the average pod number and pod weight per plant for all of the susceptible genotypes in all 3 yrs of the study (Fig 2). Thus pod number and/or pod weight per plant could be conveniently used interchangeably to compare the effect of the pathogen among genotypes on yield.

In 1987 with a lower incidence of Sclerotinia blight

due to late occurence of ideal conditions for disease development, no genotype had 100 % disease incidence as in 1986 (Table 2). However, the effect of the pathogen on pod yield was similar to 1986. Pod yield and pod weight per plant were significantly different in the healthy than infected plants among all genotypes except UF 73-4022 and Sn 55-437 (Table 2). In 1988, conditions were similar to 1987 except that all genotypes without exception had some infection by S. minor (Table 3). There was no apparent relationship between severity of infection and average pod yield per plant among genotypes for all 3 yrs. Among susceptible genotypes some with high maximum disease incidence values had better pod yield than others with lower maximum disease incidence values (Tables 1,2,&3). Pod yield per plant among resistant genotypes was greater than in susceptible genotypes.

Sclerotial Production

In all 3 yrs of evaluation, no sclerotia were collected on peanut pods from genotypes classified as resistant from maximum disease incidence and disease progress values (Table 5). Two resistant genotypes, TX 798736 and TX 798683, however, produced sclerotia on stems in 1987. All other genotypes that produced sclerotia on pods also produced sclerotia on stems (Table 4). Average number of sclerotia collected per 100 pods of susceptible peanut genotypes

sampled, varied among genotypes for all 3 yrs of evaluation (Table 5). There was no strong correlation between the degree of susceptibility and the amount of sclerotia produced. All genotypes with an average maximum disease incidence (DI) value above 40% produced some sclerotia on pods sampled (Table 5), however, the amount of sclerotia produced per genotype varied from year to year even with fairly stable DI values for each year (Fig 3). The genotype OK FH-13, for example, (Table 5), produced only 7 sclerotia per 100 pods sampled in 1986, and 21 in 1987 despite a high average DI value of 86 %. TX 833841, another susceptible genotype, produced 38 sclerotia per 100 pods sampled in 1986 and as high as 119 sclerotia per 100 pods in 1987, even though the DI values for this genotype were 100 and 86 % in 1986 and 1987, respectively.

In 1987 and 1988, some susceptible genotypes (Sn 55-437 and TX 833829) that produced no sclerotia in 1986 produced large numbers of sclerotia (Table 5). Sclerotia was collected from susceptible pods of the genotype UF 73-4022, only in 1987. Sclerotial production was significantly greater in some genotypes (Florunner, Okrun and TX 771174) in 1987 and 1988 than in 1986.

Sclerotial Viability

For all 3 yrs of evaluation, the average viability of scleroia produced on stems of susceptible peanut genotypes

was significantly higher than that of sclerotia produced on pods. Within individual genotypes, all produced sclerotia on stems that were significantly more viable than sclerotia on pods, in 1987 and 1988 (Fig 4). In 1986, however, only four genotypes, Tamnut 74, OK FH-15, TX 835841 and TP 107-3-8 produced sclerotia on stems that were significantly more viable than those from pods (Table 6). Between genotypes, there was a significant difference in viability of sclerotia produced on pods and stems for all years except in 1987 when sclerotial viability from stems was not significantly different among genotypes.

DISCUSSION

Sclerotia, usually formed on aboveground infected peanut plant tissue and infected pods are deposited on the soil surface along with infected crop debris and are incorporated into the soil at various depths during land preparation for the next crop. It is not surprising that all plants with sclerotia on pods also had sclerotia on stems. The infection process of plants is initiated at the crown region or on stem branches in close contact with soil, before progressing to other parts of the plant. It is only in severe cases of infection that the pathogen spreads through the pegs into the pods where sclerotia are eventually produced. A large number of pods may be left on

and in soil at harvest. In an irrigation study on Florunner peanut, 630-778 kg/ha of pods were removed from the soil after those on the vines were harvested (23). These loose pods normally would remain in the soil and could provide a large reservoir of inoculum for *Rhizoctonia solani*, *Pythium* spp. and *Sclerotinia* spp.

Many factors are known to affect survival and viability of sclerotia in soil (7). Survival of sclerotia varies greatly among different soils and at different soil moisture tensions (2, 10). Weather conditions, especially heavy rains where flooding is involved, seem to affect the formation of sclerotia and subsequent availability the next cropping season. In 1986, despite a 100% maximum disease incidence in some of the genotypes, the amount of sclerotia produced on/in pods was considerably less than that produced on/in pods in 1987 and 1988 for most genotypes even though the disease was less severe in the latter years. Since sclerotia form from the thickening of mycelia (9), flood waters that submerged the plots for two days in 1986, possibly washed off most of the mycelia on and thus less sclerotia could develop on the surface of stems and pods. Soil indexing for viable sclerotia may allow the prediction of the potential for Sclerotinia blight of peanut in a field and also aid in determining the effect of cropping sequence, cultural practices, and biological control strategies on survival of sclerotia in soil. Cultural practices

of soil disking and possibly rotating with a non-host crop should always be considered when disease severity in field increases and is suspected to be due to a possible inoculum buildup in the soil.

Data on numbers of sclerotia of S. minor in soil however, must pertain to sclerotia that are competent to germinate by eruptive mycelial growth because this is the infective propagule (11). Unfortunately, numbers of viable sclerotia as determined by ability to grow on nutrient media such as SPDA often correlate poorly with % infection because sclerotia capable of only weak hyphal germination do not infect unless a nonliving food base is available (20). Such data are mainly useful in comparative analysis. Conclusions made therefore, based on viability studies on media alone, are only speculative. Such comparative studies could give results that are useful in making recommendations on cultural practices for disease control.

In this study the viability of sclerotia produced on stems was significantly higher than that from pods, for most of the genotypes evaluated. This means that unharvested stem debris left on the soil surface are a more common source of inoculum carry-over than unharvested pods if we consider viability as an important prediction for infection. It will be good to include in our cultural control strategies, clearing all infected stem pieces off the field in an attempt to reduce sources of initial inoculum for

the next cropping season. Fortunately, this is easier to do than trying to get rid of buried infected pods left in the soil, if the reults were opposite. The need for clean seed with little or no debris from infected fields is also clear. Such debris carrying more viable sclerotia than pods could be a serious source of disease spread in new fields. This finding could also support the possible important role of infected peanut hay in disease dissemination, within and between fields (14).

Viability of sclerotia from stems of peanut plants may be affected by flooding. Moore (15) had reported this to be the case when he used flooding as a means of destroying the sclerotia of S. sclerotiorum. Sclerotia from stems of genotypes planted in 1987 and 1988, were significantly more viable than those of 1986 genotypes, when flooding of the plots occured and plants were submerged under water for 2 days, towards the end of the growing season. Flooding, however, seems to have had less effect on the viability of the less exposed sclerotia in infected pods. Average viability was basically the same in sclerotia from pods of all 3 years.

Peanut pods detached from infected plants before or during harvest and debris (leaves and stems) from the combine discharge may remain on or in the soil for several weeks or months until the land is prepared for planting the next crop. These become a reservoir for potential inoculum for the

next season. The tillage practices used could determine whether sclerotia contained in unharvested pods and stem debris would be a problem on the next peanut crop or another crop immediately following peanut. Inoculum levels do not necessarily increase yearly in soil just because of these unharvested pods and stem debris that serve as inoculum reservoirs. It has been shown that in Nebraska soil populations of sclerotia did not increase even in fields where annual epidemics of white mold of beans caused by S. sclerotiorum had occured (21). Similarly, the soil populations of sclerotia in a bean field in New York remained about the same even after three consecutive years of severe epidemics of white mold that resulted in complete loss of the crop each year (1). In our study, we sampled the field extensively for sclerotial density each year just prior to planting. The sclerotial density remained fairly constant at 3-5 sclerotia per 100 g of soil over a 3-year period, despite a continuous cropping on the plots with peanuts, some with annual disease incidence values of up to 100%.

Weather conditions greatly affect pod yield under severe disease conditions. Pod yield in 1986 was considerably less than for 1987 and 1988, for each genotype compared. Excess moisture in the pegging zone due to flooding of the plots, accelerated rotting of infected pegs and thus more pods were left in the ground at harvest. Early planting whenever possible, would lead to early harvesting, thus avoiding some of these weather constraints on crop yield.

