Relationship Between Components of Resistance and Disease Progress of Early Leaf Spot on Virginia-Type Peanut

C. S. Johnson, M. K. Beute, and M. D. Ricker

Former graduate research assistant, professor, and former graduate research assistant, respectively, Department of Plant Pathology, North Carolina State University, Raleigh 27695-7616.

Journal Series Paper 9855 of the North Carolina Agricultural Research Service, Raleigh 27695-7601.

Use of trade names in this publication does not imply endorsement by the North Carolina Agricultural Research Service of the products named, or criticism of similar ones not mentioned.

This work supported in part by the Peanut CRSP, USAID Grant DAN-4048-G-SS-2065-00. Recommendations do not represent an official position or policy of USAID.

The authors thank Joyce Hollowell, Barbara Shew, Steve Dean, Chris Mundt, Raymond Coltrain, and Steve Herlocker for hours of help.

Accepted for publication 6 November 1985.

ABSTRACT

Johnson, C. S., Beute, M. K., and Ricker, M. D. 1986. Relationship between components of resistance and disease progress of early leaf spot on Virginia-type peanut. Phytopathology 76:495-499.

Early leaf spot (caused by *Cercospora arachidicola*) was monitored in isolated field plots of 20 Virginia-type peanut (*Arachis hypogaea*) lines in 1982–1984. Increases in the percentage of diseased leaflets and percent defoliation fit the Gompertz model more closely than the logistic or monomolecular models. Rates of increase in leaf spot incidence and defoliation as well as areas under disease progress curves (AUDPCs) varied among peanut genotypes. Peanut genotypes with larger AUDPCs had faster rates of disease increase. Florigiant, NC 2, and selected lines from crosses of NC 5 × Florigiant and crosses of NC 2 × NC 5 were more susceptible to early leaf spot than the other entries tested. GP-NC 343 and a selected line from GP-NC 343 × NC 5 exhibited the lowest disease levels. Disease levels at 103–110 days after planting contributed most to AUDPCs. AUDPCs were more highly correlated with latent period, percent lesions sporulating, spore production, and time to defoliation than were infection and defoliation rates. The maximum percentage of lesions that sporulated was the resistance component most highly correlated with disease progress in the field.

Additional key words: components of resistance, epidemiology, horizontal resistance.

Cercospora arachidicola Hori causes early leaf spot of peanut (*Arachis hypogaea* L.), one of the most ubiquitous and economically significant peanut diseases (6). Losses attributed to this disease commonly range from less than 1% to more than 50%. Recommended control measures include crop rotation (12), burying peanut debris by deep plowing in the spring (6), and fungicides (25).

Complete resistance to early leaf spot has not been found among the various species of *Arachis* (6,16). Consequently, resistance to early leaf spot has not played a significant role in early leaf spot control strategies to date. Differences in partial resistance to early leaf spot, however, have been observed among peanut genotypes. Some genotypes develop fewer lesions or less affected leaf area per leaf (2,9,10,15,20,26). Reduced sporulation per lesion (1,5,7,17,21) and percentage of lesions sporulating (21) as well as increased latent period (5,17,21), and increased time to defoliation (21) have also been found.

As a prelude to this study, components of partial resistance for the 20 genotypes used in these studies were recently quantified (21) with detached leaf tests in the greenhouse, which have been shown to produce results generally correlated with field evaluations (9). The purpose of this study was to assess correlations between greenhouse assessments of components of leaf spot resistance and quantitative descriptions of leaf spot epidemics in the field. The objectives of this work were to determine the relative contribution of different components of early leaf spot resistance to slowing disease progress in field plots of Virginia-type peanuts, and to identify criteria that could improve the efficiency of breeding programs for selection of resistance to early leaf spot.

