Inheritance of Resistance in Corn (*Zea mays*) to Gray Leaf Spot

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ABSTRACT

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All possible crosses (including reciprocals) were made among eight corn inbred lines to study the inheritance of resistance to gray leaf spot caused by *Cercospora zeae-maydis*. Because of cold injury to the developing seeds of two inbreds, seeds from only 40 of the 56 possible crosses were planted in three-replication tests at two Pennsylvania locations. Entries at one location were planted without tillage into corn debris from the previous several years where adequate levels of natural inoculum of *C. zeae-maydis* were present. The same entries at the second location were planted without tillage into soybean stubble and were inoculated with a conidial suspension of *C. zeae-maydis*. Seeds of the inbreds were planted in separate three-replication tests at each location. Area under the disease progress curve (AUDPC) values were calculated for each entry from data collected on three dates as percent leaf area exhibiting symptoms of gray leaf spot. Analysis of variance procedures appropriate to this diallel (Griffing model I) were carried out on the AUDPC with the data from locations combined. General combining ability (GCA) effects were 18 times larger than specific combining ability (SCA) effects indicating that, for this set of inbred lines, additive gene action is more important than nonadditive gene action in controlling resistance to gray leaf spot. Reciprocal effects were significant, but the mean squares were small compared with the mean squares for GCA and SCA. Based on estimated GCA effects, inbreds Pa875, Va59, and B68Ht contributed significantly to resistance. Limited disease development on the inbreds permitted only one estimate of disease severity. A mean separation test supported the ranking of inbreds obtained through the diallel analysis. Based on the results obtained with these inbreds, breeders attempting to accumulate gray leaf spot resistance should use programs that favor additive gene action.

Additional keywords: *Cercospora zeae-maydis*, horizontal resistance, quantitative genetics, rate-reducing resistance.

Gray leaf spot (GLS) of corn, caused by *Cercospora zeae-maydis* Tehon and Daniels, was first identified in 1925 from corn leaves collected in Alexander County, IL (9). The disease remained relatively unimportant until the early 1970s when the number of reports increased (5). Yield losses were associated with gray leaf spot throughout the corn-growing regions of the southern Appalachian Mountains. The disease has been found throughout the mid-Atlantic region and recently was found in southern New York (G. C. Bergstrom, *personal communication*). Significant amounts of gray leaf spot also have been reported in the corn belt (12).

Roane et al (7) reported higher levels of GLS development in fields of continuous no-till corn. The use of no-till and other conservation-tillage methods results in large amounts of debris from the previous year's crop remaining on the soil surface. *Cercospora zeae-maydis* overwinters in corn debris, which then serves as a source of inoculum. Observations throughout the mid-Atlantic region support the earlier report (7) of increased GLS severity in fields planted with conservation-tillage methods (1,5). Early initiation of the disease plus favorable weather conditions, such as warm temperatures and extended periods of high humidity, can result in yield losses as high as 20% (1,4).

Obvious control measures for GLS include eliminating the debris from the previous corn crop by moldboard plowing or avoiding the inoculum by rotation to another crop. In Pennsylvania, many of the areas where GLS is a major problem are subject to severe soil erosion, consequently, conservation-tillage practices are preferred. In addition, a large percentage of these farms are dairy farms that traditionally use corn as a major source of feed. In most cases, the cost per unit of growing corn is less under a no-till system. Therefore, varieties that limit disease development and minimize losses are the most practical and economical control of this disease.

Extensive testing of commercially available corn varieties adapted to Pennsylvania suggests that inadequate levels of resistance to GLS are available (1). Nearly all varieties currently marketed in the state will develop high levels of disease under the proper conditions. Some varieties, however, consistently develop less disease than others. We hypothesize that these varieties possess rate-reducing resistance (6) because less disease develops even though there can be numerous susceptible-type lesions on these varieties. There are some inbred lines available that exhibit fleck-type lesions when inoculated with *C. zeae-maydis* (5) but, to date, the genetic mechanism controlling this reaction has not been defined.

