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Control of Peanut Leafspot with a Combination of Resistance and Fungicide Treatment[1]

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ABSTRACT

Three peanut (*Arachis hypogaea* L.) plant introductions (PI), eight breeding lines (BL), and the cultivar 'Florunner' were grown as subplot treatments in a randomized complete block with a split plot arrangement to evaluate their reaction to *Cercospora arachidicola* Hori and *Cercosporidium personatum* (Berk. and Curt.) Deighton in 1979 and 1980. Main plot treatments consisted of (1) no fungicide applications, (2) applications of chlorothalonil (500 g/l) on 10-day intervals and (3) 20-day intervals. Disease assessments as lesion counts were made at 20-day intervals beginning 50 days after planting. From leaf samples taken at the fourth leaf from a stem apex, *C. personatum* (CP) was the most prevalent pathogen both years. Differences among lines in susceptibility to CP were highly significant ($P = 0.001$) at 90, 110, and 130 days after planting. In unsprayed plots at 110 days, Florunner had the highest average CP lesion count at 51 lesions/leaflet compared to 21 for PI 261893. Fungicide treatment application significantly reduced CP at 110 and 130 days in both years. Also, defoliation and yield differences among lines were highly significant in both years. Five breeding lines produced pod yields exceeding 3400 kg/ha with no fungicide applied, compared to 2267 kg/ha for unsprayed Florunner. All breeding lines and Florunner showed at least some response to chlorothalonil. Pod yields had highly significant negative correlations with CP lesion counts and percent defoliation, ranging from -0.31 to -0.48.

Key Words: *Arachis hypogaea, Cercospora arachidicola, Cercosporidium personatum,* Groundnut, Leafspot Disease.

Peanut leafspot, a disease caused by *Cercospora arachidicola* Hori (CA) and *Cercosporidium personatum* (Berk. and Curt.) Deighton (CP), is a worldwide problem on peanuts (*Arachis hypogaea* L.). Leafspot is probably the major disease problem in the southeastern USA peanut production area. This disease can be reasonably controlled with fungicides but at considerable expense (2,3). Control programs in Florida are based on fungicide applications at 10-14 day intervals beginning as early as 30-40 days after planting (8,9).

All peanut cultivars grown in the USA are susceptible in varying degrees to both leafspot pathogens (1,4). Higgins (5) reported on limited sources of resistance to leafspot pathogens in *A. hypogaea* and indicated that the probability of developing an agronomically acceptable cultivar with leafspot resistance was remote. However, some potentially useful *Arachis* germplasm with various levels of resistance to CA, CP, or both have been identified (1,3,4,7,10). A disease control program combining leafspot resistance and a reduction of fungicide applications would be desirable and less costly to the grower.

A study was initiated in 1979 at the Marianna Agricultural Research Center, University of Florida, to evaluate the differential reaction of 12 peanut genotypes to CA and CP. Genotypes selected represented different levels of resistance to CA and/or CP, and were from the subspecies *hypogaea* (3). The response of the genotypes to three fungicide treatments were determined. Disease and pod yield data were collected to evaluate the relationship of disease development, peanut genotype, and fungicide application. The peanut lines included potential candidates for cultivar release.

Materials and Methods

Three peanut plant intorductions, (PI), eight advanced (F_7-F_8) breeding lines (BL), and the cultivar 'Florunner' were grown as subplot treatments in a randomized complete block with a split-plot arrangement of treatments with four replications to evaluate their reaction to CA and CP in 1979 and 1980 at the Marianna Agricultural Research Center. All entries were rated previously as having some resistance to CA and/or CP, except for Florunner (3).

Three fungicide schedules were used as the main plot treatments as follows: (1) no fungicide application; (2) chlorothalonil, as 500 g/L, applied at 2.48 L/ha on a 10-day spray interval and (3) on a 20-day schedule beginning 40 days after planting. Chlorothalonil was applied with a tractor-mounted sprayer in 168 liters of water/ha at 345 kPa. Peanut entries were planted in late May each year in 2-row plots (91 cm apart, 6.1 m long) and seeded at 60 seed per row. Unsprayed border rows of Florunner separated main plots and served as spreader rows to enhance disease pressure. All plots were irrigated as needed.