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

MATERIALS AND METHODS

Experiments were conducted at the Peanut Belt Research Station near Lewiston, NC, in 1982, at the Upper Coastal Plain Research Station near Rocky Mount, NC, in 1983, and again at the Peanut Belt Research Station in 1984. Experimental material was obtained from the parents and progeny of a six-parent complete diallel (11). Individual genotypes were selected based on disease ratings made near the end of the growing season in the F_5 generation. Genotypes were selected to obtain a range of resistance to early leaf spot. Three commercial cultivars and two germ plasms were chosen: Florigiant, NC 2, NC 5, NC 3033, GP-NC 343, as well as two entries each from crosses of Florigiant × NC 5, Florigiant × GP-NC 343, NC 2 × NC 5, NC 3033 × GP-NC 343, GP-NC 343 × NC-Ac 3139, GP-NC 343 × NC 2, and GP-NC 343 × NC 5, and a single entry from NC 3033 × NC 2.

Plots were planted on 7 May in 1982, and on 18 May in 1983 and 1984. A randomized complete block design was used with seven replications. Plots consisted of four 11-m-long rows spaced 0.91 m apart. Plots were separated from each other on the ends by two 3.0-m strips of fallow ground and a 9.2-m strip of field corn planted between the fallow areas. Plots were isolated at the sides by 8.2 m of field corn. Corn was planted on 20, 29, and 27 April in 1982–1984, respectively, and was approximately 2.0 m tall by the time leaf spot was first observed.

Standard cultural practices were followed each year, with the exception that no leaf spot fungicides were applied. Preplant herbicides (alachlor, benefin plus vernolate, and naptalam), a nematicide (ethoprop), and insecticides (carbaryl and dicofol) were applied as recommended by the North Carolina Cooperative Agricultural Extension Service. Pentachloronitrobenzene (quintozene) was also applied as 10% granules at 112 kg/ha to all plots in 1982 and 1984 at about 14 and 10 wk after planting, respectively, for control of southern stem rot.

Beginning in late June or early July, the percentage of diseased leaflets was estimated every 4–14 days from counts of the number of

leaflets containing at least one lesion in randomly selected, 61-cm lengths of row in each of the center two rows of each plot. Final assessments were made 2–30 days before harvest (128–140 days after planting). The 61-cm "rating segments" were randomly selected prior to each assessment date. Each rating segment was assessed by two assessors. Two rating segments per row were assessed in 1982; one rating segment per row was assessed in 1983 and 1984. Percent defoliation was assessed by subdividing the selected rating segments into four 15.2-cm row lengths. One 15.2-cm "rating unit" per 61-cm rating segment was randomly selected each week. The number of nodes and the number of missing leaflets were counted on one stem from each side of the row within each rating unit.

Averages of percent defoliation and diseased leaflets for each plot on each observation date were used for subsequent data analysis. Field data were transformed by using the Gompertz model to stabilize variances prior to statistical analysis. The Gompertz (3), logistic (28), and monomolecular (28) models were fitted to the data. Apparent rates of disease increase were estimated from slopes obtained by regressing transformed disease data against time. Time was expressed as the number of days after planting. Residual plots were examined for systematic patterns for each regression. Statistically significant differences in infection rates were identified by using the full- versus reduced-model method (4,27).

Areas under disease progress curves (AUDPCs) were calculated for percent diseased leaflets (AUDPC-DL) and percent defoliation (AUDPC-DF) by using Shaner and Finney's method (24):

$$\text{AUDPC} = \Sigma\,[(Y_{i+1} + Y_i)/2]\,(X_{i+1} - X_i)$$

in which Y_i = percent diseased or defoliated leaflets at the ith observation and X_i is the time of the ith observation in days after planting. Differences in AUDPCs were examined by using analyses of variance and orthogonal contrasts. A stepwise regression procedure (19) was used to identify which disease observations were most influential in the development of AUDPCs.

Relative values of components of resistance to *C. arachidicola* were obtained from detached leaf tests conducted by Ricker et al (21) in the greenhouse (Table 1). Lesions per leaf (LN), sporulation per lesion (SPL), the maximum percentage of lesions per leaf found to be sporulating within 30 days of inoculation (MPLS), time in days between inoculation and defoliation (DEFT), and latent period had been determined for the same twenty genotypes used in this study. Latent period was estimated as the number of days between inoculation and the first date on which at least two sporulating lesions per leaf were observed (T2), as well as by using Shaner's probit regression method (23) to estimate the number of days until 50% of the lesions sporulated (T50).