Preliminary evidence suggested that several inbreds in our program possess quantitative resistance (1). A recent report of research conducted concurrently with our study suggests that inheritance of resistance to gray leaf spot was predominantly additive in the group of inbreds tested (10). The purpose of our study was to determine the mode of inheritance of resistance in a group of corn inbred lines mated in a diallel fashion.

MATERIALS AND METHODS

Genetic material. Eight corn inbreds were grown at the Rock Springs Research Farm in 1984 in order to make all possible crosses. The inbreds used in this study were selected based on previous performances in tests for resistance to GLS (1). Pa875, B68Ht, Va59, and Pa887P were considered to be more resistant than Pa77-26, Pa76-22, H93, and H84. B68Ht and Pa77-26 are late-maturing lines; consequently, some crosses were not obtained with these two inbreds. An attempt to make the missing crosses in a winter nursery in Florida failed because cold temperatures in early January killed the developing seed. Therefore, only 40 of the 56 possible crosses (including reciprocals) resulted in enough seed to test in the field. The 16 missing crosses all included either B68Ht or Pa77-26.

Field plots. The 40 crosses were planted in a randomized complete block design with three replications at two Pennsylvania

locations, Franklin County and Cumberland County, on 23 April and 4 May 1985, respectively. To remove the inbreds from competition with crosses, they were planted, adjacent to the crosses, in a randomized complete block design with three replications at both locations. Tests were machine planted in rows 6.08 m in length and with a between-row spacing of 0.76 m. Thirty-five kernels were planted in each row and the resulting plants were thinned to a population of 59,300 plants/ha. Seeds from several of the crosses were limited, so the F_1's were planted in single-row plots. Coefficients of variation for disease reaction from previous studies at the Franklin County location typically range from 15 to 20%, so single-row plots were deemed adequate for this study. The inbreds were planted in two-row plots.

The tests at Franklin County were planted without tillage (no-till) into a field planted with corn the previous several years. High levels of gray leaf spot were observed in the field the previous year so substantial natural inoculum was present. At Cumberland County, the tests were planted without tillage in a field that was planted with soybeans the previous year. The Cumberland County plots were artificially inoculated because inoculum was not present in the field. Gray leaf spot was observed in surrounding fields in 1984 and prior years.

Inoculum production. Inoculum was produced from a mixture of four isolates of *C. zeae-maydis* collected from separate fields in Franklin County in 1984. They were not tested for differences in virulence. Isolates were stored on a strip of V-8 juice agar (300 ml of V-8 vegetable juice, 700 ml of double-distilled water, 3 g of $CaCO_3$, and 15 g of flake agar) under 10 ml of sterile double-distilled water in a test tube (2). The tubes were stored in the dark at 4 C. To produce inoculum, the contents of the tubes were ground aseptically with a mortar and pestle. This suspension was poured into a petri plate containing V-8 juice agar and the excess water removed. The cultures were exposed to diurnal (12 hr of light and 12 hr of dark) cool-white fluorescent light at 21 C. After 2 wk, strips of agar containing sporulating cultures were cut and removed from the plate. A strip was turned upside-down into another petri dish containing V-8 juice agar and was streaked across the surface. This dish was then incubated as described above for 12–14 days.

The inoculum was prepared as a conidial suspension of approximately 1×10^5 spores per milliliter in the laboratory before being transported 2 hr to Cumberland County. The conidiospores of *C. zeae-maydis* were dislodged by flooding the dish with double-distilled water and scraping the surface with a glass microscope slide. The suspension was filtered through cheesecloth into a flask and kept on ice until the time of inoculation.

On the evening of 26 June, the single crosses were inoculated by spraying the conidial suspension with a compressed air sprayer into the whorls of the plants. The plants were at the eight-leaf stage with the ninth leaf in the whorl. The inbreds were inoculated on 24 July at a similar growth stage.

