PEANUT CULTIVAR SELECTION FOR THE DEVELOPMENT OF RESISTANCE TO SCLEROTINIA BLIGHT

By

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Chapter I

INTRODUCTION

The cultivated peanut (*Arachis hypogaea* L.) is a self-pollinated allotetraploid (2n=4x=40) believed to have originated in the Northwestern Argentina-Southern Bolivia region of South America (Kochert et al., 1991; Paik-Ro et al., 1992). The cultivated peanut is divided into two subspecies, *hypogaea* and *fastigiata.* The subspecies *Hypogaea* is further subdivided into 'hypogaea', which includes the runner and virginia U.S. market types, and 'hirsuta', which contains the peruvian market type. The subspecies *Fastigiata* is divided into 'fastigiata', containing the valencia market type, and 'vulgaris' or 'spanish' market type (Paik-Po et al., 1992).

There are many constraints to peanut production, including a wide array of insects, diseases, and abiotic stresses. Sclerotinia blight (*Sclerotinia minor* Jagger) has become one of the major limiting factors in peanut production (Melouk & Shokes, 1995). *S. minor* was first reported to infect peanut in Virginia, in 1971. In recent years, the disease has become more severe and spread to North Carolina, Oklahoma, New Mexico, Louisiana, and Texas (Smith, et al., 1991a; Wildman et al., 1992). Yield losses of 10% are not uncommon, however in cases of severe infection losses of up to 50% may occur (Melouk & Shokes, 1995).

S. minor will attack all tissues within the peanut plant. However, stem infections are the most economically important because peg formation originates

from the stems (Chappell et al., 1995). Temperature, relative humidity and soil moisture play a vital role in the infection and colonization of plant tissues by *S. minor. S. minor* is a soil-borne pathogen that causes severe infections during cool, and wet weather. A demonstrated optimum growth range for the pathogen *S. minor* ranges from 15-25° C and a relative humidity approaching saturation (95-100%). High humidity promotes myceliogenic germination of sclerotia of *S. minor* and is positively correlated with disease development. Disease development in the field is low when plants are small and without a dense canopy or complete ground cover. Outbreaks of Sclerotinia blight are most often observed after vines are within 6 inches of touching or after vines lap between rows (Dow et al., 1988; Phipps, 1994). Sclerotinia blight disease development is greatest as the plants reach maturity in September and October, due to cooler night time temperatures and higher relative humidities normally associated with fall climate changes. During this time the plant canopies increase contributing to the maintenance of higher humidity close to the ground (Dow et al., 1988).

Symptoms of Sclerotinia blight first appear at the top of the plant, and include chlorosis and flagging of the infected plant. Examination of the lower canopy in early morning reveals the presence of cottony mycelia on the main stem, lateral branches, and the taproot near the soil line. Within 3-4 days of observing the infection, mycelia will mat and sclerotia 1-3mm in length form on the outside and inside of infected tissues. Sclerotia will infect tissues including the stem and root tissues as well as pods produced on infected plants (Melouk and Shokes, 1995). Lesions caused by the infection of stems and branches are

light tan or straw colored, turning dark brown. Once the lesions begin turning brown, shedding of infected stems, branches, and pegs may eventually cause plant death (Melouk and Shokes, 1995; Akem et al., 1992).

Current Sclerotinia blight management recommendations include: planting resistant cultivars, avoiding high seeding rates, cultivating before June 15 or eliminating cultivation all together, the use of integrated pest management to reduce the negative effects of non-target fungicide applications, and weekly field scouting for early detection and fungicide treatments (Brenneman et al., 1988). "Omega 500F" (SCP 71512-1B-1000 0503 126357, Syngenta, Greensboro, NC), a new generation fluazinam (Smith et al., 1991a), has been effective for control of Sclerotina blight in peanut, however treatments are costly, particularly with reduced prices associated with the elimination of the peanut quota system (K.E. Dashiell, personal communications, 2004). *S. minor* has a wide range of hosts that includes 21 families, 66 genera, and 94 species of both cultivated and wild plants and can survive up to 3-8 years in the soil as sclerotia without a host (Abawi et al., 1985, Melzer et al., 1997; Goldman et al., 1995). Wide host ranges and sclerotial longevity limit the effectiveness of crop rotation as a means of control *for S. minor* (Goldman et al., 1995).

Host plant resistance is viewed as the most effective solution to the Sclerotinia blight *p*roblem, however resistance inheritance is not clearly understood (Goldman et al., 1995). A single study published in 1992, utilized area under the disease progress curve (AUDPC) of disease severity to study resistance heritability (Wildman et al., 1992). This study indicated while broad

sense heritability was high (41% to 50.3%) narrow sense heritability was low (14% to 23%) (Wildman et al., 1992). There seems to be multiple mechanisms of resistance that control *S. minor* infection. These factors include avoidance of disease due to architecture, maturity, and/or greater resistance of the plant tissue (Chappell et al., 1995). Genotypes with more prostrate growth habits exhibit more susceptibility to disease than those with a more upright growth habit. Detached-shoot tests have demonstrated that there is also an additional physiological form of resistance of an unknown form (Akem et al., 1992). Peanut breeding lines with Spanish ancestry appear to be more resistant to *S. minor* than other market classes (Goldman et al., 1995).

The objectives of the first manuscript were to study inheritance of resistance to Sclerotinia blight in selected peanut cultivars using detached-shoot inoculations, and to examine the physiological mechanisms in isolation from architectural mechanisms. The objectives the second manuscript were to evaluate: 1.) the effects of seeding rate on disease incidence and severity of Sclerotinia blight in peanut research plots, 2.) level of apparent resistance at different seeding rates, 3.) determine the possibility of making early generation selections, using disease incidence and severity as forms of resistance indication, 4.) methods that would produce the best results in space planted breeding plots.

CHAPTER I

INHERITANCE OF RESISTANCE TO SCLEROTINIA BLIGHT IN SELECTED PEANUT CULTIVARS

ABSTRACT

There are many constraints to peanut (*Arachis hypogea* L.) production, including a wide array of insects, diseases, and abiotic stresses. Sclerotinia blight caused by *Sclerotinia minor* Jagger has become one of the major limiting factors in peanut production. Yield losses of 10% are not uncommon, however, in cases of severe infection losses of up to 50% may occur. Host plant resistance is viewed as the most effective management approach to Sclerotinia blight, however, resistance inheritance is not clearly understood. The objectives of this research were to determine the inheritance of resistance to Sclerotinia blight in selected peanut cultivars (utilizing detached-shoot inoculations), and to examine the existence of a physiological mechanisms of disease resistance in isolation from architectural mechanisms. Two resistant cultivars 'Tamspan 90' and 'Southwest Runner' were crossed in a 4 X 4 diallel with two susceptible cultivars 'Okrun' and 'Flavor Runner 458' to produce F_1 seed. A total of 405 F_1 plants were evaluated along with an additional 20 plants of each parent as control. A total of 1144 F_2 plants were tested along with 27 shoots of each or the four parents as control. Mean Area Under the Lesion Expansion Curve (AULEC)

ranged from 9.01 for the parental control Southwest Runner to 11.01 for Tamspan by Flavor Runner 458 in the F_1 populations. Mean F_2 AULEC values ranged from 8.6 for Southwest Runner to 11.3 for Southwest Runner by Flavor Runner 458. Large environmental variances derived by this testing method provided inconclusive measures of phenotypes. Current results suggest complex mechanisms of inheritance, which may include quantitative, dominance and cytoplasmic effects.

INTRODUCTION

The cultivated peanut (*Arachis hypogaea* L.) is a self-pollinated allotetraploid (2n=4x=40) believed to have originated in the Northwestern Argentina-Southern Bolivia region of South America (Kochert et al., 1991; Paik-Ro et al., 1992). The cultivated peanut is divided into two subspecies, *hypogaea* and *fastigiata.* The subspecies *Hypogaea* is further subdivided into 'hypogaea', which includes the runner and virginia U.S. market types, and 'hirsuta', which contains the peruvian market type. The subspecies *Fastigiata* is divided into 'fastigiata', containing the valencia market type, and 'vulgaris' or 'spanish' market type (Paik-Po et al., 1992).