Pearson's correlation coefficient (19) was used to examine associations among infection rates, AUDPCs, and components of resistance to peanut leaf spot. The Waller-Duncan procedure was used to group peanut lines according to their AUDPCs. A stepwise regression procedure (19) was used to identify which resistance components best predicted resistance in the field.

RESULTS

AUDPCs were significantly larger in 1984 than in 1982 and 1983 (Table 2). AUDPCs were smaller in 1983 than in 1982 or 1984. Differences in AUDPCs were also observed among peanut genotypes. Significant ($P = 0.01$) differences in AUDPCs were obtained even between entries resulting from crosses of the same parents. Florigiant, NC 2, entries of NC 2 × NC 5, and entries of NC 5 × Florigiant exhibited larger AUDPCs than did the other lines tested. NC 3033 had the smallest area under the defoliation curve (AUDPC-DF) in 1982 and 1984, followed by entries of Florigiant × GP-NC 343. GP-NC 343 and entry 86 (a selection from the cross of GP-NC 343 × NC 5) had the smallest area under the disease curve (AUDPC-DL).

The single daily assessments for percent diseased leaflets that were most closely correlated with variation in AUDPC-DL were at 103 days after planting in both 1982 and 1984. Assessments made 103 days after planting explained 90% of the variation in AUDPC-DL in 1982 and 78% of the variation in AUDPC-DL in 1984. When all assessments made during August were considered together, they explained more than 95% of the variation in AUDPC-DL in 1982 and 1984.

The individual estimates of percent defoliation that explained the greatest proportion of the variation in AUDPC-DF were those made 122 (87%) and 110 days (72%) after planting in 1982 and 1984, respectively. Estimates made 103, 114, and 122 days after planting (20 August, 31 August, and 9 September) in 1982 accounted for 96% of the variation in AUDPC-DF when considered together.

TABLE 1. Components of resistance to early leaf spot estimated from detached leaf tests conducted in greenhouse moisture chambers by Ricker et al (21). The same 20 peanut genotypes used in the greenhouse study were also used in field tests conducted in 1982–1984 to measure disease progress of early leaf spot

Genotype	Entry	Spores per lesion	Lesion number per leaf	Latent period (T2)[a]	Latent period (T50)[b]	Maximum lesions sporulating (%)	Time to defoliation (days)
Florigiant	81	2,639	131.4	19.38	22.00	68.45	42.14
NC 2 × NC 5	21	1,204	82.0	18.44	22.59	62.38	48.42
NC 2 × NC 5	8	903	63.5	20.38	22.90	56.56	52.08
NC 5 × Florigiant	65	1,319	118.1	17.81	21.78	57.09	43.08
NC 5 × Florigiant	66	787	134.7	17.88	21.74	68.93	42.97
NC 2	1	1,111	111.3	18.06	22.37	76.94	48.33
GP-NC 343 × NC Ac 3139	75	764	60.8	21.88	24.34	36.89	58.31
GP-NC 343 × NC 2	57	1,412	84.8	19.25	23.47	60.33	49.28
NC 3033 × NC 2	64	1,019	122.1	20.56	24.00	49.57	52.52
GP-NC 343 × NC 2	58	972	83.4	20.75	23.65	56.09	45.92
GP-NC 343 × NC 5	88	486	72.8	20.75	22.44	58.05	58.69
GP-NC 343 × NC Ac 3139	79	926	110.3	20.13	23.04	52.08	46.86
NC 3033	12	857	96.6	24.44	26.25	35.45	67.08
NC 5	3	370	89.8	19.75	22.15	38.88	52.48
Florigiant × GP-NC 343	44	810	100.4	19.69	22.80	55.55	45.94
Florigiant × GP-NC 343	50	880	76.9	21.50	23.26	43.10	59.61
NC 3033 × GP-NC 343	102	787	145.6	20.00	22.67	38.52	55.69
NC 3033 × GP-NC 343	104	1,042	96.8	18.69	22.62	53.32	44.94
GP-NC 343	336	579	93.9	21.38	24.41	33.62	45.41
GP-NC 343 × NC 5	86	972	72.9	20.31	23.17	36.78	54.83

[a] T2 = the number of days between inoculation and when at least two lesions per leaf are first observed to be sporulating.
[b] T50 = latent period estimated by using Shaner's probit regression method (23).