Disease assessments were made at 50, 70, 90, 110, and 130 days after planting. Ten leaflets per plot from the fourth fully expanded leaf from the shoot apex were collected at each assessment time. Lesions were counted and identified as being caused by either CA or CP, according to color, shape, and pattern of sporulation that occurred (6). Defoliation was assessed prior to harvest at 132 and 130 days after planting in 1979 and 1980, respectively. Defoliation was based on the number of abscised leaflets compared to the total number of leaflets produced on 10 central stems per plot.

Lesion count and defoliation data were transformed prior to statistical analyses to reduce coefficients of variation and allow more valid testing of means. Lesion count data were transformed and analyzed in 1979 as $(\text{no. of lesions/leaflet} + 1)^{1/2}$ and in 1980 as $[(\text{no. lesions} + 1)/\text{cm}^2 \text{ of leaflet area}]^{1/2}$. Arcsine transformations of percentage defoliation data were analyzed.

All plots were dug with a digger-inverter at 139 days in 1979 and 137 days in 1980 and picked with a stationary peanut thresher three days after digging. Peanuts were dried and weighed by plot.

Results and Discussion

Table 1 gives information on the pedigree and proposed resistance of peanut genotypes. The PIs were selected primarily on the basis of their resistance to CA and/or CP. The breeding lines were selected for desirable agronomic traits, such as yield potential, size and quality of fruit, as well as leafspot resistance.

Lesion counts and identification showed that very little CA was present either year; thus, only CP data are presented. This apparent shift to a predominance of CP in Florida is recent and unexplained at this time (3). Lesion counts made up to 70 days after planting were very low, averaging less than one lesion per leaflet for all genotypes under all fungicide treatments, indicating that the disease

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Table 1. Indicated resistance of peanut genotypes to *Cercospora arachidicola* (CA) and *Cercosporidium personatum* (CP).

Genotype[1/]	Generation[2/]	Pedigree	Resistance[3/]
PI-4	F+	PI306230-2-2	CA
PI-5	F+	PI262090	CP
PI-6	F+	PI261893	CA & CP
BL-1	F_{7-8}	PI306230- x Florunner	CA
BL-2	F_{7-8}	439-16-6 x PI145681	CP & CA
BL-3	F_{7-8}	PI203396 x Florunner	CA & CP
BL-9	F_{7-8}	PI203396 x Florunner	CA & CP
BL-8	F_{7-8}	Florunner x PI121067	CA & CP
BL-10	F_{7-8}	PI121067 x Florunner	CA & CP
BL-11	F_{7-8}	439-17-2- x PI259785	CA
BL-12	F_{7-8}	PI261911 x Florunner	CP
Florunner	F+	F-439-16-10-	None?

[1/] PI refers to Plant Introduction (USA) and BL indicates breeding line from the Florida peanut breeding program.

[2/] F+ indicates advanced generation beyond F_{10}.

[3/] Proposed resistance based on prior evaluations in breeding nurseries (unpublished) or detailed field tests (3). Underlined designation indicates apparent main resistance.

had not yet progressed to the upper layers of the plant canopy. However, the unsprayed border rows of Florunner created heavier disease pressure throughout the tests than would normally occur in a commercial field and insured a fairly uniform distribution of inoculum.

There were significant differences each year for CP lesion counts at 90, 110, and 130 day samplings, for defoliation, and for pod yields (Table 2). The highly significant interactions between fungicide treatments and genotypes were expected since different levels of resistance to CP were known to be present among the entries. Disease development was slightly later in 1980, accounting for the nonsignificant effect of fungicide treatment at 90 days on CP lesion counts. The latter was a function of the lower rainfall in 1980 compared to 1979.

Table 2. Statistical significance of F tests in analysis of variance for number of *Cercospora personatum* (CP) lesions/leaflet at 90, 110, and 130 days after planting, defoliation, and pod yield, as related to fungicide treatment and peanut genotype.