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The objectives of this research were to study inheritance of resistance to Sclerotinia blight in selected peanut cultivars utilizing detached-shoot inoculations, and to examine the physiological mechanisms in isolation from architectural mechanisms.

MATERIALS AND METHODS

Plant material

‘Tamspan 90’ is a Spanish market type with good resistance to Sclerotinia blight. It was released by the Texas Agricultural Experiment Station, Texas A&M University System and the USDA-ARS in 1990. Tamspan 90 is a typical spanish type peanut with typical vegetative growth, physical appearance, rate of growth, foliage density and main stem height (Smith et al., 1991b).

‘Southwest Runner’, a runner market-type peanut cultivar with good resistance to *Sclerotinia minor* comparable to that of Tamspan 90, was jointly release by the Oklahoma Agricultural Experiment Station and the USDA-ARS in 1995. The Southwest Runner plant type is intermediate between typical spanish and runner cultivars. It exhibits a unique growth habit with robust, prostrate lateral branches and a prominent vertical main stem. The main stem bears flowers, atypical for most runner type cultivars (Kirby et al., 1998).

‘Okrun’ peanut was developed and released cooperatively by the USDA-ARS and the Oklahoma Agricultural Experiment Station in 1986 as the first commercial runner peanut cultivar developed in Oklahoma. Plant, pod and seed morphology and length of growing season of Okrun resemble that of Florunner. 'Okrun' is susceptible to all common peanut diseases, but it is more drought

tolerant than Florunner. Okrun has a small but consistent advantage in yield over Florunner (Banks et al., 1989).

'Flavor Runner 458' is a 'High Oleic' runner type variety released by Mycogen Co (Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August). The plant growth habit is prostrate with an alternate branching pattern. Flavor Runner 458 is similar to Floruner in regards to pod and seed color, seedling vigor, hull thickness, and disease and insect resistance. This variety was utilized as the second *S. minor* susceptible variety (Dr. Dan Gorbet and Dr. Hassan Melouk, personal communication, 2002).

The two resistant lines Tamspan 90 and Southwest Runner were crossed in a 4 X 4 diallel with the two susceptible cultivars Okrun and Flavor Runner 458 to produce F_1 seed. Crosses were made in the greenhouse in July 2003 and June 2004. There were 405 crosses producing 153 F_1 seeds in 2003, and 280 crosses producing 250 F_1 seeds in 2004. The 153 F_1 seeds produced in 2003 were grown over the winter in the greenhouse to obtain 2484 F_2 seeds for detached shoot testing (see Table 1). The F_2 seeds produced from a single F_1 plant had germinations ranging from 42%-100%.

Testing of F_1 and F_2 lines

A total of 403 F_1 shoots from the 2003 and 2004 crosses were evaluated for reaction to S. minor along with an additional 21 shoots of each parent as control. A total of 1144 F_2 shoots, produced from F1 crosses of 2003, were tested along with 27 shoots of each or the four parents as control (Table 2). The

apical fifteen cm of the central leader was removed from each plant genotype. The shoots were individually immersed in water in 1 x 14 cm test tubes, and supported by foam plugs, with tubes supported by a wooden base. Lower leaves were removed and a 4 mm mycelial plug of *S. minor*, taken from the periphery of a 48 hour old culture grown on potato dextrose agar (PDA). Inoculum was placed between the stem and the petiole in the middle of the shoot. Inoculated shoots were placed in a fabricated polyethylene enclosure 60 X 60 X 60 cm at 22°C. Relative humidity was maintained at 95-100% for the first 48 hours by lining the bottom of the enclosure with a saturated bath towel and closing off the open end. At 48 hours humidity was allowed to drop by allowing airflow through an opening in the enclosure so that humidity could be reduced to 60-70% for the next four days per Melouk et al. 1992. Repeated lesion measurements were taken at 48, 72, 96, & 120 hours and used to calculate an Area Under the Lesion Expansion Curve (AULEC) for each genotype including the parental controls. A total of 1144 F_2 plants averaging 95 shoots per line and a total of 135 parental shoots averaging 35 shoots per parent were tested (Table 2.)

Heritability estimation and data analysis

Estimation of narrow sense heritability (h^2_n) was calculated by parent-offspring regression of the F_2 plants on parents (Smith & Kinman, 1965). Data were analyzed using regression analysis, generalized least squares method, and distribution of data in SAS 9.1 (Copyright (c) 2002-2003 by SAS Institute Inc.,

Cary, NC, USA). Hartley's (1950) F_{max} -test was used to test for equal variances (Sokal & Rohlf, 1995). The model used for computation of heritabilities was

$$h^2_n = \beta$$

Where h^2_n is narrow sense heritability and β is parent offspring regression. The model used for computation of significant differences and interaction of means was:

$$Y = \mu + \alpha_i + \beta_j + \alpha_i\beta_j + e_{ij}$$

Where μ is the overall mean, α_i is the random effect of block $_i$, β_j is the fixed effect of genotype. Interaction evaluated was $\alpha_i\beta_j$ the random interaction effect of block $_i$ and genotype $_j$, and e_{ij} as the experimental error, mean μ, variance σ^2.

RESULTS AND DISCUSSION

Means and variances for AULEC were calculated for parental controls and each diallel from which initial equality of variances was checked by Hartley's (1950) F_{max} -test. The resulting data indicated unequal variances at the p=0.01 level for both the F_1 and F_2 populations, consequently all data were transformed by taking the square root of all AULEC values for further analysis. Transformed AULEC values were ranked for both the F_1 detached shoot studies (Table 3) and the F_2 detached shoot studies (Table 4) to test specific combining ability. Increased susceptibility is indicated by larger average AULEC indicated for a given genotype. All data were grouped according to significance (p=0.05), all those included in a single letter grouping were not significantly different. Half diallel combinations were also tested to determine general combining ability for F_1 detached shoot studies (Table 5) and the F_2 detached shoot studies (Table 6). Distributions for all genotypes were evaluated to determine normality of data. All populations presented normal distributions except Okrun by Tamspan 90 (Fig. 1) in the $F_{1,}$ and Tamspan 90 by Flavor Runner 458 in the F_2 population (Fig. 2), both of which appeared slightly bi-modal, while the parental controls tended to skew slightly to resistant or susceptibility based on resistance type (Figs. 3 & 4).

F_1 results

The F_1 diallel genotypes and the parents produced two basic groups, that were not significantly different from the smallest (resistant) mean AULEC which was Southwest Runner at 9.01, and those that were not significantly different from the largest (susceptible) mean AULEC which was the cross Tamspan 90 X Flavor Runner 458 at 11.01 (Table 3.). While there was a general separation there was some overlap in these classifications, which may be due in part to the high environmental variances produced by this test and low seed numbers available for testing of all F_1 populations. The resistant group was comprised mainly of F_1 plants from resistant by resistant crosses and crosses that included Southwest Runner by susceptible with two crosses including Tamspan 90 by susceptible. The susceptible group was comprised mainly of F_1 plants from susceptible parent by susceptible parent and crosses involving Tamspan 90. The F_1 half diallel indicated that no significant difference existed for three out of the four cultivars when used as a male versus a female, however, Flavor Runner 458 demonstrated a significant difference at ($p=0.02$) level (Table 5).