Assessments made 103, 110, and 124 days after planting (20 August, 27 August, and 12 September) explained 95% of the variation in AUDPC-DF in 1984 when considered together.

The Gompertz model generally produced slightly higher coefficients of determination (R^2) and more random plots of standardized residuals versus time than did the logistic and monomolecular models. Rates of disease increase were, therefore, estimated and compared by using the Gompertz model. Goodness-of-fit of the models to disease progress data, however, appeared to vary from one peanut line to another. Coefficients of determination were higher and residual plots more random for susceptible than for resistant genotypes.

Examination of plots of nontransformed disease progress curves indicated that epidemics began at the same time on all genotypes. Maximum disease severities varied among peanut genotypes, as did the dates on which these peak severities of disease were observed. Final disease serverities also differed among the genotypes tested, but to a lesser extent than at earlier observations.

Infection rates were similar in 1982 and 1984 (Table 3). Defoliation rates, however, were greater in 1982 than in 1984. Defoliation and infection rates were smaller in 1983 than in 1982 or 1984. Differences in rates of both defoliation and infection were observed among the peanut genotypes studied. Florigiant, NC 2, entries of NC 2 × NC 5, and entries of NC 5 × Florigiant exhibited the largest rates of defoliation and infection in 1982. Percent diseased leaflets increased more slowly on NC-GP 343 and NC-GP 343 × NC 5 (entry 86) than on the other lines tested. Percent defoliation increased most slowly on NC 3033 and Florigiant × GP-NC 343 (entry 50). Florigiant, NC 2, NC 5, NC 3033, and GP-NC 343 as a group had higher infection and defoliation rates ($P = 0.01$) than those observed on the crosses between them.

AUDPC-DL was correlated ($P = 0.01$) with SPL and with the

TABLE 2. Mean area under disease progress curves for percent diseased leaflets (% Dis) and percent defoliation (% Def) from field tests conducted in North Carolina in 1982–1984 to measure disease progress of early leaf spot

Genotype	Entry	1982		1983		1984	
		% Dis	% Def	% Dis	% Def	% Dis	% Def
Florigiant	81	4,528.6	22.01	868.6	2.91	5,139.5	24.44
NC 2 × NC 5	21	4,305.4	21.79	137.0	2.45	4,485.9	22.01
NC 2 × NC 5	8	4,115.8	18.26	44.7	3.09	3,244.1	14.27
NC 5 × Florigiant	65	3,803.5	19.88	162.8	2.47	3,715.7	18.21
NC 5 × Florigiant	66	3,751.9	19.62	55.8	3.21	4,052.1	20.24
NC 2	1	3,620.7	16.65	232.8	1.89	4,866.8	20.74
GP-NC 343 × NC Ac 3139	75	3,410.3	12.70	106.9	2.56	3,445.5	13.60
GP-NC 343 × NC 2	57	3,137.2	12.54	84.4	2.16	3,593.7	14.18
NC 3033 × NC 2	64	2,973.9	13.71	33.8	3.38	3,345.3	14.49
GP-NC 343 × NC 2	58	2,973.4	14.81	63.4	2.24	2,880.7	14.05
GP-NC 343 × NC 5	88	2,963.7	13.91	57.6	3.81	2,749.6	15.07
GP-NC 343 × NC Ac 3139	79	2,914.0	13.89	38.3	3.07	2,976.5	14.76
NC 3033	12	2,701.5	9.36	91.0	2.30	3,617.5	12.19
NC 5	3	2,692.1	12.88	62.3	3.03	2,943.9	14.28
Florigiant × GP-NC 343	44	2,672.6	11.11	39.7	3.06	3,294.9	13.17
Florigiant × GP-NC 343	50	2,666.4	10.59	35.2	2.65	3,466.5	12.97
NC 3033 × GP-NC 343	102	2,652.9	11.41	60.5	2.44	3,158.0	14.51
NC 3033 × GP-NC 343	104	2,502.3	11.66	66.1	3.14	2,659.1	14.20
GP-NC 343	336	1,942.8	11.10	52.1	3.22	1,895.5	13.26
GP-NC 343 × NC 5	86	1,789.4	11.80	52.6	3.65	2,672.1	16.43
Waller-Duncan LSD (K-ratio = 100)		559.1	2.58	229.1	2.01	426.8	2.26

