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Inheritance of Gray Leaf Spot Resistance in Corn

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Gray leaf spot disease, caused by *Cercospora zeae-maydis* Tehon and Daniels, has become a significant disease in Iowa corn (*Zea mays* L.) production. Incidence of gray leaf spot has increased with the increased use of conservation tillage practices. The inheritance of resistance to gray leaf spot was studied via use of generation mean analyses for five crosses and via use of 100 S_1 progenies developed from an F_2 population. Experiments were conducted at two locations that included either natural or artificial inoculation with *C. zeae-maydis* spores. Additive and dominance effects were significant in nearly all instances. Heritability for gray leaf spot resistance among S_1 progenies was 0.78. Because resistance seemed to be determined by additive genetic variation, it seems selection for greater resistance to gray leaf spot can be effective. In all instances, the level of gray leaf spot resistance in single-cross hybrids was improved, whether the single-cross hybrid was produced with either one or both parents having resistance. It seems single-cross hybrids will have adequate levels of resistance to gray leaf spot if at least one of the parents has resistance.

INDEX DESCRIPTORS: *Zea mays*, maize, *Cercospora zeae-maydis*, genetic effects, heritability.

Gray leaf spot of corn (*Zea mays* L.) is a disease caused by the fungus *Cercospora zeae-maydis* (Tehon & Daniels) and has become prevalent throughout the U.S. Corn Belt. The increased frequency and severity of gray leaf spot incidence have been attributed to the increased use of conservation tillage practices (Roane et al. 1974, Hilty et al. 1979, Rupe et al. 1982, Beckman and Payne 1983, Stromberg 1984, Ward et al. 1999). Yield losses from disease infection have been reported to be 10 to 25% in problem areas (Ayers et al. 1984) and with use of susceptible hybrids (Smith et al. 1987, Gorman et al. 1997). Yield losses caused by gray leaf spot are due to premature loss of photosynthetic tissue. Severe reduction of photosynthetic tissue during the grain-filling period can cause direct yield losses associated with reduced grain weight (Dodd 1980) and indirect yield reductions because of increases in stalk and root lodging (Ayers et al. 1984).

Results of previous studies of gray leaf spot resistance suggested that resistance is primarily due to additive effects and is highly heritable (Thompson et al. 1987, Huff et al. 1988, Elwinger et al. 1990, Ulrich 1990, Donahue et al. 1991), but dominance effects also are important for gray leaf spot resistance (Elwinger et al. 1990, Gevers et al. 1994). Several quantitative trait loci have been identified with both additive and dominance effects (Bubeck et al. 1993, Saghai Maroof et al. 1996). Most of the information on gray leaf spot was obtained in the eastern and southern areas of the U.S. corn production. The main objective of our study was to determine the inheritance of gray leaf spot resistance in corn for newer inbred lines adapted to Iowa. Specific objectives were to estimate genetic effects for gray leaf spot resistance for five crosses with use of a generation mean analysis and to estimate the heritability for gray leaf spot resistance with the evaluation of 100 S_1 progenies in the F_2 generation for the B79 × B98 cross.

METHODS

Two experiments were conducted to study resistance to gray leaf spot. Experiment 1 included genetic generations derived from three crosses of resistant × susceptible inbred parents (B79 × B98, B99 × N192, and B100 × MS1334), a cross of resistant × resistant inbred parents (B98 × B99), and a cross of susceptible × susceptible (B79 × N192) inbred parents. The classification of the lines being either susceptible (B79, MS1334, and N192) or resistant (B98, B99, and B100) was based on field ratings by Coates and White (1994) in Illinois for the susceptible lines and by ratings in the Iowa State University corn breeding program for the resistant lines. For each of the six crosses, five generations (P_1, P_2, F_1, F_2, BC1, and BC2) were available for study under gray leaf spot infection: P_1 and P_2 are the two parents of a cross; F_1 is the cross of P_1 and P_2; F_2 is the self pollination of F_1; and the backcrosses are BC1 (P_1) and BC2 (P_2).