F_2 results

The F_2 diallel progeny and the parents produced three basic groups, those that were not significantly different from the smallest (resistant) mean AULEC which was ‘Southwest Runner’ at 8.6, and those that were not significantly different from the largest (susceptible) mean AULEC, which was the cross ‘Southwest Runner’ X ‘Flavor Runner 458’ at 11.3, and those that fell into an

intermediate group that included individuals not significantly different from either susceptibility type (Table 4.). As expected with a segregating population the variance increased by an average of 30% (1.8) over those obtained in the non-segregating F_1 population. The F_2 half diallel indicated a significant difference ($p=0.01$) for Flavor Runner 458 when used as a male versus a female parent. Used as a female parent, Flavor Runner 458 produced mean AULEC scores that put it in the resistant group. However, when Flavor Runner 458 was used as the male parent the resulting mean AULEC scores put it in the susceptible group which is consistent with the F_1 half diallel findings. These results indicate that cytoplasm may influence the inheritance of resistance to *Sclerotinia minor*. Those crosses that included the resistant parent Tamspan 90 typically demonstrated the most susceptible AULEC scores in the F_2 which is bourn out by the negative heritability scores though not significant do trend the same direction as the means obtained for all reciprocal crosses with this parent (Table 7.). This is somewhat inconsistent with previous findings by Goldman et al. (1995) who reported that backcrossing to Tamspan 90 and using it as a single cross parent produced progeny with good resistance in the field. The generations utilized in their test, however, were more advanced ($F_{2:3}$ backcross; $F_{4:5}$ single cross) compared to our progeny populations. This incongruous finding could be attributable to epistasis that is recovered in the backcross populations or simply that the high variances produced by this test make it unsuitable for this type of analysis.

Heritability Results

Narrow sense heritability was calculated by parent offspring regression for each cross. All heritability results were low ranging from -0.32 to 0.24, with the exception of the Flavor Runner 458 by Southwest Runner with a result of -0.97. Based on confidence intervals none of the heritability values obtained were significant (Table 7.), and little real information may be derived from these values. This outcome may be attributable to high environmental variances produced by this method of testing, and fairly high standard errors with the population sizes utilized in this test. Variance increased from F_1 populations with a mean variance of 0.46 and a range of 2.9-6.9 to the F_2 populations with a mean variance of 6.13 and a range of 3.1-7.9. The larger population sizes tested in the F_2 did reduce the mean standard error to 0.37 (range 0.27-0.59) from the F1 population mean standard error of 0.46 (range 0.40-.056). In order to obtain valid heritability values utilizing these test populations sizes, would need to be greatly increased and/or a means of reducing environmental variance would need to occur.

CONCLUSIONS

High environmental variances produced inconclusive measures of phenotype. Current results suggest complex mechanisms of inheritance which may include quantitative, dominance, epistasis, and cytoplasmic effects. The cytoplasmic effects were indicated by Flavor Runner 458 as it consistently produced lower mean AULEC scores when used as a female parent. Dominance is indicated by increased mean AULEC from the F_1 to F_2 in those lines with resistant parents included in the cross. The negative heritability scores when previous work indicated positive heritable variation in backcross populations for Tamspan 90 may indicate epistasis.

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Table 1. Number of peanut seeds produced for each of the diallel crosses. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), with each four letter combinations representing the female X male cross.

	F_1 seed produced		F_2 Seed Produced
Cross	2003	2004	2004
FLSW	13	22	179
FLOK	13	22	224
FLTS	13	19	244
SWOK	13	16	154
SWFL	2	18	22
SWTS	12	24	223
OKSW	17	23	353
OKTS	10	19	210
OKFL	11	20	212
TSFL	24	24	262
TSOK	11	17	200
TSSW	14	26	201
TOTALS	153	250	2484

Table 2. Number of parental, F_1, and F_2, shoots tested for each of the diallel crosses. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), with each four letter combination representing the female X male cross.

CROSS	SHOOTS TESTED		CROSS	SHOOTS TESTED	
	F1	F2		F1	F2
FLSW	35	98	OKFL	31	89
FLOK	32	112	TSFL	48	86
FLTS	35	97	TSOK	21	81
SWOK	28	107	TSSW	41	99
SWFL	20	17	FL	21	37
SWTS	36	110	OK	20	35
OKSW	39	147	SW	21	34
OKTS	29	101	TS	21	32
Total				403	1282

Table 3. Parental and F_1 means, standard errors, variances, and significant differences (P=0.05) for transformed area under the lesion expansion curve using a detached shoot technique for peanut, where different letters represent significant differences. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), and each four letter combinations represent the female X male cross.

Line	Mean	Variance	Std error	Significance group
SW	9.01	2.9	.50	A
FL	9.12	3.2	.52	A
TS	9.16	4.5	.50	A
FLTS	9.18	4.1	.46	A
SWOK	9.40	4.0	.47	AB
SWFL	9.70	3.6	.56	ABC
SWTS	9.79	3.4	.44	ABC
FLSW	10.08	4.6	.42	ABCD
TSSW	10.10	4.3	.40	ABCD
OKTS	10.19	6.3	.44	ABCD
TSOK	10.35	3.3	.46	BCD
OKSW	10.43	3.9	.43	BCD
FLOK	10.67	3.5	.45	CD
OK	10.67	5.8	.49	CD
OKFL	11.07	3.5	.44	D
TSFL	11.01	6.9	.41	D

Table 4. Parental and F_2 means, standard errors, and significant differences (P=0.05) for transformed area under the lesion expansion curve using a detached shoot technique for peanut, where different letters represent significant differences. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), and each four letter combinations represent the female X male cross.

Cross	Mean	Variance	Std Error	Significance Group
SW	8.6	6.0	.57	A
TS	8.8	3.1	.49	AB
OK	9.1	7.0	.49	ABC
TSSW	9.2	6.5	.30	ABC
FLOK	9.5	4.8	.30	ABCD
FLSW	9.6	6.2	.31	ABCD
OKTS	9.8	7.1	.32	BCDE
OKFL	9.8	7.9	.35	BCDEF
FL	9.9	5.6	.31	BCDEF
SWOK	9.9	5.4	.29	CDEF
FLTS	10.0	6.7	.31	CDEF
OKSW	10.1	6.1	.27	DEF
TSOK	10.3	5.8	.33	EFG
SWTS	10.4	6.3	.29	FG
TSFL	10.4	7.2	.35	FG
SWFL	11.3	6.4	.59	G

Table 5. Parental and F_1 Half diallel means, standard errors, and significant differences (P=0.05) for transformed area under the lesion expansion curve using a detached shoot technique for peanut, where different letters represent significant differences. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), and parent name followed by F represents use as a female parent and M represents use as a male parent.

Cross	Mean	Std Error	Significance Group
SW	9.01	.50	A
FL	9.12	.53	AB
TS	9.17	.5	AB
SWF	9.42	.47	AB
TSM	9.95	.44	AB
FLF	9.98	.33	AB
TSF	10.18	.40	BC
SWM	10.40	.42	BC
OKM	10.46	.29	C
OK	10.67	.49	C
FLM	10.75	.34	C
OKF	11.05	.28	C

Table 6. Parental and F_2 Half diallel means, standard errors, and significant differences (P=0.05) for transformed area under the lesion expansion curve using a detached shoot technique for peanut, where different letters represent significant differences. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL), and parent name followed by F represents use as a female parent and M represents use as a male parent.

Cross	Mean	Std Error	Significance Group
SW	8.61	.57	A
TS	8.81	.50	AB
OK	9.11	.50	AB
OKM	9.52	.30	ABC
FLF	9.71	.23	BCD
TSF	9.73	.25	BCD
OKF	9.81	.36	BCD
FL	9.91	.50	BCDE
SWF	9.92	.30	CDE
SWM	10.08	.27	CDE
TSM	10.11	.25	DE
FLM	10.28	.28	E

Table 7. Narrow sense heritability estimates for resistance of peanut to Sclerotinia blight for combined reciprocal crosses. Parents are listed as follows: 'Southwest Runner' (SW), 'Tamspan 90' (TS), 'Okrun' (OK), and 'Flavor Runner 458' (FL).