TABLE 3. Rates of increase[a] in percent diseased leaflets and percent defoliation for 20 peanut genotypes in North Carolina in 1982–1984

Genotypes	Entry	Diseased leaflets (%)		Defoliation (%)		
		1982/1984[b]	1983	1982	1983	1984
Florigiant	81	0.0666	0.0216	0.0418	0.0132	0.0355
NC 2 × NC 5	21	0.0593	0.0083	0.0425	0.0084	0.0314
NC 2 × NC 5	8	0.0502	0.0040	0.0374	0.0105	0.0220
NC 5 × Florigiant	65	0.0577	0.0079	0.0394	0.0093	0.0287
NC 5 × Florigiant	66	0.0612	0.0020	0.0387	0.0083	0.0311
NC 2	1	0.0562	0.0121	0.0353	0.0107	0.0351
GP-NC 343 × NC Ac 3139	75	0.0475	0.0073	0.0302	0.0084	0.0249
GP-NC 343 × NC 2	57	0.0454	0.0050	0.0284	0.0083	0.0233
NC 3033 × NC 2	64	0.0455	0.0025	0.0306	0.0088	0.0211
GP-NC 343 × NC 2	58	0.0408	0.0048	0.0318	0.0105	0.0246
GP-NC 343 × NC 5	88	0.0442	0.0048	0.0318	0.0131	0.0241
GP-NC 343 × NC Ac 3139	79	0.0429	0.0036	0.0318	0.0107	0.0266
NC 3033	12	0.0463	0.0059	0.0253	0.0082	0.0244
NC 5	3	0.0451	0.0046	0.0293	0.0126	0.0242
Florigiant × GP-NC 343	44	0.0437	0.0018	0.0274	0.0069	0.0251
Florigiant × GP-NC 343	50	0.0498	0.0022	0.0268	0.0072	0.0210
NC 3033 × GP-NC 343	102	0.0457	0.0013	0.0276	0.0070	0.0264
NC 3033 × GP-NC 343	104	0.0402	0.0041	0.0281	0.0061	0.0230
GP-NC 343	336	0.0325	0.0034	0.0269	0.0111	0.0217
GP-NC 343 × NC 5	86	0.0372	0.0037	0.0291	0.0118	0.0241

[a] Rates of increase were obtained by regressing Gompertz-transformed disease assessments against time (expressed as days after planting).

[b] Rates of increase in percent diseased leaflets were not significantly different in 1982 and 1984. Infection rates are, therefore, presented for combined 1982 and 1984 data.

MPLS (Table 4). Reductions in AUDPC-DL, however, were correlated ($P = 0.01$) only with MPLS. AUDPC-DF was also correlated ($P = 0.01$) positively with SPL and with MPLS. Significant negative correlations were observed between AUDPC-DF and length of latent period (estimated as T2 and T50), as well as with DEFT. Reductions in AUDPC-DF were correlated with increased latent period (T2 and T50), decreased MPLS, and decreased DEFT. MPLS correlated more highly with both AUDPC-DL and AUDPC-DF than did the other estimated components of peanut leaf spot resistance. Approximately 50% of the variation in AUDPC-DL and AUDPC-DF could be accounted for by MPLS alone. Approximately 65% of the variation in AUDPC-DL was accounted for by SPL, MPLS, and DEFT. A combination of SPL, T50, and DEFT also accounted for about 65% of the variation in AUDPC-DF.

Infection rates were positively and negatively correlated with MPLS ($P = 0.01$) and latent period (T50), respectively (Table 4). Rates of defoliation were positively correlated ($P = 0.01$) with SPL and MPLS. Negative correlations ($P = 0.01$, $P = 0.01$, and $P = 0.02$) were observed between defoliation rates, latent period (as measured by T2 and T50), and DEFT.