Variable	Source of Variation					
	Fungicide Treatment		Genotype		Interaction	
	1979	1980	1979	1980	1979	1980
CP-90[1/]	***	NS[2/]	***	***	***	•
CP-110	***	**	***	***	***	**
CP-130[3/]	***	**	***	***	NS	NS
Defoliation	***	***	**	***	**	***
Pod Yield	•	***	***	***	***	***

[1/] CP = $\sqrt{\ }$ transformation of number of CP lesions/leaflet at 90, 110, and 130 days. Defoliation = Arcsine transformation of %.

[2/] Statistical significance: NS = not signficiant; *, **, *** indicate significance at P = 0.05, 0.01, and 0.001, respectively.

[3/] Sampling affected by defoliation at 130 days in unsprayed plots.

The number of CP lesions increased between successive samplings. In 1979, the unsprayed plots were too severely defoliated at 130 days to permit accurate lesion counts at the prescribed sample site in some plots. Disease progression in time was marked by a sharp increase between 90 and 110 days, conforming to the exponential phase. Disease pressure was clearly greater in the unsprayed plots as evidenced by the mean CP lesion counts (Table 3). Lesion counts averaged somewhat higher in 1980 than in 1979 at the 110 days sampling, even though lesion counts at 90 days were greater in 1979 than in 1980, at 4.5 vs. 0.2 in the unsprayed plots.

Table 3. Mean number of Cercosporidium personatum (CP) lesions per leaflet for each peanut genotype (cultivar, breeding lines - BL; and plant introductions - PI) sprayed with chlorothalonil (10 or 20 day interval) and unsprayed at 110 days after planting in 1979 and 1980.

Genotype	Number of CP lesions/leaflet[1/]					
	1979 Fungicide Treatment			1980 Fungicide Treatment		
	Unsprayed	20-day	10-day	Unsprayed	20-day	10-day
Florunner	40 abc[2/]	1.4 b	0.1 a	62 a	2.6 a	6.4 a
BL-1	47 a	5.8 a	0.1 a	38 bcd	5.5 a	0.6 a
BL-10	42 ab	2.3 ab	0.5 a	48 bcd	4.9 a	1.2 a
BL-2	35 bcd	0.7 b	0.1 a	27 de	3.3 a	1.5 a
BL-11	31 cd	2.8 ab	0.2 a	50 ab	3.3 a	0.8 a
BL-12	31 cd	1.9 ab	0.3 a	29 de	3.1 a	1.7 a
BL-8	30 cd	2.4 ab	0.0 a	48 bcd	3.8 a	2.0 a
PI-4	30 d	2.8 ab	0.1 a	35 cde	3.6 a	3.8 a
BL-9	18 e	0.9 b	0.1 a	30 de	2.1 a	0.6 a
BL-3	18 e	0.6 b	0.0 a	24 e	4.1 a	1.1 a
PI-5	17 e	0.9 b	0.2 a	30 de	3.4 a	0.7 a
PI-6	14 e	1.1 b	0.2 a	27 de	2.0 a	0.4 a
$\bar{x}$[3/]	29 a	2.0 b	0.2 b	37 a	3.4 b	1.7 b

[1/] Defoliation affected sampling in some plots.

[2/] Genotype means followed by a common letter (a, b, etc.) in a column are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test; analyses on CP data (see Table 2).

[3/] Column means followed by a common letter in the row within years are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test.

All genotypes had greatly reduced numbers of CP lesions at 110 days when chlorothalonil was applied, compared to unsprayed plots. Florunner was the most susceptible entry and showed the greatest response to fungicide treatment. PI-5, PI-6, BL-3, and BL-9 were among the most resistant genotypes, according to lesion counts in both years. Lesion counts were not significantly different among entries within the 10-day treatment either year or within the 20-day treatment in 1980. Chlorothalonil was clearly the dominant factor in leafspot control with the 10-day spray schedule both years and with the 20-day schedule in 1980. CP lesion counts for each genotype at 110 days in the unsprayed treatment generally supported previous assessments for relative resistance to CA and/or CP. BL-2, BL-3, BL-9, BL-12, PI-5, and PI-6 were proposed as having some resistance to CP, and they did have the lowest lesion counts. BL-3 and BL-9 are sisterlines with resistance from PI 203396 and they responded similarly (3). FL-11 and BL-8 showed the greatest differences in lesion counts between years in the unsprayed treatment, except for Florunner which could not be sampled in all plots due to defoliation in 1979.