Reciprocal Cross	Narrow Sense Heritability	Confidence intervals	
		Lower	Upper
FL by SW	-0.97	-0.89	0.21
FL by OK	.24	-0.52	0.24
FL by TS	-.02	-0.17	0.25
OK by TS	-.10	-0.28	0.40
OK by SW	.04	-0.22	0.15
SW by TS	-.32	-0.39	0.13

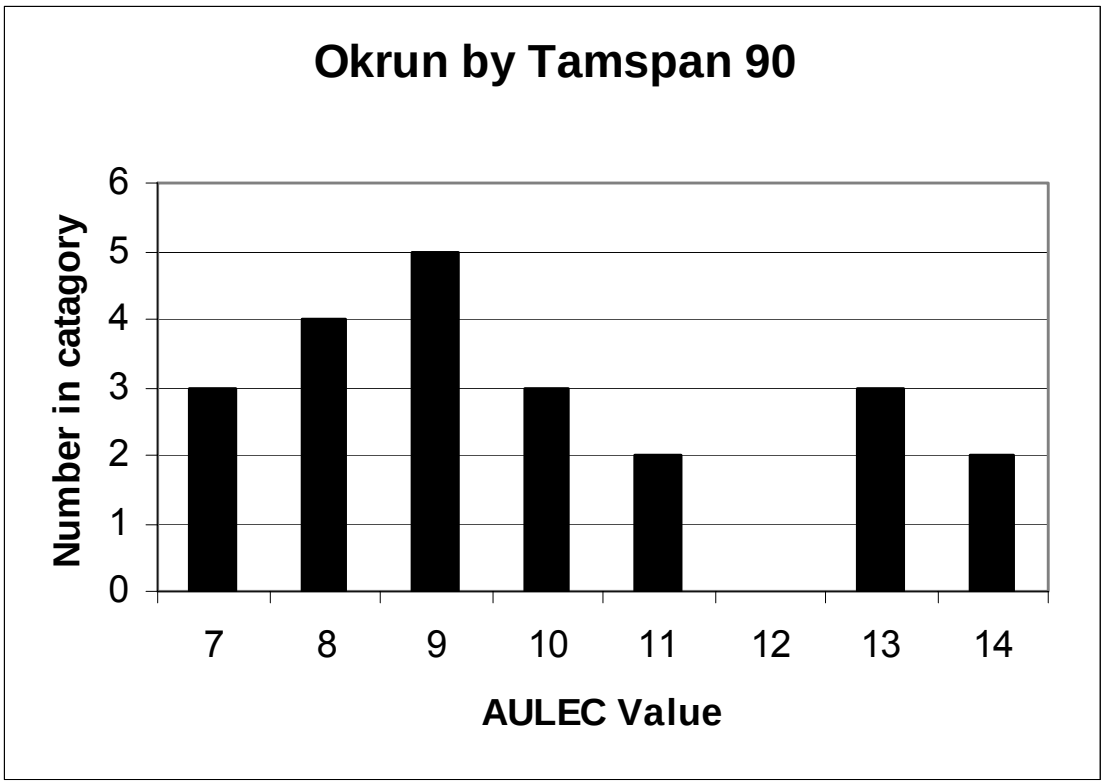

Figure 1. Distribution of area under the lesion expansion curve (AULEC) values for resistance of peanut to Sclerotinia blight the cross Okrun by Tamspan 90 in the F_1 population.

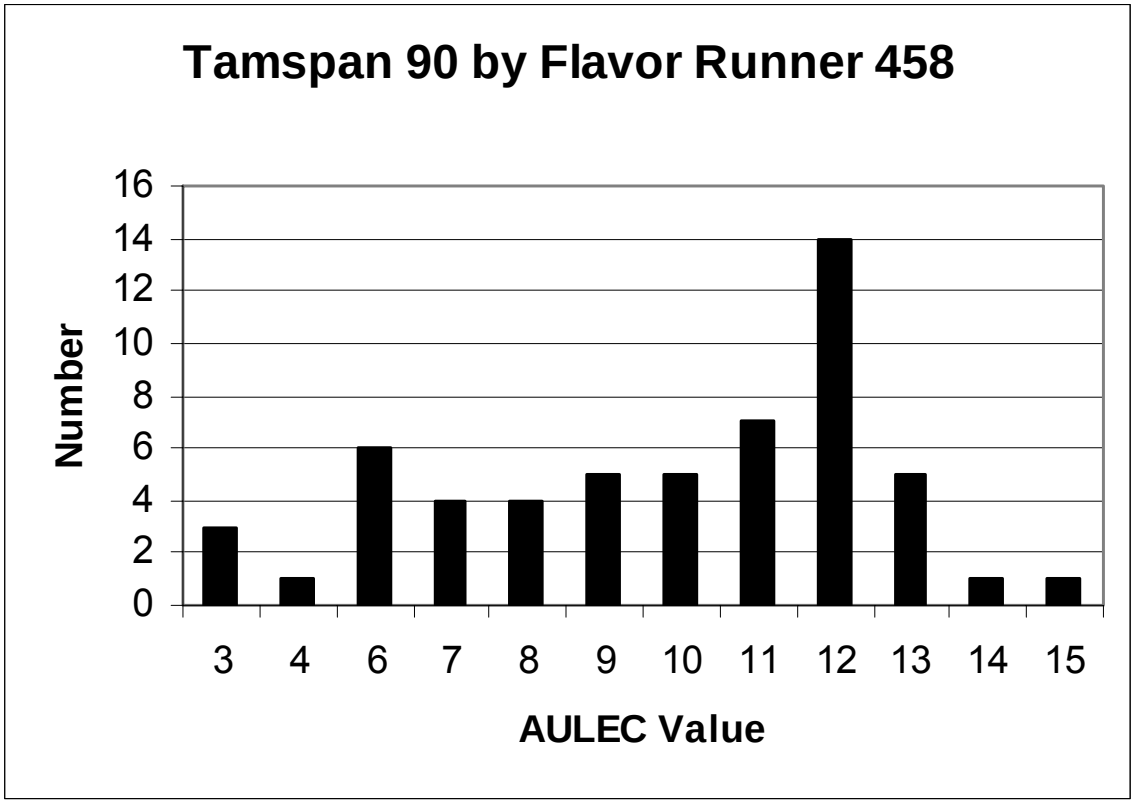

Figure 2. Distribution of area under the lesion expansion curve (AULEC) values for resistance of peanut to Sclerotinia blight the cross Tamspan 90 by Flavor Runner 458 in the F_2 population.

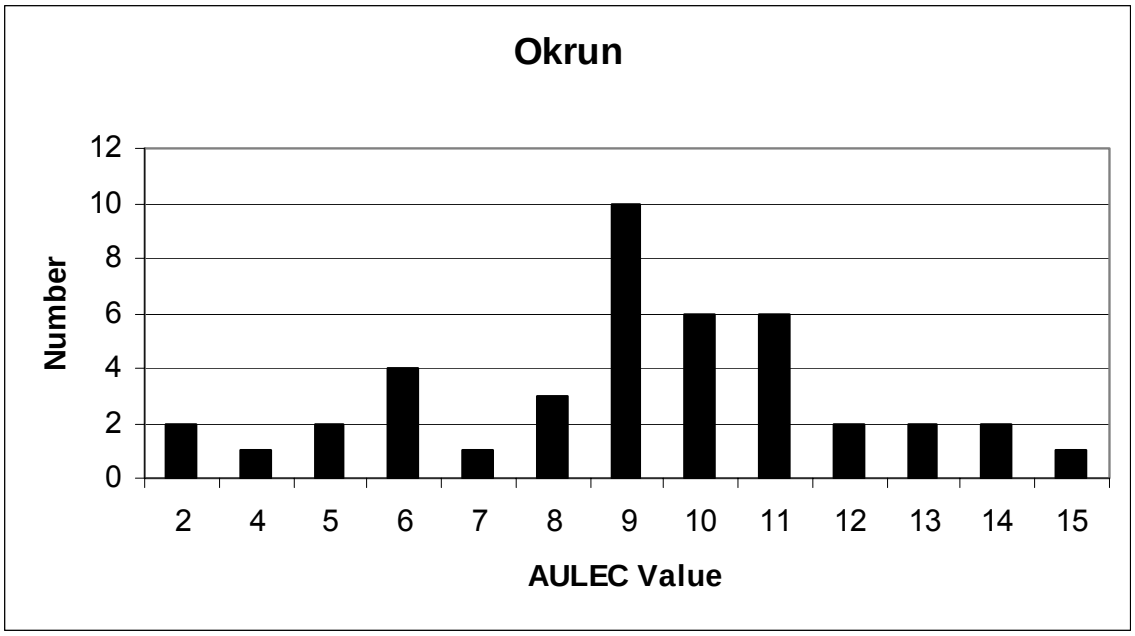

Figure 3. Distribution of area under the lesion expansion curve (AULEC) values for resistance of peanut to Sclerotinia blight the susceptible parent Okrun.