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Table 1. Effect of *Sclerotinia minor* on pod yield and pod weight of peanut genotypes in field plots; 1986

Genotype	Maximum DI	Pod #/plant INF	Pod #/plant HLTHY	P	Pod wt(g)/plant INF	Pod wt(g)/plant HLTHY	P
Toalson	0	*	56		*	51	
TX 804475	0	*	70		*	65	
TX 798731	10	36	61	.01	24	55	.01
TX 798736	16	44	69	.01	30	49	.01
TX 798683	16	38	70	.01	31	64	.01
Sn 55-437	21	35	47	ns	28	35	ns
UF 74-4022	24	23	45	.01	26	48	.01
TX 771108	48	28	58	.01	26	52	.01
Tamnut 74	42	23	53	.01	20	42	.01
Sn 73-33	76	22	35	.05	20	38	.05
TX 833829	40	19	34	.05	17	39	.05
TP 107-3-8	82	21	**		21	**	
Florunner	98	27	**		26	**	
TX 835841	100	24	**		28	**	
OK FH-13	100	20	**		22	**	
TX 833841	100	40	**		42	**	
Okrun	100	21	**		22	**	
TX 771174	100	25	**		25	**	
OK FH-15	100	25	**		26	**	

* None of the plants were infected

** None of the plants were healthy

P = Probability

Table 2. Effect of *Sclerotinia minor* pod yield and pod weight of peanut genotypes in field plots; 1987

Genotype	Maximum DI	Pod #/plant INF	Pod #/plant HLTHY	P	Pod wt(g)/plant INF	Pod wt(g)/plant HLTHY	P
Toalson	0	*	73		*	63	
TX 804475	0	*	75		*	66	
TX 798731	2	*	95		*	98	
TX 798736	5	54	98	.01	47	93	.01
TX 798683	5	53	93	.01	47	94	.01
UF 43-4022	9	54	65	ns	58	72	.05
TX 771108	24	52	76	.05	44	73	.05
Tamnut 74	32	53	89	.05	44	74	.05
Sn 55-437	43	44	59	ns	46	58	ns
TX 833829	56	34	43	.05	24	40	.05
TX 835841	61	50	71	.01	48	68	.01
TX 771174	69	62	92	.01	59	83	.01
Sn 73-33	74	36	53	.01	34	47	.01
OK FH-13	76	53	68	.05	60	74	.05
Okrun	86	51	71	.01	53	76	.05
TX 833841	86	41	59	.05	47	64	.05
Florunner	92	57	76	.05	59	81	.05
OK FH-15	92	49	69	.05	50	71	.05
TP 107-3-8	95	46	68	.05	51	74	.05

* None of the plants were infected

P = Probability

Table 3. Effect of *Sclerotinia minor* on pod yield and pod weight of peanut genotypes; 1988

Genotype	Maximum DI	Pod #/plant INF	Pod #/plant HLTHY	P	Pod wt(g)/plant INF	Pod wt(g)/plant HLTHY	P
TX 804475	5	56	71	.05	50	60	.05
TX 798683	5	53	70	.05	48	64	.05
TX 798731	8	59	81	.05	59	75	.05
UF 73-4022	8	56	64	ns	57	65	ns
Toalson	12	55	70	.05	44	55	.05
TX 798736	14	69	71	ns	59	66	ns
Sn 55-437	20	53	87	.05	39	69	.05
TX 771108	39	52	81	.05	47	77	.05
TX 833829	42	23	39	.01	17	29	.01
Tamnut 74	47	50	71	.05	44	53	.05
Sn 73-33	51	30	52	.05	25	38	.05
TX 833841	53	49	76	.01	46	73	.01
TX 835841	68	33	71	.01	33	69	.01
TX 771174	68	57	*		58	*	
OK-FH 15	81	35	*		33	*	
OK-FH 13	81	38	*		35	*	
TP 107-3-8	87	39	*		36	*	
Florunner	90	41	*		39	*	
Okrun	95	41	*		38	*	

* No healthy plants were available

P = Probability

Table 4. Production of sclerotia of *Sclerotinia minor* on pods and stems of peanut genotypes in field plots.

Genotypes	Maximum DI	Reaction a)	Sclerotia produced on Pods	Stems
Toalson	4	R	–	–
TX 804475	2	R	–	–
TX 798731	7	R	–	–
TX 798736	12	R	–	+
TX 798683	6	R	–	+
Sn 55-437	28	S	+	+
UF 43-4022	14	S	+	+
TX 771108	37	S	+	+
Tamnut 74	40	S	+	+
Sn 73-33	67	S	+	+
TX 833829	46	S	+	+
TP 107-3-8	88	S	+	+
Florunner	93	S	+	+
TX 835841	76	S	+	+
OK FH-13	86	S	+	+
TX 833841	79	S	+	+
Okrun	94	S	+	+
TX 771174	79	S	+	+
OK FH-15	91	S	+	+

R = Resistant
S = Susceptible

– = No sclerotia produced
+ = Sclerotia produced

a) Classification based on average maximum DI for 3 yrs in field evaluations

Table 5. Amount of sclerotia of Sclerotinia minor produced on/in pods of peanut genotypes grown in S. minor-infested field plots for 3 years

Genotype	DI [b]	Rxn [c]	Number of sclerotia /100 pods [a] 1986	1987	1988
Toalson	4	R	0	0	0
TX 804475	2	R	0	0	0
TX 798736	12	R	0	0	0
TX 798731	7	R	0	0	0
TX 798683	6	R	0	0	0
TX 833829	46	S	0	73	23
UF 73-4022	14	S	0	12	0
Sn 55-437	28	S	0	26	19
TX 771108	37	S	5	40	25
Tamnut 74	40	S	9	0	7
TX 771174	79	S	6	44	29
Florunner	93	S	14	39	42
Okrun	94	S	10	41	58
TX 833841	79	S	38	119	53
Sn 73-33	67	S	50	49	50
TP 107-3-8	88	S	13	33	25
TX 835841	76	S	43	73	67
OK FH-13	86	S	7	21	70
OK FH-15	91	S	22	66	62

a) Means of 4 replications with estimations based on 100 pods per genotype in each replication.
b) Averaged from maximum disease incidence values in 3 yrs
c) Based on 3 yrs classification from maximum DI averages.

Table 6. Viability of sclerotia of _Sclerotinia minor_ formed on pods and stems of susceptible peanut genotypes grown in field plots for 3 years.

	Percent Viability [a]								
	1986			1987			1988		
	Source			Source			Source		
Genotype	Pods	Stems	P [b]	Pods	Stems	P	Pods	Stems	P
Tamnut 74	40	58	.05	44	82	.01	41	68	.01
OK FH-15	32	53	.05	37	74	.01	45	70	.01
OK FH-13	42	57	ns	27	62	.01	39	60	.01
Florunner	60	74	ns	57	76	.05	45	81	.01
Okrun	37	39	ns	42	70	.01	34	62	.01
TX 835841	13	42	.01	42	72	.01	37	76	.01
TX 833841	39	31	ns	33	65	.01	55	82	.01
TX 771174	37	29	ns	40	62	.01	39	64	.01
SN 55-437	32	48	ns	28	74	.01	35	75	.01
TP 107-3-8	43	62	.05	30	68	.01	44	74	.01
Probability [c]	.01	.01		.01	ns		.05	.01	

a) One hundred sclerotia were plated in each treatment in 10 replicated plates of 10 sclerotia in each. Sclerotia were germinated on potato dextrose agar in darkness for 3-5 days

b) Probability within genotypes

c) Probability between genotypes

Fig 1. Effect of *Sclerotinia minor* on pod yield of peanut genotypes grown in *Sclerotinia minor*-infested field plots in 1986.

- Genotype A (resistant genotype) All plants were healthy, thus no infected plants to compare pod yield of healthy plants with.

- When healthy and infected plants were compared, (Genotypes B-F), there was a significant difference (P = 0.05, or P = 0.01) between pod yield of infected and healthy plants

- Genotypes G-J (very susceptible genotypes) All plants were infected, thus no healthy plants to compare pod yield of infected plants with.