Table 4 gives the mean percentage defoliation at 7 days before harvest for each genotype within each fungicide treatment in both years. Defoliation generally followed a similar pattern to CP lesion counts with higher defoliation percent associated with higher CP lesion counts. However, there were significant differences among genotypes within all fungicide treatments in both years, even with the 10-day treatment where leafspot was probably not a significant cause of defoliation. Each fungicide treatment

Table 4. Percent defoliation of peanut genotypes (cultivar, breeding lines - BL; and plant introductions - PI) sprayed with chlorothalonil (10 or 20 day interval) and unsprayed at 7 days before harvest in 1979 and 1980.

Genotype	Percent defoliation					
	1979 Fungicide Treatment			1980 Fungicide Treatment		
	Unsprayed	20-day	10-day	Unsprayed	20-day	10-day
Florunner	97 a[1]	55 ab	20 cd	93 a	42 ef	10 bc
BL-2	90 b	57 ab	32 a-d	81 b	44 ef	11 bc
BL-3	89 bc	56 ab	18 d	88 ab	52 cde	13 abc
BL-10	87 bc	60 a	33 a-d	85 ab	61 a-d	10 bc
BL-12	80 bc	63 a	35 abc	86 ab	49 def	12 abc
BL-1	88 bc	64 a	22 bcd	82 b	68 a	11 bc
BL-11	87 bc	61 a	36 abc	84 b	65 abc	21 a
BL-8	82 bc	65 a	39 a	79 b	38 f	10 bc
BL-9	80 bc	42 b	20 cd	84 b	45 ef	8 c
PI-6	78 bc	60 a	35 abc	79 b	53 b-e	14 abc
PI-4	77 bc	56 ab	24 a-d	84 b	66 ab	17 abc
PI-5	75 c	57 ab	31 a-d	80 b	48 ef	18 ab
$\bar{x}$ [2] =	84 a	58 b	29 c	84 a	52 b	13 c

[1] Genotype means followed by a common letter in a column are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test; analysis on angular transformation of %.

[2] Column means followed by a common letter in the row within years are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test.

produced significant differences in defoliation both years, with more frequent application of chorothalonil reducing defoliation in all genotypes. Florunner gave the greatest response to fungicide application with PI-5 and PI-6 being the least responsive. The mean percent defoliation values for fungicide treatment were similar between years, except for the 10-day treatment being much lower in 1980 than in 1979. The percent defoliation was intermediate for the 20-day treatment, being more than expected considering CP lesion counts.

All genotypes gave some pod yield response to fungicide application (Table 5). Florunner clearly gave the greatest response to increase fungicide application. All unsprayed entries had yields over 3000 kg/ha one or both years except for PI-4, PI-5, BL-1, and Florunner. BL-10 gave the highest unsprayed yields at 4285 kg/ha in 1979,

Table 5. mean pod yields for each genotype (cultivar, breeding lines - BL; and plant introductions - PI) sprayed with chlorothalonil (10 or 20 day interval) and unsprayed in 1979 and 1980.

Genotype	Pod yields (kg/ha)					
	1979 Fungicide Treatment			1980 Fungicide Treatment		
	Unsprayed	20-day	10-day	Unsprayed	20-day	10-day
BL-10	4285 a[1]	4598 a	5062 a	3435 ab	4301 a	4736 a
BL-2	3814 ab	4289 ab	4253 b	3659 a	4078 a	4375 a
BL-8	3757 abc	4106 abc	4033 bc	3370 ab	4391 a	4391 a
BL-12	3476 bc	3875 bc	3972 bc	3126 b	3618 b	3724 bc
BL-9	3456 bc	3883 bc	3667 bc	3659 a	4350 a	4395 a
BL-3	3452 bc	3854 bc	4029 bc	3387 ab	4090 a	3996 b
PI-6	3204 cd	3570 c	3631 c	2313 c	2716 de	2931 de
BL-11	3183 cd	4110 abc	4216 bc	2212 c	3151 c	3232 d
BL-1	2781 de	3740 bc	4139 bc	2187 c	2984 cd	3610 c
PI-5	2492 c	3049 d	2943 d	2155 c	2403 e	2700 e
Florunner	2443 ef	4228 ab	5541 a	2090 c	3631 b	4391 a
PI-4	1923 f	2175 e	2029 e	1488 d	1931 f	1996 f
$\bar{x}$ [2] =	3189 b	3790 a	3960 a	2757 c	3470 b	3706 a

[1] Means followed by a common letter in column are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test.