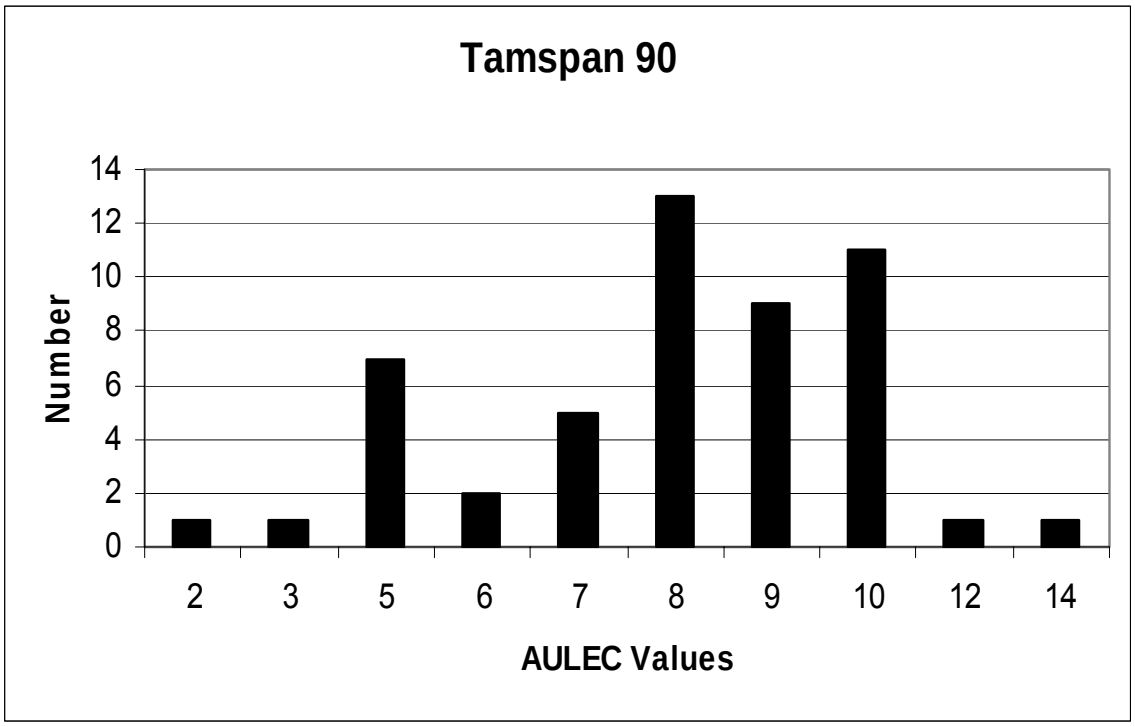

Figure 4. Distribution of area under the lesion expansion curve (AULEC) values for resistance of peanut to Sclerotinia blight the susceptible parent Tamspan 90.

CHAPTER II

EFFECTS OF SPACE PLANTING ON DISEASE INCIDENCE AND SEVERITY OF SCLEROTINIA BLIGHT IN PEANUT

ABSTRACT

There are many constraints to peanut (*Arachis hypogaea* L.) production, which include a wide array of insects, diseases, and abiotic stresses. Sclerotinia blight caused by *Sclerotinia minor* Jagger has become one of the major limiting factors in peanut production. The objectives of this research were to evaluate the effects of space planting on incidence and severity of Sclerotinia blight of peanut, evaluate the level of apparent resistance at different seeding rates, determine if making early generation selections would be effective, and what evaluation method would produce the best results in space-planted breeding plots. Four peanut cultivars, 'Tamspan 90', 'Southwest Runner', 'Okrun', and 'Flavor Runner 458', were evaluated in small field plots at four seeding rates, 75 seeds/4.57m (6.1 cm spacing), 30 seeds/4.57 m (15.3 cm spacing), 15 seeds/ 4.57 m (30.5 cm spacing), and 10 seeds/4.57 m (45.7 cm spacing), in 2003 and 2004. Plots that were evaluated on a presence/absence for date of disease onset, indicated that disease would be present in susceptible plots within two weeks of disease onset

provided suitable environment occurs. Plots which were evaluated for disease incidence presented clear trends of having increasing levels Sclerotinia blight with cultivar susceptability and increased plant spacing at a significance level of p=0.05. When disease severity was used as a measure of level of cultivar resistance, infected plots failed to demonstrate significant differences to determine level of overall resistance of those cultivars included in this test with the exception of 'Okrun' which was significantly different from the resistant cultivars at p=0.05 when disease severity computed on the basis of infected stems per infected plant only. Use of a combination of date of disease onset, and final disease incidence may provide an efficient selection tool for resistance to *Sclerotinia minor.*

Key Words: *Arachis hypogaea* L., *Sclerotinia minor* J., seeding rate, disease incidence, disease severity.

INTRODUCTION

There are many constraints to peanut (*Arachis hypogaea* L.) production, which include a wide array of insects, diseases, and abiotic stresses. Sclerotinia blight caused by *Sclerotinia minor* Jagger has become one of the major limiting factors in peanut production (10,16). The first report of *Sclerotinia minor* affecting peanuts in the United States was in Virginia in 1971. In recent years, the disease has become more severe and has spread to North Carolina, Oklahoma, New Mexico, Louisiana, and Texas (17,19). Yield losses of 10% are not uncommon, however in cases of severe infection, yield losses of up to 50% may occur in a single field (10).

S. minor will attack all tissues within the peanut plant. However, stem infections are the most economically important because pegs form from the stems (5). Temperature, relative humidity and soil moisture play vital roles in the infection and colonization of plant tissues by *S. minor. Sclerotinia minor* is a soil-borne pathogen that is most severe during cool, wet weather, with a demonstrated optimum growth range of 15-25° C and a relative humidity approaching saturation (95-100%). These high humidities promote myceliogenic germination of sclerotia and are positively correlated with disease development (6,7). Disease development in the field is low when plants are small and without a dense canopy or complete ground cover. Outbreak of Sclerotina blight is most

often observed after vines are within 6 inches of touching or after vines lap between rows (6,14). Sclerotinia blight development is greatest as the plants reach maturity in September and October, due to cooler night time temperatures and higher relative humidities normally associated with fall climate changes. During this time the plant canopies increase contributing to the maintenance of higher humidity close to the ground (6).

Symptoms of *Sclerotinia* blight first appear at the top of the plant, and include chlorosis and wilting of the infected tissues. Examination of the lower canopy in early morning reveals the presence of cottony mycelia on the main stem, lateral branches, and the taproot near the soil line. Within 3-4 days of infection, the mycelia will mat and form sclerotia, 1-3mm in length on the outside and inside of infected tissues. Sclerotia will infect tissues including the stem and root tissues as well as pods produced on infected plants (10). Lesions caused by the infection of stems and branches are light tan or straw colored, turning dark brown. Once the lesions begin turning brown, shedding of infected stems, branches, and pegs may eventually cause plant death (2,10).