Data collection. Disease assessments for the single crosses began at both locations 1 wk after silking and were made on 30 July (1 wk after silking), 15 August (milk stage), and 29 August (dough stage). The inbred tests were rated on 29 August only. At Cumberland County, only two replications were rated in the inbred test because of a severe weed infestation in one replication. Visual estimates of percent leaf area affected by GLS were recorded on six plants per plot for the crosses and inbreds. The six ratings were averaged for further analyses. Area under the disease progress curve (AUDPC) was calculated for each experimental unit with the formula published by Tooley and Grau (11).

Experimental units were harvested with a combine modified for use in research plots. Percent grain moisture at harvest was determined with a grain moisture tester (DICKEY-john Corporation, Auburn, IL). Field weights were adjusted to kilograms per hectare at 15.5% moisture.

Data analysis. Initial plans were to construct a diallel like Griffing's (3) analysis I in which all possible $p(p-1)/2$ crosses, and their reciprocals were included. Because several crosses were missing, methods presented by Searle (8) for analysis of linear models were used to partition the genotype sum of squares into general combining ability (GCA), specific combining ability (SCA), and reciprocal effects. The linear model used in the analysis was:

$$Y_{ijkl} = u + l_i + p_{j(i)} + g_k + g_l + s_{kl} + r_{kl} + lg_{ik} + lg_{il} + ls_{ikl} + lr_{ikl} + e_{ijkl},$$

where u = a constant;

Y_{ijkl} = the observation on the cross between parents k and l in replication j and location i;

l_i = the effect of location i;

$p_{j(i)}$ = the effect of replication j in location i;

g_k = the general combining ability effect of parent k;

g_l = the general combining ability effect of parent l;

s_{kl} = the specific combining ability effect of parents k and l;

r_{kl} = reciprocal effect of parents k and l ($r_{kl} = -r_{lk}$);

e_{ijkl} = a random error term.

Parameters with double letters indicate interaction effects. The model is similar to Griffing's (3), except that location (l), replication (p), and interactions involving locations are included. Sums of squares were computed for the terms in the model in the order in which they appear. Each sum of squares was computed with adjustments for prior fitted terms and ignoring terms that had not been fit.

Although computation of the sums of squares for a diallel with missing observations is a straightforward procedure, calculation of estimates of the parameters and their interpretation is made more difficult by the missing data. Parameters estimated to aid in interpretation of the results included the following:

$$\bar{X}_{\mathrm{GCA}_i} = \hat{u} + \hat{g}_i$$

where $\hat{u}$ and $\hat{g}_i$ are estimates obtained from the least squares solution to the model (8).

A second parameter:

$$\hat{s}^*_{\mathrm{kl}} = \bar{y}_{\cdot\cdot\,\mathrm{kl}} - (\bar{X}_{\mathrm{GCA}_k} + \bar{X}_{\mathrm{GCA}_l})_{12}$$

was calculated as an indicator of specific combining ability. In the above equation, $\bar{y}_{\cdot\cdot\,\mathrm{kl}}$ is the average of the cross between inbred lines k and l and its reciprocal when both were present, and s^*_{kl} is an estimate of deviation of the cross between lines k and l from the average of the two parents. This estimate is similar to but not identical to the estimate of the specific combining ability parameter in complete dialleles.

The estimate of the reciprocal effect was calculated as:

$$\hat{r}_{\mathrm{kl}} = (\bar{Y}\cdot\cdot\mathrm{kl} - \bar{Y}\cdot\cdot\mathrm{lk})$$

Estimates of r_{kl} could not be calculated for those combinations for which the cross or its reciprocal were absent.

Standard errors for the estimated parameters were calculated from the formula for the variance of a linear combination of variables. Estimates that exceeded twice their standard error were judged to be significantly different from zero.

Because of the large number of missing crosses, all crosses involving B68Ht and Pa77-26 were removed from the data set and the analysis completed according to Griffing's (3) analysis I model. There were no missing crosses in this reduced data set.