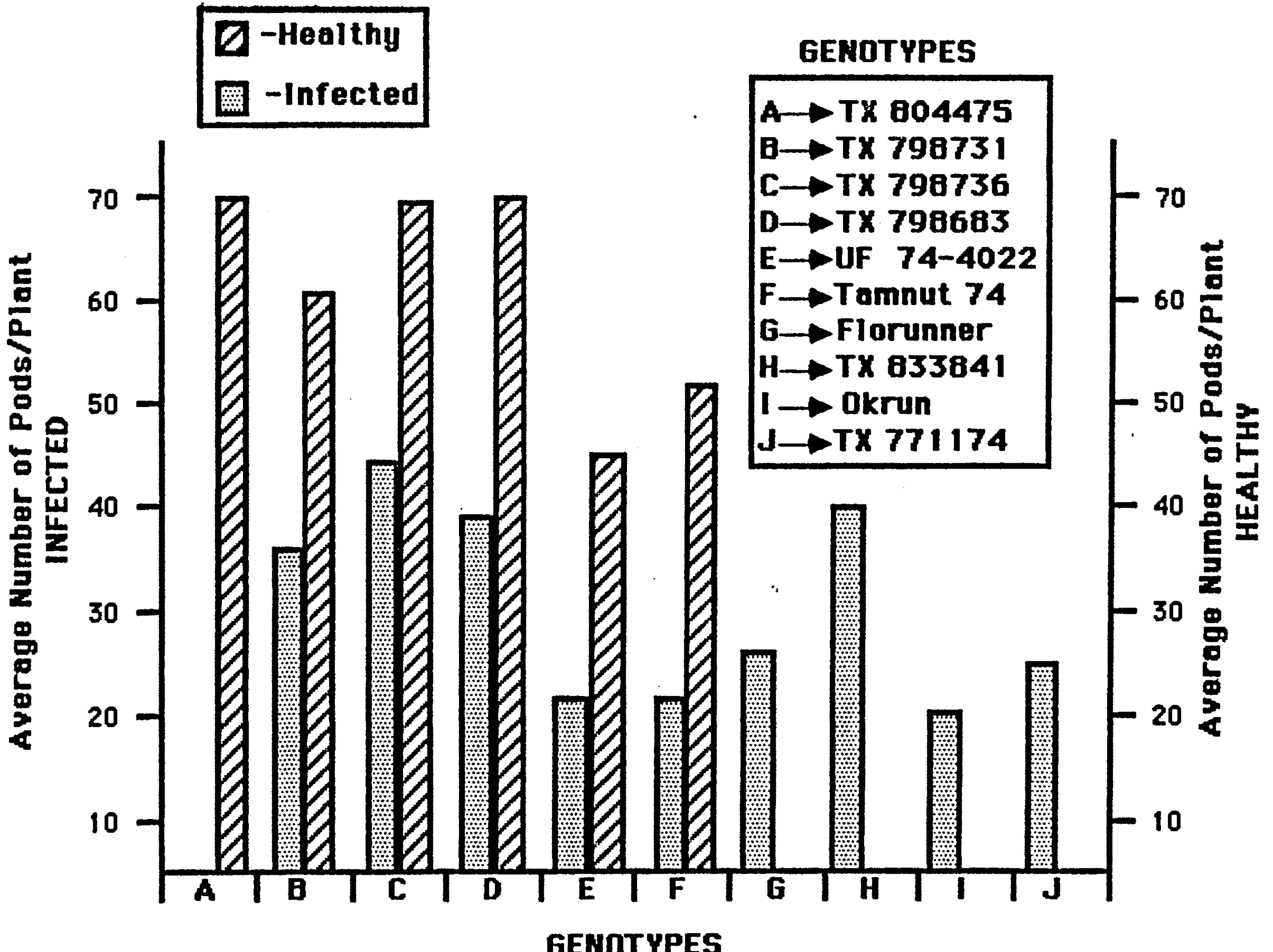
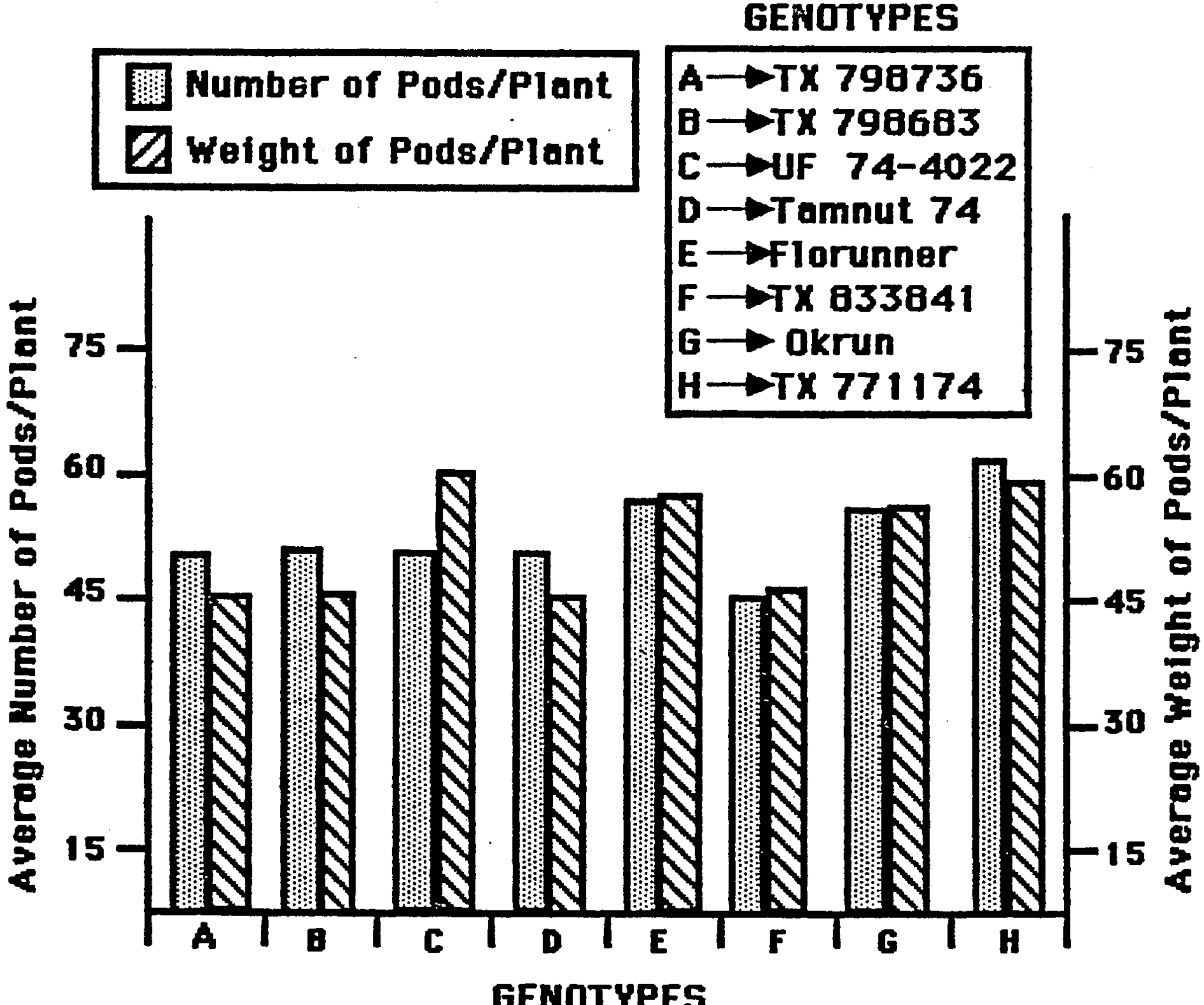

-Healthy
-Infected
GENOTYPES
A→TX 804475
B→TX 798731
C→TX 798736
D→TX 798683
E→UF 74-4022
F→Tamnut 74
G→Florunner
H→TX 833841
I→Okrun
J→TX 771174
Average Number of Pods/Plant
INFECTED
Average Number of Pods/Plant
HEALTHY
70
60
50
40
30
20
10
A
B
C
D
E
F
G
H
I
J
GENOTYPES

Fig 2. Comparison of pod number and pod dry weight among susceptible peanut genotypes grown in a *Sclerotinia minor* infested field plot.

- In all genotypes, there was no significant difference between pod weight (g) and pod number



GENOTYPES
Number of Pods/Plant
Weight of Pods/Plant
A → TX 798736
B → TX 798683
C → UF 74-4022
D → Tamnut 74
E → Florunner
F → TX 833841
G → Okrun
H → TX 771174
Average Number of Pods/Plant
Average Weight of Pods/Plant
75
60
45
30
15
A
B
C
D
E
F
G
H
GENOTYPES

Fig 3. Total amount of sclerotia of *Sclerotinia minor* produced on/in pods of peanut genotypes grown in *S. minor*-infested field plots in 1986 and 1987.

- Sclerotia were estimated from surface and from inside peanut pods. Two hundred and forty pods randomly selected from susceptible peanut genotypes were examined for sclerotial production in 4 row plot replications of 60 pods in each.