Current Sclerotinia blight management recommendations include: planting resistant cultivars, avoiding high seeding rates, cultivating before June 15 or eliminating cultivation all together using integrated pest management to reduce the negative effects of non-target fungicide applications, weekly field scouting for early detection and fungicide treatments (4). “Omega 500F” (SCP 71512-1B-1000 0503 126357, Syngenta, Greensboro, NC), a new generation fluazinam (17), has been effective for control of Sclerotina blight in peanut, however

treatments are costly, particularly with the reduced prices associated with the elimination of the peanut quota system (K.E. Dashiell, personal communications, 2004). *S. minor* has a wide range of hosts that includes 21 families, 66 genera, and 94 species of both cultivated and wild plants and can survive up to 3-8 years in the soil as sclerotia without a host (1,8,11). The survival may be modified by soil type, saturation, soil texture and nutritive properties. Those soils with higher water holding capacities such as a clay/sand with high organic matter will have a shorter sclerotia survival then a sandy clay with low organic matter (1). Wide host ranges and sclerotial longevity limit the effectiveness of crop rotation as a means of control *for S. minor* (8).

Host plant resistance is viewed as the most effective solution to the *S. minor* problem, however resistance inheritance is not clearly understood with quantitative inheritance suggested by a study using family resistance to infections as the basis of plant selection for development of resistant runner lines (8). A single study by Wildmen et al.,(19) utilized Area Under the Disease Progress Curve (AUDPC) of disease severity to study resistance heritability. This study indicated that broad sense heritability was high (41% to 50.3%), and narrow sense heritability was low (14% and 23%) (19). There seems to be multiple mechanisms of resistance that control *S. minor* infection. These mechanisms include avoidance of disease due to architecture, maturity, and/or greater resistance of the plant tissue (5). Those genotypes with more prostrate growth habits exhibit greater susceptibility to disease than those with a more upright growth habit (2).

The objectives of this research were to evaluate: 1.) the effects of space planting on disease incidence and severity of Sclerotinia blight in peanut research plots, 2.) level of apparent resistance at different seeding rates, 3.) determine the possibility of making early generation selections, using disease incidence and severity as forms of resistance indication, and 4.) methods that would produce the best results in space planted breeding plots.

MATERIALS AND METHODS

Four peanut cultivars were evaluated for Sclerotinia blight incidence and severity in small field plots at four seeding rates, 75 seeds/4.57m (6.1 cm spacing), 30 seeds/4.57 m (15.3 cm spacing), 15 seeds/ 4.57 m (30.5 cm spacing), and 10 seeds/4.57 m (45.7 cm spacing), in 2003 and 2004. Four cultivars 'Tamspan 90', 'Southwest Runner', 'Okrun', and 'Flavor Runner 458' were used in this study.

Plant material. Tamspan 90 is a spanish market type with good resistance to sclerotinia blight. It was released by the Texas Agricultural Experiment Station, Texas A&M University System and the USDA-ARS in 1990 (18). Tamspan 90 is a typical spanish type peanut with typical vegetative growth, physical appearance, rate of growth, foliage density and main stem height (18).

Southwest Runner is a runner U.S. market-type peanut cultivar with moderate resistance to *S. minor* comparable to Tamspan 90. Southwest Runner was a joint release by the Oklahoma Agricultural Experiment Station and the USDA-ARS in 1995 (9). The Southwest Runner plant architecture is an intermediate between typical spanish and runner cultivars. It exhibits a unique growth habit with robust, prostrate lateral branches and a prominent vertical main stem. The main stem bears flowers, atypical for most runner type cultivars. (9).

Okrun peanut was developed and released cooperatively by the USDA-ARS and the Oklahoma Agricultural Experiment Station in 1986 as the first commercial runner peanut cultivar developed in Oklahoma (3). Plant, pod and seed morphology and length of growing season of 'Okrun' resemble that of 'Florunner'. Okrun is susceptible to all common peanut diseases, but it is more drought tolerant than Florunner. 'Okrun' was a small but consistent advantage in yield over florunner in Oklahoma (3).

Flavor Runner 458 is a 'High Oleic' runner type variety released by Mycogen Co (Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August). The plant growth habit is prostrate with an alternate branching pattern. Flavor Runner 458 is similar to 'Florunner' in regards to pod and seed color, seedling vigor, hull thickness and disease and insect resistance. This cultivar is also susceptible to *S. minor* (Dr. Dan Gorbet and Dr. Hassan Melouk, personal communication, 2002).

Field and planting design. The field site was at the Caddo Research Station near Fort Cobb, Oklahoma. Plots were artificially infested with 3.3 grams of inoculum per meter in 2003 when testing indicated that sclerotia density was below one sclerotia per 100 g of soil. Plots were not artificially infected in 2004. *S. minor* was grown on sterilized oat seeds which were inoculated with three to four day old cultures grown on potato dextrose agar for two and a half to three weeks until sclerotia formed. Cultures were then spread flat and allowed to bench dry for an additional three to four weeks. The dried inoculum was then used in the field to inoculate plots. Mean low ambient temperature was 17 °C for

both 2003 and 2004; mean high temperatures were 30°C and 29°C for 2003 and 2004, respectively for the months of May through October. Total rainfall was 37cm in 2003 and 43cm in 2004 for the months of May through October. The soil was a moderately deep, well drained loamy soil, nearly level to slightly sloping of the cobb soil series.

A randomized complete block experimental design with split plots and four replications was used during each of the two years of this study. Main plots were seeding rates and sub-plots were cultivars. Each block consisted of 16-two row plots, 4.57m long with rows 0.91m apart, and a 1.5m separation every 4.57m for stacked plots. Stands were planted at desired rates, 75 seeds/4.57m (6.1 cm spacing) which was the control rate as used in grower fields, 30 seeds/4.57 m (15.3 cm spacing), 15 seeds/ 4.57 m (30.5 cm spacing), and 10 seeds/4.57 m (45.7 cm spacing) to allow for differential stands as would occur in a breeding program. Stands were counted post emergence for later disease incidence scoring. Planting occurred on May 20, 2003 and May 11, 2004 and harvested/scored, October 17, 2003 and Oct. 6, 2004, allowing an average of 148 growing days. Recommended standard production practices for fertilizer, herbicide and irrigation for Oklahoma were followed for both years. Leaf spot was controlled with Headline (BASF, Research Triangle Park, NC) and Folicur (Bayer CropScience, Research Triangle Park, NC), for both years but no other fungicidal applications were utilized.

Scoring and data analysis. Disease incidence (DI) was determined by the percentage of plants infected with Sclerotina blight by the presence of visible

above-ground symptoms. A plant having any evidence of Sclerotina blight was scored as infected. Each two row plot was scored prior to harvest each season and plants that were dead due to other diseases were eliminated from the Incidence and Severity scorings. Disease severity was calculated in two ways: 1. as the total number of primary lateral stems and the main stem infected per plot divided by total number of infected plants per plot (DS), and 2. as the total number of lateral stems and main stem infected per plot divided by total number of plants per plot (DSP). Generalized least squares were used to separate means of disease incidence and severity among genotypes and seeding rates (SAS 9.1, Copyright (c) 2002-2003 by SAS Institute Inc., Cary, NC, USA). Unless otherwise indicated, a significance level of P= 0.05 was used to determine significant differences between treatments. The model used to compute significant differences and interactions was:

$$Y=\mu+\alpha_i+\beta_{j(i)}+ \gamma_k+\tau_l + \alpha_i\beta_{j(i)}+ \alpha_i\gamma_{ik} + \alpha_i\tau_l + \alpha_i\beta_{j(i)}\gamma_k + e_{ijkl}$$

Where μ is the overall mean, α_i is the random effect of year $_i$, $\beta_{j(i)}$ is the random effect of blocks nested within year $_i$, γ_k is the fixed effect of rate $_k$, and τ_l Is the fixed effect of cultivar $_l$. Interactions evaluated were $\alpha_i\beta_{j(i)}$ the random interaction effect of year $_i$ and block $_j$, $\alpha_i\gamma_{ik}$, the fixed interaction effect of year $_i$ and rate $_k$, $\alpha_i\tau_l$ the fixed interaction effect of year $_i$ and cultivar $_l$, $\alpha_i\beta_{j(i)}\gamma_k$ the random effect of block $_j$ and rate $_k$ nested within year $_i$, and e_{ijk} as the experimental error, mean μ, variance σ^2.