DISCUSSION

Hassan and Beute (9) identified NC 3033, NC Ac 3139, and NC 5 as potentially valuable parent lines for an early leaf spot breeding program and Kornegay et al (11) recommended NC 3033 and GP-NC 343 as the most useful parents. The significant decrease in disease observed between parents and progeny in this study showed that these parent genotypes are indeed valuable sources of partial resistance to early leaf spot in Virginia-type peanut. Of the progeny examined in this study, GP-NC 343 × NC 5 (entry number 86) appeared to be the most promising.

When disease increase occurs may be a critical factor influencing peanut crop losses caused by Cercospora leaf spot and should be considered in evaluations of leaf spot resistance. Although epidemics began at approximately the same time on all lines in both 1982 and 1984, differences in percent diseased leaflets among lines were greater between 95 and 124 days after planting than they were at the final rating taken (138 days after planting; approximately 2 wk before harvest). Many entries exhibited different rates of disease increase and AUDPCs, but still attained final disease severities similar to the standard susceptible cultivar, Florigiant. Peanuts also have a critical period for yield accumulation (8,22). Results of recent work suggest that, under favorable conditions, most of the mature pods harvested are initiated before peak flowering in the crop (22). First flowers usually occur in early July in the peanut-growing area of North Carolina. This time period usually coincides with the time when early leaf spot is first observed. Disease severities often remain at low levels for several weeks. In both 1982 and 1984, disease increase was not observed until about mid-July. Over 90% of all leaflets, however, were diseased on the most susceptible genotypes (Florigiant and NC 2) by the end of August. The development of pods initiated prior to peak flowering also probably occurs from mid-July through August. Much of the yield loss due to early leaf spot, therefore, probably develops during this time. Our results suggest that the critical period for assessing resistance to early leaf spot occurs approximately 103–110 days after planting in North Carolina.

AUDPCs appeared to be better descriptors of resistance to Cercospora leaf spot than estimates of infection or defoliation rate. Rates of disease increase were averages over the course of the entire season, while AUDPCs were calculated from averages of disease over 4- to 14-day intervals. AUDPCs may, therefore, reflect the timing of disease increase more than rates of increase in infection or defoliation. AUDPCs also correlated more highly with components of leaf spot resistance than did infection or defoliation rates. The smaller correlations between rates of disease increase and resistance components may have arisen from the imperfect fit of the Gompertz model to the data and the resulting differences between the "true" rates of increase and those estimated by regression analysis.

The disease progress models did not fit disease data from all peanut genotypes equally well. Although the Gompertz model provided statistically significant fits to disease progress data for all of the peanut genotypes tested, coefficients of determination and residual plots from regression of Gompertz-transformed disease assessments against time varied across peanut lines. There may have been enough variation in "fit" among peanut entries, due to the simplicity of the Gompertz model, to reduce the apparent size of correlations between rates of disease increase and components of leaf spot resistance. These problems might be avoided by using more mathematically explicit models, such as the Weibull model (18), but these models may be too computationally complex to use in evaluating more than a few genotypes. AUDPCs, on the other hand, avoid possible problems arising from imperfect fits of data to statistical models and are relatively easy to calculate.

The proportion of lesions of *Cercospora* that never sporulate appears to be an important parameter of resistance in the peanut genotypes examined in this study. Higher correlations were observed between disease progress and MPLS than with the number of spores produced, either of two estimates of latent period, or with time to leaflet defoliation. MPLS was calculated from counts made after the largest estimated latent period had expired. Its effects on the increase of Cercospora leaf spot, therefore, should be separate from the consequences of latent period. MPLS was also correlated with other components of resistance to Cercospora leaf spot (21), especially latent period. Selection for decreased MPLS, therefore, should also often result in selection for increased latent periods.