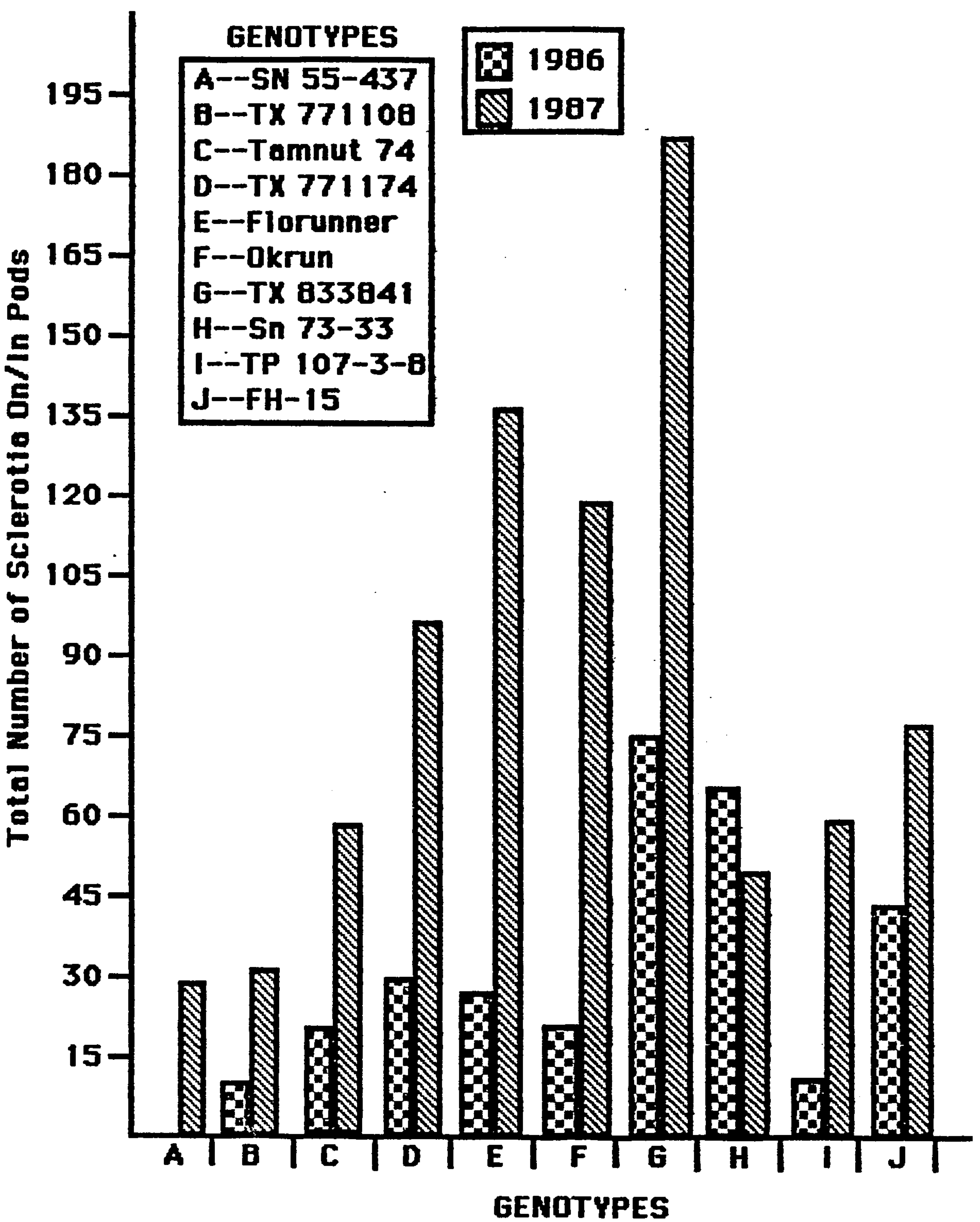
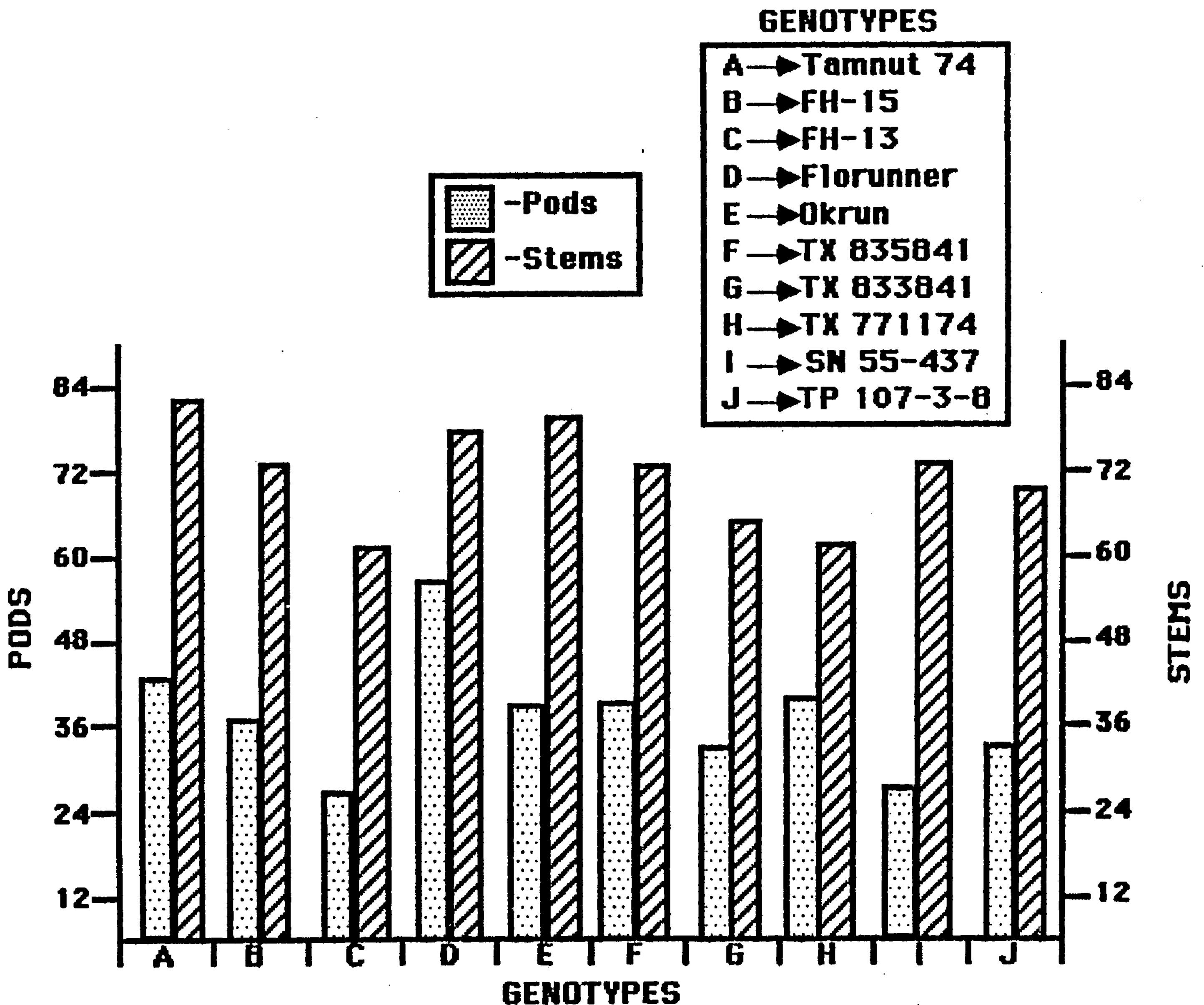

GENOTYPES
A--SN 55-437
B--TX 771108
C--Tamnut 74
D--TX 771174
E--Florunner
F--Okrun
G--TX 833841
H--Sn 73-33
I--TP 107-3-8
J--FH-15
1986
1987
Total Number of Sclerotia On/In Pods
195
180
165
150
135
120
105
90
75
60
45
30
15
A
B
C
D
E
F
G
H
I
J
GENOTYPES

Fig 4. Viability of sclerotia of *Sclerotinia minor* formed on pods and stems of susceptible peanut genotypes grown in *S. minor*-infested field plots

- Sclerotial viability was determined by plating sclerotia on potato dextrose agar and incubating plates in darkness at 25 C for 3-5 days. One hundred sclerotia were plated in each treatment in 10 replicated plates of 10 sclerotia in each.


GENOTYPES
A→Tamnut 74
B→FH-15
C→FH-13
D→Florunner
E→Okrun
F→TX 835841
G→TX 833841
H→TX 771174
I→SN 55-437
J→TP 107-3-8
-Pods
-Stems
84
72
60
48
36
24
12
PODS
STEMS
A
B
C
D
E
F
G
H
I
J
GENOTYPES

CHAPTER V

TRANSMISSION OF SCLEROTINIA BLIGHT OF PEANUT FROM INFECTED SEED

ABSTRACT

Four *Sclerotinia minor*-susceptible peanut genotypes were among 19 genotypes grown in infested field plots in 1986 and 1987 at Stillwater, OK. Disease incidence (DI) values of 85-95 % were reported for all genotypes in both years. *S.minor* was recovered from an average of 12.3, 9.4, 9.7, and 6.8% of seed from cv. Florunner, TX 833841, cv. Okrun, and TX 771174, respectively. Two hundred seeds from each genotype were planted, two seeds/pot (10.5 cm diameter), in a steam pasteurized mixture of soil, peat, and sand (1:2:2; v/v). Pots were placed closely on a greenhouse bench to obtain a thick canopy. Plants were watered daily and fertilized bi-monthly with 0.2% NH_4NO_3 from time of planting. Temperature and relative humidity were monitored by a recording hygrothermograph. Typical Sclerotinia symptoms, appeared on plants about 60 days after planting. DI was recorded at five times, with highest DI values of 0.0, 1.7, 3.5, and 3.2 % recorded for TX 833841, TX 771174, cv. Okrun, and cv. Florunner, respectively.

INTRODUCTION

Sclerotinia blight of peanut caused by the soilborne fungus, *Sclerotinia minor* was first observed in Virginia in 1971, in North Carolina in 1972 (12) and in Oklahoma in 1972 (19). The disease was also reported in Texas in 1981 and in Louisiana in 1982 (18). In less than two decades Sclerotinia blight has become the most important disease of peanut in Virginia and a major disease in Oklahoma.

Species of *Sclerotinia* become established and are spread from field to field, and from one geographical area to another, by several means. Windblown ascospores can be a major means of field-to field spread (1). *Sclerotinia* spp. also may be disseminated from field to field in soil adhering to seedlings, farm equipment, animals or man (6, 16) in the form of sclerotia or as mycelium in infected host tissue. On farms where diseased plant tissue is used as cattle feed or bedding, the spreading of manure on fields has been shown to be a likely means of introducing the pathogen to uncontaminated fields (6). In this connection, Brown (4) showed that less than 2% of the sclerotia of *S. sclerotiorum* fed to sheep passed through the digestive tract in a viable condition. Melouk et al (9) also showed that viable sclerotia passing through the digestive tract of a ruminant can be an important source for spread of the pathogen from infested

areas to clean areas within a field, or from infested fields to clean fields. Thus sheep, cattle and possibly other animals, fed diseased plant debris and turned out to pasture, could spread the pathogen to *Sclerotinia*-free fields. Irrigation also has been shown to be involved in the spread of *Sclerotinia* spp. from field to field (17).

Probably the greatest potential for long distance dissemination of *Sclerotinia* spp is either by seed infected with mycelia or by seed contaminated with sclerotia (2). The host range of the genus *Sclerotinia* is extensive, and Sclerotinia-infected or infested seed has been reported for sunflower (22), cabbage (10), cauliflower (10), clover (6), beans (16), and peanuts (12,21).

Wadsworth and Melouk (21) reported on the potential for transmission and spread of *S. minor* by infected peanut seed and debris. They compared three methods of harvesting and handling of peanut seed for seed infection and debris contamination by *S. minor*, and showed that seed processed by hand and by hand and machine showed infection levels of 25.4 and 8.9 %, respectively, while seeds processed by machine showed 1.4% infection. They speculated that seed infected by *S. minor* or seed contaminated with sclerotia had the potential to result in long distance dissemination of *S. minor*.