RESULTS AND DISSCUSSION

Sclerotinia blight was first noted in field plots on September 19, 2003, and August 9, 2004, after which plots were then evaluated bi-weekly for disease onset. Plot evaluations were scored on a disease presence/ absence for date of disease onset for all plots. Only the resistant lines of Tamspan 90 and 'Southwest Runner' presented no disease symptoms in a few plots at the final scoring before the comprehensive; incidence and severity scores were completed at harvest. The susceptible lines all presented some level of disease, with the exception of one plot of Flavor Runner 458 planted at 30.5 cm in 2003, by the second disease scoring. The presence/absence of disease onset method of scoring could allow for a rapid evaluation of a large number of genotypes in a breeding program. Those families which generally failed to show disease by the second evaluation could be used to indicate those lines which may provide some level of resistance, and require further detailed evaluation at harvest. Dow (7) Demonstrated that maturity of the plant has an effect on the ability of *S. Minor* to infect plant tissue with six week old tissue producing 100% infection while 13 week old tissue producing only 67% infection. Additional work is Brenneman (4) also supported the effect of maturity of plant tissues affecting infection in detached shoot studies with apical tissue having a greater susceptibility than

more mature basal tissues. A second factor that would effect disease onset by *S. Minor* is vine growth as reported by Phipps (13) when stems in adjacent rows were less than 15 cm from touching or overlapping the likelihood of infection became significant in the infection process. Based on these factors evaluation of plots based on infection onset date within families would require that those families of similar maturity be assessed together. An early maturing variety should achieve sufficient canopy for disease development earlier in the season and have more mature tissues at basal locations in the plant. A later maturing variety could begin initial disease development later due to reduced vine growth however there would also be more immature tissue at soil level increasing infection potential.

Disease initiation between the two years was 41 days apart with first wilting noted in 2003 on the 19th of September versus the 9th of August in 2004. The reason this occurred is an unusually cool weather pattern that provided for an average high of 27°C and low of 19°C for the dates of August 5th-8th,2004, where a more typical average high of 40°C and an average low of 24°C for the same period in 2003 was observed. When these data were analyzed for final disease incidence (DI) and disease severity on both infected plants (DS) and whole plot basis (DSP) produced no significant difference for any interaction involving year. P. M. Phipps, (13) in a 16 year study found that for Virginia, weekly scouting and application of fungicides at the first appearance of disease was most appropriate. These results would suggest that a review of this method for the southern great plains region may be appropriate.

Field infection of Sclerotinia blight was present in all peanut genotypes evaluated in the field, with mean DI ranging from 6 % to 99% (Table 1.). In the case of the two susceptible lines, Flavor Runner 458 the DI ranged from 49.5% to 98.6%, and Okrun ranged from 66.1% to 98.8%. DI values for the two resistant lines were Southwest Runner ranged from 21.3% to 48.8%, and Tamspan 90 ranged from 6.3% to 36.3%. Disease incidence presented clear trends of increasing with level of susceptability and increased plant spacing at a significance level of p=0.05 (Table 4.). This correlates well with a previous study by Akem et al. (2) which looked at disease incidence and disease progress values for genotype evaluation for plots planted at 0.3 m. Additional findings to the previous research work of Akem et al. 1992 is that increased plant spacing provided an increase in disease incidence even in resistant cultivars. The results presented here would suggest that space planting those genotypes that are to be selected for resistance to *S. minor* may be appropriate. The lowest mean DI for susceptible cultivars planted at 30.5 cm and 45.7cm was 86.6% indicating that the chance of selecting an apparently resistant plant over two years would be about two percent. These values would provide a positive opportunity for early individual plant selections so as to decrease the number of families that would have to be carried to late generation testing before determination of resistance to *Sclerotinia minor* could be carried out. This also supports Akem et al. (2) which looked at disease incidence and disease progress for the evaluation of genotype resistance. The results reported in this paper would suggest that the labor

intensive disease progress method may not be necessary for evaluation of plant resistance.

Disease severity, when considering only diseased plants, while somewhat reduced overall in the two resistance cultivars provided no clear picture of overall resistance of those cultivars included in this test (Table 2.). Resistant cultivars produced a spread of 1.4 stems per plant to 4.1 stems per plant while the range for susceptible lines was 1.8 stems per plant to 7.8 stems per plant. Although the susceptible lines seem to show a higher degree of severity there was minimal significance ($p=0.05$) within or among lines at the various seeding rates for disease severity (Table 5). The only significant differences for cultivar or rate was Okrun which had an increased severity with decreased seeding rate, although Tamspan 90 seemed to trend opposite of Okrun, however the differences between the control rate and 45.7 cm was only $p=0.22$ and thus not significant. This method of evaluation was labor intensive and yielded little to no useful information and there for lacks a real value as a useful breeding tool.

Disease severity, when the entire plot was considered, provided no additional separation of genotypes to either DI or DS analysis methods (Table 3). Resistance cultivars produced a two year mean spread of 1.4 stems per plant to 2.5 stems per plant with the range for susceptible lines was 2.0 stems per plant to 4.1 stems per plant the variability due to multiple infection was diluted and produced no significant differences for either cultivar or seeding rate (Table 6).

Brenneman et al. (4) indicated that avoiding high seeding rates was a current recommendation for disease reduction, however Phipps (15) found no

significant effect of disease incidence for whole plot factors such as planting date or seeding rate. The results presented in this paper tend to support the findings of Phipps (15) that reduced initial seeding rate will not reduce disease and in fact may increase disease. Dow (6) conducted a study of rows thinned after bloom to prevent compensation of plant canopy and unthinned rows that indicated while thinning reduced disease incidence and severity it also reduced yield. Based on these previous findings and those reported here re-evaluation of seeding rate recommendations may be indicated.

CONCLUSIONS

Plots evaluated on a presence/absence for date of initial disease symptoms indicated that Sclerotinia blight would be present in susceptible plots within two weeks of disease initiation. Disease incidence presented clear trends of increasing with level of susceptability and increased plant spacing at a significance level of p=0.05. Disease severity while somewhat reduced overall in the two resistance cultivars provided no clear picture of overall resistance of those lines included in this test with only 'Okrun' significantly different then the resistant cultivars at p=0.05. Use of a combination of final disease incidence and onset may provide an efficient selection tool for resistance to *Sclerotinia minor.*

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Table 1. Mean by year and rate of final Sclerotinia blight incidence of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma.

		Mean Disease Incidence		
Cultivar	Plant Spacing(cm)	2003 mean	2004 mean	Two yr mean
Flavor Runner 458*	6.1	49.5	96.9	73.2
Flavor Runner 458*	15.3	72.9	98.6	85.8
Flavor Runner 458*	30.5	77.3	95.8	86.6
Flavor Runner 458*	45.7	89.6	97.4	93.5
Okrun	6.1	66.1	97.4	81.7
Okrun	15.3	75.6	97.6	86.6
Okrun	30.5	98.8	96.6	97.7
Okrun	45.7	97.9	96.0	97.0
Southwest Runner	6.1l	30.7	21.3	26.0
Southwest Runner	15.3	36.2	20.3	28.2
Southwest Runner	30.5	51.4	22.0	36.7
Southwest Runner	45.7	48.8	26.9	37.8
Tamspan 90	6.1	8.7	8.1	8.4
Tamspan 90	15.3	6.3	16.1	11.2
Tamspan 90	30.5	17.3	18.2	17.8
Tamspan 90	45.7	36.3	26.1	31.2

[1] Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August.