TABLE 4. Pearson correlation coefficients relating area under disease progress curves (AUDPCs) and rates of increase in percent diseased leaflets and percent defoliation to estimated levels of components of resistance to early leaf spot of peanut. All data were taken from the same set of 20 peanut genotypes. Disease progress data was obtained from field tests conducted in North Carolina in 1982–1984[a]

Component of resistance[b]	Diseased leaflets (%)			Defoliation (%)			
	AUDPC		Rate of increase	AUDPC		Rate of increase	
	1982	1984	1982/1984[c]	1982	1984	1982	1984
SPL	0.573**	0.672**	0.600**	0.562**	0.662**	0.540**	0.571**
LN	0.165	0.350	0.383	0.247	0.403	0.213	0.485*
T2	−0.430*	−0.382	−0.471*	−0.630**	−0.645**	−0.611**	−0.556**
T50	−0.415	−0.312	−0.494*	−0.604**	−0.599**	−0.596**	−0.505*
MPLS	0.708**	0.680**	0.669**	0.723**	0.702**	0.709**	0.674**
DEFT	−0.324	−0.180	−0.264	−0.562**	−0.511**	−0.523**	−0.437**

[a] Correlation coefficients marked by one or two asterisks are statistically significant at $P = 0.05$ or $P = 0.01$, respectively.

[b] Components of resistance were obtained from detached leaf tests conducted in greenhouse moisture chambers by Ricker et al (21). SPL = spores per lesion. LN = lesions per leaf. T2 = the number of days between inoculation and the first observation of at least two sporulating lesions per leaf. T50 = the number of days between inoculation and the estimated point when 50% of the lesions per leaf are sporulating (23). MPLS = the maximum number of sporulating lesions per leaf observed over a 30-day period, divided by the number of lesions per leaf observed 21 days after inoculation. DEFT = the number of days between inoculation and defoliation.

[c] Rates of increase in percent diseased leaflets were not significantly different in 1982 and 1984. Infection rates are, therefore, presented for combined 1982 and 1984 data.

Melouk et al (14) suggested a leaf spot reaction index similar to MPLS for field screening of peanut genotypes for leaf spot resistance. Determining the degree of sporulation, however, would be a ponderous task in the field. Assessments of percent diseased leaflets or percent defoliation taken at the right time would be easier, faster, and would provide as much relevant information about field performance relative to the other lines being tested. While MPLS may not be practical for screening in the field, it is well suited for use with detached leaf tests under greenhouse moisture chamber conditions.

Leonard and Mundt (13) suggested that breeding programs for partial disease resistance should emphasize selection for decreased sporulation or inoculum efficiency for diseases which increase at a relatively slow rate. Reducing latent period would be more desirable with explosive pathosystems. Ideally, however, the resistant response would include the effects of several mechanisms. Early leaf spot of peanut appears to be an intermediate case in terms of rate of disease increase. MPLS appears to be a distinct effect relative to latent period and inoculum efficiency (number of lesions per leaflet) (21). Latent period and MPLS, however, are frequently correlated. Selection based on MPLS should, therefore, result in genotypes with longer latent periods as well as decreased percentages of sporulating lesions. This resistance should be more durable and effective than resistance acting on a single stage in the pathogen's life cycle.