The transmission of *S. minor* from infected seed has not yet been demonstrated either in the greenhouse or in the

field. The objectives of this study were therefore, to determine the level of seed infection from *S. minor*-susceptible peanut genotypes planted in infested field plots, and to determine seed transmission of the disease in the greenhouse by planting contaminated or infected seed in a disease-free environment.

MATERIALS AND METHODS

Seed Infection

Four *S. minor*-susceptible peanut genotypes (Florunner, Okrun, TX 833841 and TX 771174) were among 19 genotypes planted in *S. minor*-infested field plots in 1986 and 1987. The plots, infested with *S. minor* in 1981 had an inoculum density of 3-5 sclerotia per 100 g of soil. Plots were arranged in a randomized complete block design with four replications. Blocks consisted of 19 rows, each 4.55 m long and 0.91 m apart. Blocks were separated by 1.5 m alleys.

At the end of the growing season, (about 150 days after planting), plants were hand-dug and separated into diseased and healthy groups. Plants were sacked in burlap sacs and taken to the greenhouse where they were stored on benches for about 60 days to dry. Pods from infected plants were used to determine % seed infection by *S. minor*. Pods from all genotypes were hand-shelled and seed was

collected and plated on potato dextrose agar containing 100 mg/L of streptomycin sulfate (SPDA),to determine % seed infection. Two hundred and fifty seeds from each genotype were placed on a # 20 mesh screen and gently washed under running tap water. Seeds in screen were submerged in a container with 0.5% sodium hypochlorite (NaClO) and surface sterilized for about 2 minutes. Paper towels were used to remove excess moisture. Using sterile forceps, five seeds were plated on potato dextrose agar in each petri plate (9 cm). Plates were incubated in darkness for 3-7 days at 25 2C and then examined for S. minor growth from seeds. Numbers of seeds with mycelia of S. minor growing out from them were recorded. A total of 1000 seeds were plated for each genotype in 4 replications. Percent seed infection was calculated by the formula :

$$\% \text{ seed infection} = \frac{X}{Y} \times 100$$

where X = # of seeds from which S. minor grew,
and Y = # of seeds plated

Seed Transmission

Two hundred and sixty seeds, randomly selected from the infected seed lot of each of the genotypes, were germinated in an incubator at 28 C in darkness for 24 hrs. Two hundred germinating seeds were selected and planted, 2

seeds per pot in 10.5 cm dia pots, in a steam pasteurized mixture of soil, peat, and sand (1:2:2; v/v). Pots were placed in close proximity to each other on greenhouse benches to obtain a thick canopy necessary to retain moisture and provide ideal humid conditions necessary for Sclerotinia blight development (13). Plants were watered daily and fertilized bi-monthly with 0.2 % NH_4NO_3 throughout the growth period. Temperature and relative humidity were monitored by a recording hygrothermograph. Plants were examined on a daily basis for any changes. Wilting or dying plants when observed were immediately sampled to determine cause of death. Stem and crown segments from such plants were surface sterilized with 0.5% sodium hypochlorite for 3 min, plated on SPDA and incubated in darkness at 26 C for 3-5 days to allow for growth of any associated microorganisms. Other plants in greenhouse were monitored closely for typical Sclerotinia blight symptoms. Plants with symptoms were flagged and counted to determine the number of infected plants. Disease incidence was recorded at intervals for each of the plantings until maximum DI was obtained when no further infections were detected.

Recovery of *S. minor* from greenhouse plants

At maturity, pods were harvested from infected plants. Pods were air dried in paper bags on greenhouse benches at

26 2C, and then hand-shelled to collect seeds. All seeds collected were surface sterilized with 0.5% sodium hypochlorite as previously described, and plated on SPDA to determine % seed infection. Pods from all other noninfected plants were also harvested and plated on SPDA to determine seed infection, even though there were no above-ground symptoms observed on the plants.

RESULTS

In 1986 and 1987, average maximum disease incidence values of 95, 93, 92, and 85 %, respectively, were recorded for cv Florunner, Okrun, TX 833841, and TX 771174 (Table 1). Seed samples from these entries and two resistant entries TX 804475, and TX 798736, exhibited 0 to 12.3 % infection with _S. minor_ when plated on SPDA (Table 2). Florunner, (highly susceptible) had the highest level of infected seed (12.3%), while TX 804475 and TX 798736 considered resistant to _S. minor_ from three years of evaluation in field tests (3), had the lowest level (0%).

Seeds harvested from the 1986 field trial had infection levels with _S. minor_ of 12.3, 11.2, 9.6, and 6.4 for cv Florunner, TX 833841, cv Okrun, and TX 771174, respectively (Table 2), while 12.2, 9.8, 7.6, and 7.2 % seed infection was respectively recorded for cv. Florunner, Okrun, TX 833841, and 771174 from 1987 field seed. Other fungi

commonly associated with seeds included *Fusarium* spp., *Trichoderma* spp., and *Aspergillus* spp.

Typical Sclerotinia symptoms of stem wilting stem necrosis and white fluffy mycelial signs under humid conditions (20) appeared on the 1986 plants in mid September, about 60 days after planting. Symptoms observed on the 1987 seed lot plants in the second test included wilting and subsequent death of plants, appearing in early February, about 50 days after planting. Sclerotia started forming on the surface of infected stems shortly after signs of the pathogen were observed on the stems.

Percent seed transmission of Sclerotinia blight as determined by maximum disease incidence was 4.5, 3.5, 1.0 and 0% for cvs. Florunner, Okrun, TX 771174, and TX 833841, respectively, from the 1986 field seeds in the first test, and 3.5, 2.5, 2.0, and 0 for cvs Okrun, TX 771174, Florunner, and TX 833841, respectively, from the 1987 seed lot in the second test (Table 3). In both tests, 5 disease incidence readings were taken from first appearance of typical symptoms until no further infection was detected or was obviously from plant contacts and cross infection within the pots. *S. minor* was recovered from seeds of infected plants of all genotypes that showed some degree of seed transmission (Table 4). The pathogen was also recovered from some seed of noninfected plants among genotypes that showed capability of seed transmission. Percent *S. minor* recovered

was significantly higher in seeds from infected plants than from the noninfected plants (Table 4). S. minor was not recovered from any seeds of the genotype TX 833841. As before, the same saprophtes that grew out from previously plated seeds were present in plated seeds of infected and noninfected plants.

When the transmission efficiency of Sclerotinia blight was calculated for all the genotypes evaluated, Florunner and Okrun showed very high values of transmission efficiency in both tests. The genotype TX 771174 also had a high % transmission efficiency (Table 5). Despite a high % seed infection for the genotype TX 833841, it had zero % transmission efficiency, since no seed transmission of S. minor was obtained in the greenhouse from both trials (Figs 1 and 2).

DISCUSSION

The life cycle of a plant pathogen can be viewed as consisting of four basic phases : survival, transmission, infection, and disease development. Seeds can be involved in each phase. They can act as a means of survival of a pathogen from one growing season to the next. They can provide a means of transmission if a pathogen associated with the planted seed can move to the new crop. The infection and disease development phases of the life cycle

also are important for diseases in which seeds produced in the field are infected by pathogens that can reduce yield or seed quality.

The annotated list of seedborne diseases published in 1979 (15) records almost 1500 seedborne microorganisms on about 600 genera of agricultural, horticultural and tree crops. From the plant quarantine standpoint, these figures do not exaggerate the magnitude of the problems involved in controlling the movement of seedborne pathogens into areas where they have previously been recorded.

Diseased seeds can sometimes be detected by visual examination of dry seed, but this method of assessing seedborne inoculum rarely is sensitive enough to be of practical value (8). Most tests involve plating seeds on culture media. Serological tests for detection of seedborne bacteria and viruses also have been developed (5, 7).

Significant contributions have been made in developing laboratory testing procedures for many seedborne pathogens (11). Unfortunately, for many pathogens the values obtained in laboratory tests cannot be related to the risk of disease development once the seed is planted. The test that provides the highest count for a pathogen on media may not be the most useful in predicting field disease. In our study, even-though TX 833841 showed a high seed infection % of 11.2, in the first test and 7.6 in the second, no seed transmission of _S. minor_ was recorded on this genotype in both greenhouse

tests. Thus we could not simply deduce potential for seed transmission based on media counts of seed infection alone. The transmission efficiency of each genotype could give us an idea of what to expect when we evaluate contaminated seed for seed transmission. From results of both tests conducted, different genotypes had different transmission efficiencies. We could thus conclude from these results that seed transmission of S. minor may be genotype dependent. Unless other epidemiological studies are made to relate laboratory seed infection tests to the actual risk of subsequent field disease, these tests will continue to be of little practical value.

The temperature ranges in the greenhouse as recorded on the hygrothermograph was 26-32C in the day and 22-26C at night. This range is quite within the desired range necessary for S. minor to infect when the inoculum is present (13). Relative humidity averaged 75 to 100%, a desired range for disease development.