[2] Means followed by a common letter in the row within years are not significantly different at P = 0.05 according to Duncan's New Multiple Range Test.

even though only BL-1 and Florunner had CP lesion counts as high. Several genotypes gave little or no additional yield response when the fungicide schedule was intensified from 20- to 10-days. Sisterlines BL-3 and BL-9 responded similarly. Since the entire test was dug on the same day each year, some entries, especially Florunner, were not harvested at peak pod yield potentials within the unsprayed treatments, thus probably exaggerating yield differences. PI-4 apparently has the lowest yield potential and is affected the least by leafspot (CP) of the genotypes tested, even though it was not among the most resistant entries according to lesion count data. BL-10 apparently has a high level of tolerance to CP since it had the highest pod yield in 1979 and near the highest in 1980 (unsprayed treatment) even with large lesion counts.

Correlation coefficients between pairs of variables, pod yields, CP lesion counts, and defoliation, were very highly significant (Table 6). All CP lesion count data and percent defoliation showed highly significant negative correlations with pod yield. CP lesion counts at 110 and 130 days showed strong positive correlations with defoliation. This relationship reflects that CP was the dominant pathogen and the primary factor in defoliation.

Table 6. Correlation of disease asessment variables of *Cercosporidium personatum* (CP) with each other.

Pair of Variables	R[1]	
	1979	1980
Yield vs CP-90[2]	-0.31	-0.31
Yield vs CP-110	-0.31	-0.46
Yield vs CP-130	-0.34	-0.32
Yield vs percent defoliation	-0.32	-0.48
Percent defoliation vs CP-110	0.80	0.71
Percent defoliation vs CP-130	0.81	0.83

[1] All values statistically significant at P = 0.001.

[2] CP = 1979: $(\text{No. CP lesions/leaflet} + 1)^{\frac{1}{2}}$

1980: $[(\text{No. CP lesions} + 1)/\text{cm}^2 \text{ of leaf area}]^{\frac{1}{2}}$

The correlations between CP lesion counts and yield were negative and highly significant, but not of the magnitude noted for defoliation and CP lesion counts. However, numerous other factors effect yield, including inherent genetic differences between genotypes and relative stage of maturity of the crop at harvest. The genotypes in these tests represented a range of maturities and pod yield potentials. However, no extremely late maturing or poor yielding genotypes were included in these tests.

The ultimate value of resistance or tolerance to CA and/or CP or any pathogen is stabilization of pod yields by minimizing yield losses due to leafspot while reducing fungicide use. Although genotypes in this study showed marked improvement in disease control and at least some pod yield response with chlorothalonil application, there was little additional yield response or reduction in number of lesions when comparing the 10-day to the 20-day schedule for most of the lines, with the exceptions of

Florunner, BL-1, and BL-10. A number of the genotypes tested have pod yield potentials exceeding 3000 kg/ha, even when CP is not controlled with a fungicide. Some entries produced yields exceeding 4000 kg/ha with a moderate 20-day fungicide program. This offers the possibility of saving production cost, using less fungicide, reducing energy use, and reducing the risk of losing the crop to CP leafspot. It therefore appears feasible to control peanut leafspot using sources of resistance presently available in conjunction with less intensive fungicide programs than currently employed.

Further studies are needed to determine the mechanisms of resistance to leafspot that are involved, i.e., resistance to infection, lengthening of the latent period, reduction of sporulation, resistance to defoliation, and possibly others. This information could help the breeder, pathologist, and agronomist in more fully utilizing this germplasm.

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