LITERATURE CITED

1. Abdou, Y. A-M., and Cooper, W. E. 1974. Effect of culture medium and light on sporulation of two peanut-leafspotting fungi, *Cercospora arachidicola* and *Cercosporidium personatum* (Berk. & Curt.) Deighton. Peanut Sci. 1:11-14.
2. Aulakh, K. S., Sandhu, R. S., and Sunas, M. S. 1972. Resistance to "tikka" leafspot in groundnut germplasm. Indian J. Agric. Sci. 42:952-955.
3. Berger, R. D. 1981. Comparison of the gompertz and logistic equations to describe disease progress. Phytopathology 71:716-719.
4. Draper, N. R., and Smith, H. 1981. Applied Regression Analysis. 2nd ed. John Wiley & Sons, New York. 709 pp.
5. Foster, D. J., Beute, M. K., and Wynne, J. C. 1980. Spore production and latent period as mechanisms of resistance to *Cercospora arachidicola* in four peanut genotypes. Peanut Sci. 7:88-90.
6. Garren, K. H., and Jackson, C. R. 1973. Peanut diseases. Pages 429-494 in: Peanuts—Culture and Uses. Am. Peanut Res. and Educ. Assoc., Stillwater, OK. 684 pp.
7. Gobina, S. M., Melouk, H. A., and Banks, D. J. 1983. Sporulation of *Cercospora arachidicola* as a criterion for screening peanut genotypes for leafspot resistance. Phytopathology 73:556-558.
8. Hang, A. N., McCloud, D. E., Boote, K. J., and Duncan, W. G. 1984. Shade effects on growth, partitioning and yield components of peanuts. Crop Sci. 24:109-115.
9. Hassan, H. N., and Beute, M. K. 1977. Evaluation of resistance to Cercospora leaf spot in peanut germplasm potentially useful in a breeding program. Peanut Sci. 4:78-83.
10. Hemingway, J. S. 1954. Cercospora leaf spots of groundnuts in Tanganyika. E. Afr. Agr. J. 19:263-271.
11. Kornegay, J. L., Beute, M. K., and Wynne, J. C. 1980. Inheritance of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in six Virginia-type peanut lines. Peanut Sci. 7:4-9.
12. Kucharek, T. A. 1975. Reduction of Cercospora leaf spots of peanut with crop rotation. Plant Dis. Rep. 59:822-823.
13. Leonard, K. J., and Mundt, C. C. 1983. Methods for estimating epidemiological effects of quantitative resistance to plant diseases. J. Theoret. Appl. Genet. 67:219-230.
14. Melouk, H. A., and Banks, D. J. 1984. Assessment of resistance to *Cercospora arachidicola* in peanut genotypes in field plots. Plant Dis. 68:395-397.
15. Monasterios, T., Jackson, L. F., and Norden, A. J. 1978. Reaction of peanut *Arachis hypogaea* L. genotypes to two Cercospora leafspot diseases. Proc. Am. Peanut Res. Educ. Soc. 10:64.
16. Nevill, D. J. 1979. An investigation of the disease reaction to *Cercosporidium personatum* in *Arachis hypogaea*. Trop. Legume Bull. 15:18-22.
17. Nevill, D. J. 1981. Components of resistance to *Cercospora arachidicola* and *Cercosporidium personatum* in groundnuts. Ann. Appl. Biol. 99:77-86.
18. Pennypacker, S. P., Knoble, H. D., Antle, C. E., and Madden, L. V. 1980. A flexible model for studying plant disease progression. Phytopathology 70:232-235.
19. Ray, A. A. 1982. SAS User's Guide: Statistics 1982 Edition. SAS Institute, Inc., Cary, NC. 584 pp.
20. Reyes, G. M., and Romasanta, R. 1940. Varietal susceptibility of peanuts to blackspot (*Cercospora personata* (B. & C.) Ell. & Ev.). Philippines J. Agric. 11:371-381.
21. Ricker, M. D., Beute, M. K., and Campbell, C. L. 1985. Components of resistance in peanut to *Cercospora arachidicola*. Plant Dis. 69:1059-1064.
22. Santos, R. B., and Sutton, B. G. 1983. Effect of defoliation on reproductive development of the peanut. Austr. J. Agric. Res. 34:527-535.
23. Shaner, G. 1980. Probits for analyzing latent period data in studies of slow-rusting resistance. Phytopathology 70:1179-1182.
24. Shaner, G., and Finney, R. E. 1977. The effect of nitrogen fertilization on the expression of slow-mildewing resistance in Knox wheat. Phytopathology 67:1051-1056.
25. Smith, D. H., and Littrell, R. H. 1980. Management of peanut foliar diseases with fungicides. Plant Dis. 64:356-361.
26. Sowell, G., Smith, D. H., and Hammons, R. D. 1976. Resistance of peanut introductions to *Cercospora arachidicola*. Plant Dis. Rep. 60:494-498.
27. Steel, R. G. D., and Torrie, J. H. 1980. Principles and Procedures of Statistics, a Biometrical Approach. 2nd ed. McGraw-Hill, New York. 633 pp.
28. Vanderplank, J. E. 1963. Plant Diseases: Epidemics and Control. Academic Press, New York. 349 pp.