In the area of dissemination of S. minor, we can speculate a great deal, yet, we do not know for sure how new fields brought into cultivation become infested with the pathogen. The possibility exists that long distance spread of S. minor could result from infected seed. This study clearly demonstrates that possibility under greenhouse conditions maintained to favor disease development. Perhaps the findings of this research will throw more light and

challenge us to focus more attention on studying the actual role of seed transmission in the long distance spread of the pathogen. Infected seed could be responsible for introducing the pathogen into new areas. Discovery of a pathogen, however, need not be from recent seed introduction. Sclerotinia spp. have a wide host range (14), and S. minor may have been present in low incidence until changes in the environment or farming practices permitted its development (21)

It is posible that fields in North Carolina, Oklahoma and Texas where peanuts were grown for years with no evidence of Sclerotinia blight now sustain severe loses due to the disease because of contaminated seed brought in from severely infected fields. It would be important to carefully consider sources of seed before planting in disease-free or new peanut fields. It may also be important to consider crops with which we rotate peanuts as potential Sclerotinia hosts may introduce the pathogen into clean fields.

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Table 1: Maximum disease incidence (DI) and disease progress (DP) for peanut genotypes in field plots in 1986 and 1987.

	1986		1987	
Genotype	DI (%) [a]	DP (r) [b]	DI	DP
Florunner	98 a	0.138 a	92 a	0.080 a
TX 833841	98 a	0.156 a	86 a	0.067 a
OKRUN	100 a	0.159 a	86 a	0.061 a
TX 771174	100 a	0.167 a	69 a	0.036 ab
TX 798736	16 b	0.006 b	5 b	0.002 b
TX 804475	0 b	0.0 b	0 b	0.0 b

a) Sclerotinia blight incidence was recorded on September 24 and October 11 for 1986 and 1987, respectively, and percentage was obtained by dividing the number of infected plants by the total number of plants in row and multiplied by 100.

b) Values of DP were obtained by linearly regressing $\log_e \frac{1}{1-X_t}$ over time, where X equal to the proportion of infected plants in each row.

Means within the same column followed by the same letter are not significant at P = 0.05, according to Duncan's multiple range test.

Table 2: Recovery of *Sclerotinia minor* from peanut seed grown in *S. minor* infested field plots in 1986 and 1987.

Genotype	% Recovery of *S. monir* [a)] 1986	1987
Florunner	12.3 a	12.2 a
Okrun	9.6 ab	9.8 b
TX 833841	11.2 a	7.6 c
TX 771174	6.4 b	7.2 c
TX 804475	0.0 c	0.0 d
TX 798736	0.0 c	0.0 d

a) Obtained by plating a total of 1000 seeds in 4 replications of 250 seeds in each, on potato dextrose agar containing 100 mg/L of streptomycin sulfate. Positive identification of *S. minor* was made after incubation at 26 C in darkness for 5-7 days.

Means within the same column followed by the same letter are not significant at P = 0.05, according to Duncan's multiple range test.

Table 3: Transmission of Sclerotinia blight from infected peanut seed in greenhouse tests.

Genotype[a]	1986 Seed		1987 Seed	
	# of infected plants[b]	% Tran-mission[c]	# of infected plants	% Trans-mission
Florunner	9 a	4.5 a	5 a	2.5 a
Okrun	7 a	3.5 a	7 a	3.5 a
TX 771174	2 b	1.0 b	4 a	2.0 a
TX 833841	0 b	0 b	0 b	0 b

a) Seeds from genotypes were obtained from field plots in 1986 and 1987.

b) Total of 200 plants in tests representing 4 replications of 50 seeds each. (Each pot-10.5 cm dia, contained 2 plants).

c) Obtained by dividing maximum number of infected plants by the total number of plants in each treatment multiplied by 100.

Means followed by the same letter within columns are not significantly different at P = 0.05 according to Duncan's multiple range test.

Table 4: Recovery of *Sclerotinia minor* from infected and healthy peanut seed in the greenhouse.

Genotype	% Recovery of *S. minor* from seed obtained from [a]	
	Infected plants	Noninfected plants [b)]
Okrun	27.5 a	1.5 a
Florunner	17.7 ab	1.0 a
TX 771174	11.0 b	0.0 b
TX 833841	0.0 c	0.0 b

a) Obtained by plating all seeds collected from plants of each genotype in 4 replications, on potato dextrose agar containing 100 mg/L of streptomycin sulfate

b) Plants were grown in the same pot with infected plants.

Means followed by the same letter within columns are not significantly different at P = 0.05 according to Duncan's multiple range test.

Table 5: Transmission efficiency of Sclerotinia blight by infected peanut seed in greenhouse tests

Genotype	% seed Infection [a]	% seed Transmission [b]	Transmission Efficiency
1986 Seed			
Florunner	12.3 a	4.5 a	36.6 a
Okrun	9.6 ab	3.5 a	36.5 a
TX 771174	6.4 b	1.0 b	15.6 b
TX 833841	11.2 a	0 b	0 c
1987 Seed			
Florunner	12.2 a	2.5 a	20.5 ab
Okrun	9.8 ab	3.5 a	35.7 a
TX 771174	7.2 b	2.0 a	27.8 a
TX 833841	7.6 b	0 b	0 c

a) Obtained by plating 1000 seeds in 4 replications of 250 seeds in each.

b) Total of 200 plants in tests representing 4 replications of 50 plants in each.

c) Transmission efficiency was calculated as follows:

$$\frac{\%\ \text{seed transmission}}{\%\ \text{seed infection}} \times 100$$

Means followed by the same letter within columns are not significantly different at P=.05, according to Duncan's multiple range test.

Fig 1. Percent infection and transmission of *Sclerotinia minor* from peanut seed grown in *Sclerotinia minor*-infested field plots in 1986.

- Seed infection was determined by plating surface sterilized seeds on potato dextrose agar and incubating plates in darkness at 25 C for 3-7 days. Positive identification of *S. minor* was made from presence of mycelia growing out of seed after 3 days and/or formation of sclerotia from mycelia after 7 days

- There was significant difference (P = 0.05) between % seed infection and % seed transmission for all 4 genotypes.

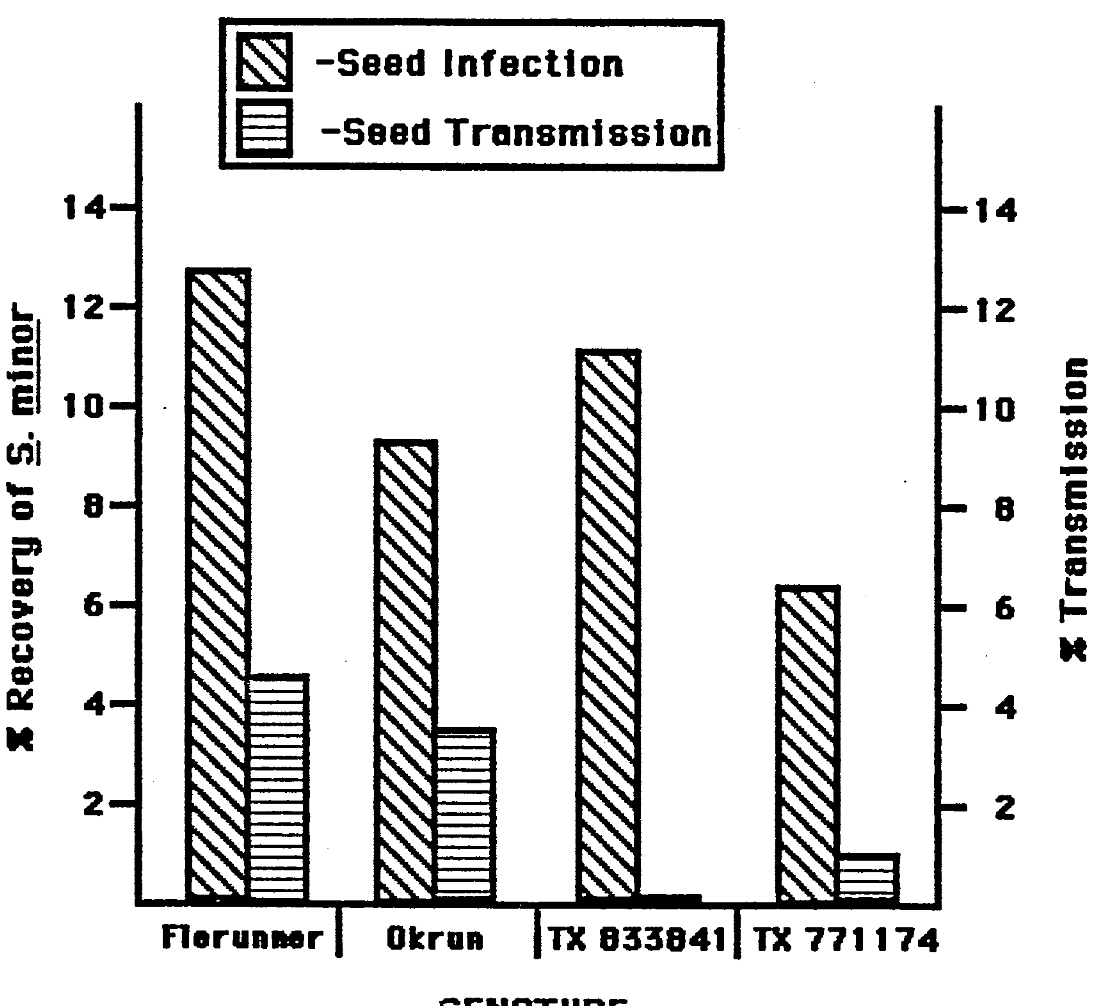
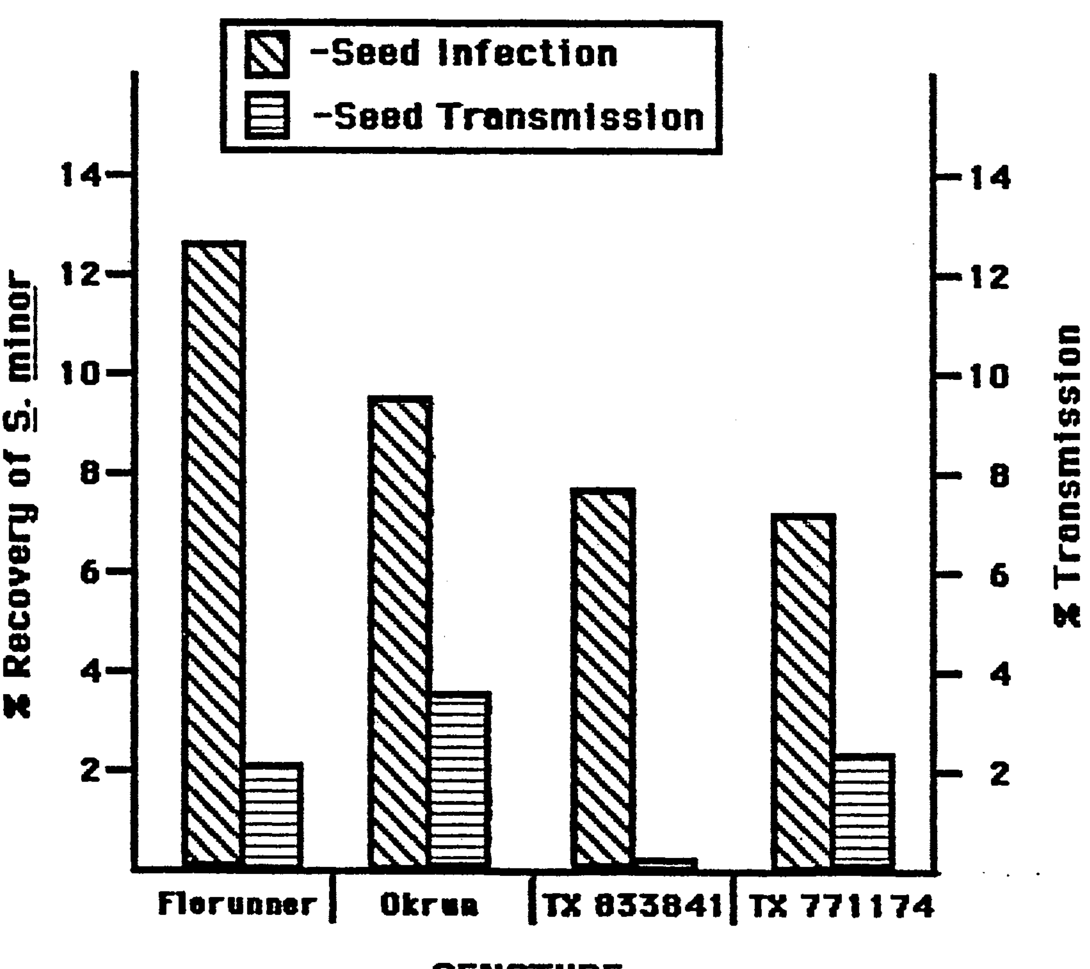