The inbred lines were analyzed separately from the single crosses with locations combined.

RESULTS

Single crosses. Analysis of variance procedures were carried out on data collected on each of the rating dates as well as the AUDPC. Because significant sources of variation were similar in all analyses and because the AUDPC integrates disease development over the total assessment period, it is the value reported here.

Analysis of variance of the single cross data with locations combined revealed that a significant portion of the variation was attributed to genotypes (Table 1). The average AUDPC was greater for the single crosses at the Franklin County location than

at the Cumberland County location, but the difference was not statistically significant (Table 2).

Mean squares for each of the genetic effects were highly significant, although the GCA mean square was more than 18 times larger than the mean square for SCA or reciprocal effects (Table 1). The location × genotype interaction was significant, with the location × GCA mean square the only significant term when the main effect was partitioned.

General combining ability means for AUDPC and percent diseased tissue for the inbred lines evaluated separately indicated that the most resistant lines in this experiment were B68Ht, Pa875, and Va59 (Table 2). The GCA effects were negative and significantly different from zero for these inbreds. Significant positive GCA effects were associated with H93, H84, Pa76-22, and Pa77-26, suggesting that these inbred lines were the most resistant. With the exception of Pa77-26, the data from the inbred lines analyzed separately supported this conclusion. The analysis of AUDPC of the single crosses indicated a high level of susceptibility for Pa77-26, but the analysis of inbreds grown separately suggested an intermediate level of susceptibility for this inbred. A higher level of susceptibility was indicated for B68Ht in the analysis of single crosses than in the separate analysis of inbred lines. All of the missing crosses in the diallel had either B68Ht or Pa77-26 as one or both parents, and the difference between ranking from the two analyses was undoubtedly affected by the missing crosses.

Significant SCA effects indicated that there were crosses that did not react as expected based on the average performance of the parents. Individual cross means differed significantly from their respective midparent values in 10 of 22 comparisons (Table 3).

TABLE 1. Analysis of variance table for area under disease progress curve (AUDPC) of gray leaf spot on 40 corn single crosses with locations combined

Source	df	Mean square (AUDPC × 10)[a]
Location (L)	1	163.0
Replications in L	4	83.1
Genotypes (G)	39	209.8**
GCA[b]	7	1,006.6**
SCA[c]	14	55.9**
Reciprocal (r)	18	19.7**
L × G	39	9.1*
L × GCA	7	14.7*
L × SCA	14	8.8
L × R	18	7.2
Error	156	5.5

[a] Asterisks (* and **) indicate significance at $p = 0.05$ and $p = 0.01$, respectively.
[b] GCA = general combining ability.
[c] SCA = specific combining ability.

Individual cross means for AUDPC ranged from 125.9 for Va59 × Pa875 to 1026.0 for Pa76-22 × Pa77-26. The difference in ranking of the inbred lines on the basis of the single cross analysis and the inbred analysis was probably caused by the AUDPC value for Pa76-22 × Pa77-26, which had a mean greater than twice the mean of all crosses. The most resistant 16 crosses in the experiment had either Pa875 or Va59 as one of the parents. The three most resistant inbred lines based on the inbred experiment, B68Ht, Pa875, and Va59 (Table 2), appeared as one or both parents in 23 of the 40 crosses in the experiment, and the 22 most resistant crosses were in the set of 23 that involved one of these three parents. All crosses involving the three most resistant parents had means that were not significantly different from or were significantly less than the average of the respective GCA means.

Estimates of reciprocal effects were significantly different from zero in only three of the 18 combinations for which reciprocal effects could be estimated (Table 3). The small significant mean square for reciprocal effects in comparison with the mean squares for GCA and SCA are probably due to the three combinations mentioned above.