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Table 2. Means by year and rate of Sclerotinia blight as a percent of infected stems per infected plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma.

		Mean Disease Severity[1]		
Cultivar	Plant Spacing (cm)	2003 mean	2004 mean	Two yr mean
Flavor Runner 458*	6.1 cm	7.1	3.3	5.2
Flavor Runner 458*	15.3 cm	3.6	6.3	4.9
Flavor Runner 458*	30.5 cm	4.4	7.3	5.8
Flavor Runner 458*	45.7 cm	3.6	7.8	5.8
Okrun	6.1 cm	1.8	2.9	2.4
Okrun	15.3 cm	2.8	5.4	4.1
Okrun	30.5 cm	4.3	5.4	4.9
Okrun	45.7 cm	4.4	5.5	6.3
Southwest Runner	6.1 cm	3.2	3.9	3.5
Southwest Runner	15.3 cm	1.8	4.1	2.9
Southwest Runner	30.5 cm	2.9	3.6	3.2
Southwest Runner	45.7 cm	3.1	3.8	3.4
Tamspan 90	6.1 cm	2.6	3.8	3.2
Tamspan 90	15.3 cm	1.4	3.2	2.3
Tamspan 90	30.5 cm	1.7	2.8	2.2
Tamspan 90	45.7 cm	1.7	2.1	1.9

[1.] Total number of primary lateral stems infected per plot divided by total number of infected plants per two row plot.

[2]. * Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August.

Table 3 Means by year and rate of Sclerotinia blight as a percent of infected stems per total plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma.

		Mean Disease Severity[1]		
Cultivar	Plant Spacing (cm)	2003 mean	2004 mean	Two yr mean
Flavor Runner 458*	6.1 cm	2.4	2.8	2.6
Flavor Runner 458*	15.3 cm	2.7	3.4	3.0
Flavor Runner 458*	30.5 cm	3.6	1.0	2.2
Flavor Runner 458*	45.7 cm	3.4	2.3	2.9
Okrun	6.1 cm	1.2	2.8	2.0
Okrun	15.3 cm	2.1	3.4	2.0
Okrun	30.5 cm	4.3	1.0	4.1
Okrun	45.7 cm	4.3	3.2	2.8
Southwest Runner	6.1 cm	0.8	2.8	1.8
Southwest Runner	15.3 cm	0.9	2.0	1.4
Southwest Runner	30.5 cm	1.4	2.6	2.0
Southwest Runner	45.7 cm	1.8	2.3	2.1
Tamspan 90	6.1 cm	0.2	4.8	2.5
Tamspan 90	15.3 cm	0.2	4.8	2.5
Tamspan 90	30.5 cm	0.4	1.7	1.0
Tamspan 90	45.7 cm	0.8	3.6	2.1

1. Total number of primary lateral stems infected per plot divided by total number of plants per two row plot.

2. * Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August.

Table 4. Overall means by year and rate of sclerotinia blight incidence of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma

	Plant Spacing (cm)			
	6.1 cm	15.3cm	30.3 cm	45.7
Cultivar	%	%	%	%
Okrun	81.7 a A	86.6 a A	97.67 a A	96.9 a A
Flavor Runner 458*	73.2 a A	85.8 a AB	86.6 a AB	93.5 a B
Southwest Runner	25.9 b A	28.2 b A	36.7 b A	37.2 b A
Tamspan 90	8.4 b A	11.2 b A	17.8 b AB	31.2 b B

[1] Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August.

[2.] Significance (p=0.05) for rate within a cultivar given by lower case letters (columns) and significance (p=0.05) among lines for a given seeding rate given as an uppercase letter (rows).

Table 5. Overall means by year and rate of Sclerotinia blight as a percent of infected stems per infected plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma.

	Plant Spacing (cm)			
	Control	15.3cm	30.3 cm	45.7
Cultivar	%	%	%	%
Okrun	2.39 b A	4.1 ab AB	4.9 ab BC	6.3 a C
Flavor Runner 458*	5.2 a A	4.9 a A	5.8 a A	5.8 a A
Southwest Runner	3.5 ab A	2.9 b A	3.3 bc A	3.5 b A
Tamspan 90	3.2 b A	2.3 b A	2.2 c A	1.9 b A

1. Significance (p=0.05) for rate within a cultivar given by lower case letters (columns) and significance (p=0.05) among lines for a given seeding rate given as an uppercase letter (rows).

2. * Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August

Table 6. Overall means by year and rate of Sclerotinia blight as a percent of infected stems per total plants per two row plot of peanut in 2003 and 2004 trials near Fort Cobb, Oklahoma.

	Seeding Rate (cm)			
	6.1	15.3cm	30.3 cm	45.7
Cultivar	%	%	%	%
Flavor Runner 458*	2.6 aA	3.0 aA	2.2 aA	2.9 aA
Okrun	2.0 aA	2.0 aA	4.1 aA	2.8 aA
Southwest Runner	1.8 aA	1.4 aA	2.0 aA	2.1 aA
Tamspan 90	2.5 aA	2.5 aA	1.0 aA	2.1 aA

[1.] Significance (p=0.05) for rate within a cultivar given by lower case letters (columns) and significance (p=0.05) among lines for a given seeding rate given as an uppercase letter (rows).

[2] Moore, K.M. 1999. High Oleic Acid Peanut. U.S. Plant Patent 5945578. Date issued: 31 August

VITA

Andrea Lynn Maas

Candidate for the Degree of

Doctor of Science

Thesis: PEANUT CULTIVAR SELECTION FOR THE DEVELOPMENT OF RESISTANCE TO SLEROTINIA BLIGHT

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Name: Andrea Lynn Maas Date of Degree: May, 2005

Institution: Oklahoma State University Location: Stillwater Oklahoma

Title of Study: PEANUT CULTIVAR SELECTION FOR THE DEVELOPMENT OF RESISTANCE TO SCLEROTINIA BLIGHT

Pages in Study: 58 Candidate for the Degree of Doctor of Science

Scope and Methods of Study: The objectives of these studies were two fold and Included a study to determine inheritance of resistance to Sclerotinia blight in peanut utilizing detached-shoot inoculations, and a field study to evaluate the effects of space planting on disease incidence and severity of Sclerotinia blight in peanut research plots. Diallelic crosses were made utilizing 'Tamspan 90', 'Southwest Runner', 'Okrun' and 'Flavor Runner 458' as parents. 365 F1 plants and 1144 F2 plants were utilized to calculate AULEC values for analysis of inheritance. The four cultivars were also studied in the field utilizing a randomized complete block design with split plots for four seeding rates. Disease incidence and severity were recorded for all plots over two years and analyzed for differences among treatments.

Findings and conclusions: In the detached shoot study high environmental variances produced inconclusive measures of genotype. Current results suggest complex mechanisms of inheritance which may include quantitative, dominance, epistasis, and cytoplasmic effects. In the field study plots evaluated on a presence/absence for date of initial disease symptoms indicated that disease would be present in susceptible plots within two weeks of disease initiation. Disease incidence presented clear trends of increasing with level of susceptability and increased plant spacing at a significance level of $p=0.05$. Disease severity was reduced overall in the two resistant cultivars but was not significant at $p=0.05$ and thus not sufficient to differentiate susceptible from resistant plants. Use of a combination of disease onset and final disease incidence may provide an efficient selection tool for resistance to *Sclerotinia minor*.

Advisor's Approval:___Kenton Dashiell_____