-Seed Infection
-Seed Transmission
% Recovery of S. minor
% Transmission
14
12
10
8
6
4
2
Florunner
Okrun
TX 833841
TX 771174
GENOTYPE

Fig 2. Percent infection and transmission of *Sclerotinia minor* from peanut seed grown in *Sclerotinia minor*-infested field plots in 1987.

- Seed infection was determined by plating peanut seed on potato dextrose agar and incubating in darkness at 25 C for 3-7 days. Positive identification of *S. minor* was made from growth of mycelia from seed or formation of sclerotia after incubation.

- There was a significant difference (P = 0.05) between % seed infection and % seed transmission.


-Seed Infection
-Seed Transmission
14
12
10
8
6
4
2
% Recovery of S. minor
% Transmission
Florunner
Okrun
TX 833841
TX 771174
GENOTYPE

APPENDICES

APPENDIX A

INFECTION PROCESS OF *SCLEROTINIA MINOR* ON A SUSCEPTIBLE AND A RESISTANT PEANUT CULTIVAR

APPENDIX A

STUDIES ON THE INFECTION PROCESS SCLEROTINIA MINOR ON THE STEMS OF A SUSCEPTIBLE AND A RESISTANT PEANUT CULTIVAR

The objective of this study was to follow the infection process of *S. minor* on stems of a susceptible and a resistant peanut cultivar from the time mycelia is placed in contact with the stem until establishment in and destruction of the tissues.

Inoculum of *S. minor* was prepared by germinating sclerotia produced on oat seed on SPDA, and transfering actively growing mycelia for sub culture onto other plates. Stem bases of peanut cultivars Tamnut 74 (susceptible) and Toalson (resistant) were inoculated with *S. minor* by placing mycelial plugs (4mm dia) from the leading edge of a 2 day old culture, in contact with the stems in petri dishes lined with damp whatman # 1 filter paper. Inoculated samples were collected at 3, 6, 9, 12, 24, 48 and 72 hrs following inoculation.

Infection sites of stems were trimmed into square pieces (5 sq mm) and fixed in 2 % gluteraldehyde in sodium cacodylate buffer for 24 hrs. Samples were post-fixed with 2 % Osmium tetraoxide in sodium cacodylate buffer for 2 hrs, then dehydrated in an alcohol series of 60, 70, 80, 90 and

100 % alcohol for 6 hrs in each concentration. After critical-point drying in a 3-point critical dryer, specimens were coated with gold palladium in a Hummer II Coater and viewed with a scanning electron microscope, Joel (JSM-35).

The following observations were recorded on specimens of each of the cultivars.

TAMNUT 74

- Zero to 3 hrs after inoculation.

  No mycelia was detected on host tissue. Pathogen was still establishing on agar mycelial plug.
- 3 to 6 hrs.

  Mycelia proliferated beyond plug and stuck to host tissue. Infection structures (cushions) were initiated in clusters following profuse proliferation of mycelia. Following establishment of cushions, mycelia strands grouped to push host tissue apart in an effort to start penetration.
- 6 to 12 hrs. (Pre-penetration)

  Tip ends of some cushion strands swelled up, enlarged, and adhered tightly to host surface. Penetration of host tissue at "weak" points using concentrated efforts of cushions and the bulging mycelial tips began.
- After 24 hrs (Post-penetration)

  Physical changes started to show on inoculated stems. Lesions were initiated by a water-soaked appearance at point of infection, that gradually turned dark-brown as they enlarged.

- Folial Infection

Eventhough not a folial pathogen, mycelia of S. minor successfully penetrated peanut leaves through the stomata, without the formation of an appressorial swelling. This demonstrates the strong saprophytic ability of S. minor.

TOALSON

- 3 to 6 hrs after inoculation

No mycelial growth was observed on the surface of host tissue. Establishment on agar plug was observed.

- 6 hrs

Mycelia, rather scanty, started to form on plant tissue after proliferation beyond the agar plug.

- 6 to 12 hrs.

Extensive proliferation of mycelia. Instead of cushions being formed mycelia intertwined to form a rope-like structure.

- 12 to 24 hrs.

Cushions began to form at the end of mycelial strands from rope-like structures. Cushions were few in numbers, had greater numbers of mycelial strand constituents, and strands appeared to vary in age. Cushions were fairly complicated in structure. Apparently not all mycelial strands successfully penetrated host tissue. Failure to penetrate by a single swollen mycelial strand was observed.

- 48 hrs

Visible symptoms of infection started to show up. Water-

soaked lesions were less extensive and rather restricted to the site of infection. Tissue eventually started to disintergrate.

From the above observations in the infection process of _S. minor_ on the two separate cultivars, the following differences could be noted.

1. Mycelia started to proliferate and establish on Tamnut 3 to 6 hrs after inoculation. Mycelial growth was observed on Toalson only after 6 hrs following inoculation.
2. There was more mycelial growth and spread through intertwining rope-like structures on Toalson before cushion formation. On Tamnut, cushions formed soon after proliferation, with no massing or intertwining of mycelia.
3. Number of cushions formed on Tamnut were more than on Toalson, but the few on Toalson were made up of more strands and were more complicated in structure with varied shapes and sizes.
4. Unsuccessful penetration by a single mycelial strand was observed on Toalson but not on Tamnut. Apparently, all infection structures formed on Tamnut succeeded to penetrate. Some mycelial strands penetrated Toalson tissue without first forming swollen tips or typical cushions.
5. Following infection and penetration of host tissue, visible symptoms, upon close examination, were observed on Tamnut after 24 hrs following inoculation, but only apparent on Toalson after 48 hrs.

The study demonstrates the following :

- The formation of a specialized infection structure by a pathogen is dependent on the nature of the host surface under attack.
- The number, size and complexity of infection structure formed is also determined by the nature of the host tissue.
- Resistance of a host to a pathogen may be due to the failure of the pathogen to penetrate or failure to establish in the host tissue following penetration.