Because the location × genotype interaction was significant, AUDPC values from each location were analyzed separately. Significant sources of variation were the same as for locations combined; therefore, the analysis of variance table is not presented here. The location × GCA effect interaction was significant, but the mean square was small compared with the mean square for GCA. Estimated GCA means for the inbreds were similar for the inbreds at each location (Table 2). H84 and Pa76-22 ranked differently in Cumberland County compared with Franklin County, but both had a significant positive GCA effect at each location. The GCA mean of B68Ht was much greater at Franklin County than at Cumberland County, which probably contributed to the significant location × GCA effects interaction. The location × SCA effects and location × reciprocal effects interactions were not significant.

As a check on the effect of missing values on conclusions reached from this diallel analysis, analyses of AUDPC values were conducted on the data set with crosses involving B68Ht and Pa77-26 excluded. The results were similar in that the mean squares associated with the GCA effects were 40 times greater than the mean squares for SCA effects. Ranking of the GCA means for inbreds was the same as in the eight-entry analysis, except that H93 and Pa76-22 had the fourth and fifth highest GCA means, respectively, in the eight-entry analysis and the fifth and fourth highest GCA mean, respectively, in the six-entry analysis.

Mean squares from the analysis of variance for yield of single crosses with locations combined were highly significant for GCA and SCA effects with GCA effects being slightly larger. General combining ability effects of inbred parents on grain moisture at the time of harvest (a measure of maturity) suggested that late

TABLE 2. General combining ability (GCA) means for area under disease progress curve (AUDPC) for gray leaf spot at the Franklin County and Cumberland County locations and for locations combined, GCA effects for yields and percent grain moisture at harvest, and the percent disease on 29 August of corn inbreds used in a diallel crossing scheme

	GCA Means					
	AUDPC					
Inbred	Franklin County	Cumberland County	Locations combined	Yield	Percent grain moisture	Percent disease (29 Aug.)
Pa875	270.4–[y]	205.4–	237.9–	10,091.7+	23.7	1.7 c[z]
Va59	352.9–	331.5–	342.2–	9,094.4+	22.6–	8.7 c
B68Ht	384.9–	451.1	418.0–	8,975.3+	25.4+	5.8 c
Pa887P	535.9	478.2	507.1	9,332.8+	25.1+	20.2 b
H93	582.4+	556.1+	569.3+	8,824.7–	22.3–	40.5 a
H84	608.3+	551.0+	579.7+	8,279.1–	22.1–	33.8 a
Pa76-22	609.5+	546.9+	578.2+	8,429.6–	23.6	32.0 a
Pa77-26	701.3+	629.0+	665.2+	8,172.4–	22.3–	10.3 bc
Mean	505.7	468.7	487.2	8,900.0	23.4	

[y] Pluses (+) and minuses (–) indicate the GCA effects (used to calculate the GCA means) were significantly greater or less, respectively, than zero at $p = 0.05$.
[z] Means are the average of five replications (three at Franklin County and two at Cumberland County). Means followed by the same letter are not statistically different ($k = 100$) as determined by Duncan's (Bayesian) modified significant difference test.

TABLE 3. Mean area under disease progress curve (AUDPC) for gray leaf spot, estimated specific combining ability value (SCA), and reciprocal effect (REC) of corn inbreds used as parents in a diallel analysis

Male parent		Female parent							
		B68-t	Pa77-26	Pa875	Va59	Pa887P	Pa76-22	H93	H84
B68Ht	AUDPC					448.8[a]	474.6		651.1
	REC\SCA[b]					−13.1	−23.5		50.4
Pa77-26	AUDPC				449.5		1,026.0	665.8	740.0
	REC\SCA				−54.1		404.3*[c]	48.6	109.4*
Pa875	AUDPC				125.9	246.6	231.5	318.0	352.2
	REC\SCA				−146.1*	−132.2*	−89.7*	−93.7*	−25.1
Va59	AUDPC			162.0		456.0	487.0	408.4	432.7
	REC\SCA			−36.0		5.3	−8.8	−55.9	−30.8
Pa887P	AUDPC	450.0		233.9	403.9		529.0	617.7	620.1
	REC\SCA	−1.2		12.7	52.0		27.2	87.6*	65.2*
Pa76-22	AUDPC			405.2	415.8	610.7		711.1	691.1
	REC\SCA			−173.7*	71.2	−81.7		122.4*	38.2
H93	AUDPC			301.8	391.2	633.9	681.1		759.7
	REC\SCA			16.2	17.2	−16.2	29.9		162.8*
H84	AUDPC	447.4	723.6	415.2	427.9	596.9	543.0	714.9	
	REC\SCA	203.6*	16.4	−63.0	5.1	23.2	148.1*	44.8	