APPENDIX B

DISEASE INCIDENCE READINGS AND CLASSIFICATION OF PEANUT GENOTYPES BASED ON MAXIMUM DI IN 3 YRS OF FIELD STYDIES

APPENDIX B

Table 1. Percent of Peanut Plants Infected with Sclerotinia blight on different observation dates in 1986

	Infected plants in plots (%)					
Genotype	8-22	8-30	9-6	9-14	9-21	9-24
TX 804475	0.00	0.00	0.00	0.00	0.00	0.00
Toalson	0.00	0.00	0.00	0.00	0.00	0.00
Sn 55-437	2.56	7.69	12.82	15.38	17.94	20.51
TX 798683	0.00	0.00	5.00	6.66	15.38	16.66
UF 73-4022	0.00	0.00	5.17	13.79	22.41	24.13
Florunner	13.50	40.00	86.66	98.33	98.33	98.33
TX 798731	0.00	0.00	3.33	8.33	8.33	10.00
TX 771108	0.00	0.00	23.33	41.66	48.33	48.33
TX 835841	5.00	40.00	76.66	88.33	100.00	100.00
Tamnut 74	0.00	3.33	21.66	33.33	41.66	41.66
TX 798736	0.00	0.00	1.81	5.45	16.36	16.36
TP 107-3-8	18.42	31.58	60.52	68.42	81.57	81.57
Sn 73-33	6.06	15.15	33.33	48.48	63.63	75.75
TX 833829	2.00	8.00	20.00	34.00	40.00	40.00
TX 833841	8.33	40.00	75.00	90.00	96.66	98.33
OK FH-13	10.00	46.66	78.33	100.00	100.00	100.00
Okrun	8.47	49.15	93.22	100.00	100.00	100.00
OK FH-15	16.95	45.76	83.05	94.91	100.00	100.00
TX 771174	10.17	38.98	77.96	93.22	100.00	100.00

Table 2. Classification of peanut genotypes based on maximum disease incidence in the field in 1986

Classification	Genotype	% Disease Incidence
Very Resistant	Toalson	0.00
	TX 804475	0.00
Resistant	TX 798731	10.00
	TX 798736	16.36
	TX 798683	16.66
Low Resistance	Sn 55-437	20.51
	UF 73-4022	24.13
Susceptible	TX 833829	40.00
	Tamnut 74	41.66
	TX 771108	48.33
Very Susceptible	Sn 73-33	75.75
	TP 107-3-8	81.57
	TX 833841	98.33
	Florunner	98.33
	TX 835841	100.00
	TX 771174	100.00
	OK-FH 13	100.00
	OKRUN	100.00
	OK-FH 15	100.00

Table 3: Percent of Peanut Plants Infected With Sclerotinia blight on different observation dates in in 1987.

	Infected plants in plots (%)					
Genotype	9-6	9-13	9-17	9-22	9-29	10-11
TX 771108	1.72	8.62	8.62	8.62	15.51	24.13
TX 833841	13.79	51.72	58.62	70.68	81.03	86.20
Toalson	0.00	0.00	0.00	0.00	0.00	0.00
Florunner	30.00	60.00	70.00	81.66	88.33	91.67
TX 804475	0.00	0.00	0.00	0.00	0.00	0.00
TX 771174	19.23	38.46	44.23	55.76	63.46	69.23
OK FH-15	31.66	61.67	65.00	78.33	85.00	91.67
UF 73-4022	0.00	0.00	0.00	3.70	7.85	9.25
TP 107-3-8	22.41	56.89	67.24	75.86	82.75	94.82
Okrun	21.81	38.18	45.45	60.00	80.00	85.45
TX 798731	0.00	0.00	0.00	0.00	0.00	1.67
Sn 55-437	0.00	20.00	23.33	31.67	36.67	43.33
Tamnut 74	0.00	5.36	8.92	19.64	21.42	32.14
TX 833829	15.25	23.72	30.50	47.45	54.23	55.93
OK FH-13	20.37	48.14	53.70	61.11	70.37	75.92
TX 798736	0.00	0.00	1.69	1.69	5.08	5.08
TX 835841	8.47	20.33	23.72	40.67	52.54	61.01
Sn 73-33	18.96	48.27	48.27	63.79	68.96	74.13
TX 798683	0.00	0.00	0.00	3.33	5.00	5.00

Table 4. Classification of peanut genotypes based on maximum disease incidence in the field in 1987.

Classification	Genotype	% Disease Incidence
Very Resistant	Toalson	0.00
	TX 804475	0.00
Resistant	TX 798731	1.67
	TX 798683	5.00
	TX 798736	5.08
	UF 73-4022	9.25
Low Resistance	TX 771108	24.13
	Tamnut 74	32.14
Susceptible	Sn 55-437	43.33
	TX 833829	55.93
	TX 835841	61.01
Very Susceptible	TX 771174	69.23
	Sn 73-33	74.13
	OK FH-13	75.92
	Okrun	85.45
	TX 833841	86.20
	Florunner	91.67
	OK FH-15	91.67
	TP 107-3-8	94.82

Table 5. Percent of Peanut Plants infected with Sclerotinia blight on different observation dates in 1988.

	Infected Plants in Plots (%)					
Genotype	9-5	9-8	9-12	9-15	9-19	9-22
TX 804475	0.0	0.0	0.0	0.0	3.3	5.0
TX 798683	0.0	0.0	0.0	1.8	5.3	5.3
TX 798731	0.0	0.0	0.0	5.0	6.6	8.3
UF 73-4022	0.0	1.7	5.0	6.7	8.3	8.3
Toalson	0.0	0.0	3.3	6.7	11.7	11.7
TX 798736	0.0	0.0	6.9	6.9	12.1	13.8
Sn 55-437	0.0	0.0	10.0	11.7	16.7	20.0
TX 771108	7.0	8.8	19.3	19.3	28.1	38.6
TX 833829	3.3	8.3	20.0	21.7	33.3	41.7
Tamnut 74	0.0	11.7	25.0	28.3	28.3	46.7
Sn 73-33	8.9	24.4	40.0	42.2	51.1	51.1
TX 833841	0.0	15.3	33.9	42.4	50.8	52.5
TX 835841	6.6	21.7	46.7	56.7	60.0	68.3
TX 771174	10.0	16.7	35.0	41.7	51.7	68.3
OK FH-15	15.8	33.3	54.4	61.4	75.4	80.7
OK FH-13	15.3	33.9	45.8	62.7	71.2	81.4
TP107-3-8	15.0	33.3	60.0	66.7	78.3	86.7
Florunner	9.8	27.5	51.0	68.2	80.4	90.2
Okrun	10.9	27.3	52.7	69.1	80.0	94.5

Table 6. Classification of peanut genotypes based on maximum disease incidence in field in 1988.

Classification	Genotype	% Disease Incidence
Resistant	TX 804475	5.0
	TX 798683	5.3
	TX 798731	8.3
	UF 73-4022	8.3
Low Resistance	Toalson	11.7
	TX 798736	13.8
	Sn 55-437	20.0
Susceptible	TX 771108	38.6
	TX 833829	41.7
	Tamnut 74	46.7
	Sn 73-33	51.1
	TX 833841	52.5
Very Susceptible	TX 835841	68.3
	TX 771174	68.3
	OK-FH 15	80.7
	OK-FH 13	81.4
	TP 107-3-8	86.7
	Florunner	90.2
	Okrun	94.5

APPENDIX C

LESION LENGTHS OF PEANUT SHOOT TIPS INOCULATED WITH *SCLEROTINIA MINOR* USING THE DETACHED SHOOT TECHNIQUE

APPENDIX C

Table 7: Average length (cm) of lesion per shoot tip of peanut genotype in a 7-day period following inoculation with _Sclerotinia minor_.

Test # 1

	Days after inoculation				
Genotype	3	4	5	6	7
TX 798736	.20	.72	1.70	2.10	3.10
TX 804475	.20	.64	1.70	2.15	2.65
TX 798731	.25	.65	1.85	2.55	3.45
TX 798683	.25	.50	1.20	1.60	1.95
UF 73-4022	.40	.97	2.25	3.00	4.20
TX 771174	.45	1.75	3.60	4.80	5.50
TX 771108	.32	1.10	2.60	3.75	5.00
TP 107-3-8	.45	1.55	3.50	4.75	5.95
TP 107-11-4	.30	.80	2.20	3.05	4.00
TX 833829	.50	1.40	3.35	4.35	5.65
TX 835841	.60	.85	2.10	3.20	4.25
TX 833841	.90	1.50	3.50	4.60	5.85
Florunner	.40	1.35	3.25	4.30	5.55

Shoots were inoculated with 4 mm mycelial plugs of a two-day old culture of _Sclerotinia minor_ taken from the leading edge of the plate.

Table 8: Average length (cm) of lesion per shoot tip of peanut genotype in a 7-day period following inoculation with S. minor.

Test # 2

	D A I [1]				
Genotype	3	4	5	6	7
TX 798736	.48	1.60	2.45	3.40	3.80
TX 804475	.25	.55	.80	1.25	1.85
TX 798731	.00	.55	.85	1.40	2.25
TX 798683	.17	.65	1.15	1.90	2.50
UF 73-4022	.28	1.35	1.85	3.40	4.40
TX 771174	.80	1.80	2.80	4.75	5.65
TX 771108	.68	1.00	1.65	2.85	3.65
TP 107-3-8	1.20	2.80	3.85	5.30	6.35
TP 107-11-4	.65	1.65	2.35	4.15	5.20
TX 833829	.30	1.25	1.85	3.55	4.50
TX 835841	.53	.85	1.35	2.45	3.50
TX 833841	.75	1.85	2.40	4.45	6.15

[1] DAI = Days After Inoculation

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