[a]Values represent the mean of three replications at two locations.
[b]SCA, above the diagonal; REC, below the diagonal. See Materials and Methods for calculation of these terms.
[c]Asterisks (*) indicate that SCA or REC value is significant with significance assumed if the value was twice the standard error.

maturing as well as early maturing genotypes contained resistance genes (Table 2).

DISCUSSION

The use of AUDPC as an indication of disease development (10) over the measurement period provided a useful tool for measuring the resistance of corn inbred lines to GLS. The results of this experiment demonstrated that GCA effects were of major importance. Significant SCA and reciprocal effects were observed also, but these effects were of much less importance than were the GCA effects. The results indicated that additive gene action is much more important in determination of GLS resistance than nonadditive gene action. The research of Thompson et al (10) also supports our conclusion that resistance to GLS is due predominantly to genes with additive effects.

The most resistant inbred lines in this experiment were Pa875, Va59, and B68Ht. Va59 was one of the most resistant inbred lines in the experiment of Thompson et al (10). Previous research on inheritance of GLS resistance at Franklin County (1) indicated that Pa887P has some resistance to GLS. An intermediate level of resistance was suggested in the analysis of the diseased tissue on the inbreds, and an intermediate to near-susceptible reaction was suggested in the analysis of the single crosses. The present experiment did confirm previous research that indicated Pa887P was more resistant than H93, H84, and Pa76-22.

The location × GCA mean square was significant, but the relative ranking of the inbreds, based on GCA effects, changed only slightly between locations. Pa875, Va59, B68Ht, Pa887P, and H93 ranked the same at both locations and Pa77-26 was the most susceptible inbred at both locations (Table 2).

Although the presence of disease undoubtedly affected yield, the GCA means (Table 2) suggested that useful yield genes were in the more resistant lines and that breeding for resistance to GLS would not automatically exclude yield genes. There was a significant correlation ($r = -0.88$) between AUDPC and yield of the single crosses. However, since a nondiseased control was not available, this relationship should be interpreted with caution.

A concern in this experiment is the effect of interplot interference. To minimize interplot interference, three-row experimental units would have been necessary. Although limited seed quantities restricted the size of the experimental units, other factors also impinged on the decision to use one-row experimental units. Reasonably uniform areas are limited in fields in the two areas of the state where the research was carried out, and thus the space available for experiments is restricted. In addition, the primary purpose of this research was to determine the inheritance of resistance to GLS and not to describe epidemics on a group of genotypes. In all probability, interplot interference was a factor, but the results clearly show that we can identify resistant genotypes with the design used. The benefits of conducting this type of research or of selecting improved genotypes in these locations far outweigh the disadvantages of interplot interference.

The good agreement between ranking of GCA means and the means from inbred test indicates that inbred lines with high levels of resistance generally produce hybrids with high levels of resistance. Selections could be made during inbred line development, with susceptible lines discarded as development proceeds. Crosses between resistant inbred lines could then be evaluated for GLS resistance, yield, and other agronomic traits. Specific combining ability was not important enough to indicate that large numbers of specific combinations need be evaluated, and the most resistant crosses would most likely be obtained from the most resistant inbred lines. The specific combining ability noted in this experiment appears to be due to combinations such as Pa77-26 × Pa76-22, where inbred lines with intermediate disease rankings in the inbred test resulted in a very susceptible hybrid.

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