The six generations were evaluated in field trials conducted at two locations (Hinds Farm and Agronomy Research Center) near Ames, IA. Field design was a split-plot design with three replications at each location. The generations were the whole plots while entries within each generation were the subplots. Two border rows of similar vigor were included on each side of each whole plot. Plot lengths were 3.8 (Hinds Farm) and 5.5 m (Research Center) with 0.76 m between rows at both locations. Planting dates were April 29 (Hinds Farm) and May 5, 1998 (Research Center). Plant densities were 51 M plants ha^{-1} (Hinds Farm) and 54 M plants ha^{-1} (Research Center). Plots included two rows for P_1, P_2, and F_1 generations, four rows for the BC1 and BC2 generations, and eight rows for the F_2 generation. Previous year crop was oats (*Avena sativa* L.) at both locations, and minimum tillage practices were used before planting.

Experiment 2 included 100 S_1 progenies obtained from the cross of B79 × B98 by selfing F_2 plants. The 100 S_1 progenies were evaluated in a randomized complete block design with three replications at Ames, IA, (Hinds Farm) and Crawfordsville, IA. Planting dates were April 29 for Ames and May 11, 1998 for Crawfordsville. Preceding crops were oats at Crawfordsville and soybeans [*Glycine max* (L.) Merrill] at Ames. One-row plots were used at both locations with plot lengths of 3.8 m at Ames and 5.5 m at Crawfordsville. Plant densities were about 51 M plants ha^{-1} at Ames and 54 M plants ha^{-1} at Crawfordsville.

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Disease Inoculation and Assessment

Artificial infection of gray leaf spot was done for both locations for Experiment 1, but only for the Ames location for Experiment 2. Cultures were stored on 15% sterile glyceol. Inoculum used for infection was spores produced on 1-year-old V-8 agar plates (Beckman and Payne 1983) and infected wheat (*Triticum aestivum*) grains. All culture spore suspensions for flood seeding of V-8 plates and seeding of wheat kernels were in 10% sterile skim milk. For inoculum produced on wheat grain, hard red wheat grain was boiled for 20 min, washed to remove stickiness, placed in autoclavable polypropylene bags (25 cm × 30 cm × 5 cm thick), autoclaved on successive days at 121 C for 30 and 60 min, respectively, and seeded with *C. zeae-maydis* spores in a 10% skim milk suspension. The bag opening had been sealed around a 4.5 cm length of polypropylene pipe 3.37 cm I.D. The pipe was stuffed with cotton to maintain sterility. After 2 weeks the wheat grain inoculum was ready for use for Experiment 1 at both locations and Experiment 2 at Ames. Plants were inoculated weekly during the early evenings for the two experiments located near Ames. Plants were inoculated by spraying the conidial suspension of harvested spores with a backpack sprayer in the whorl June 23 and to the underside of the leaves on July 7 and July 14. Plants were inoculated June 30 by placing approximately 2 g of infested wheat kernels in each whorl. Natural inoculum was relied upon for gray leaf spot infection at Crawfordsville because high levels of gray leaf spot infection occur in this area.

Disease assessments for Experiment 1 were made August 5, 12, 19, and 26 at the Hinds Farm and August 6, 13, 20, and 27 at the Agronomy Research Center. Disease assessments were based on visual estimates of percentage of leaf area affected (PLAA). Assessments were taken on the ear leaf of five consecutive plants in the center of a row using the gray leaf spot assessment scale developed by Smith (1989). The disease assessments of the five plants within each row within each replication were averaged to obtain a plot mean. The PLAA mean data were fit to disease progress models in calculating the area under the disease progress curve (AUDPC) (Nutter and Parker 1997). The AUDPC was calculated as:

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

where Y_i = PLAA at the ith assessment, X_i = days at the ith assessment, and n = total number of assessments (Shaner and Finney 1977). Relative AUDPC values were obtained by dividing AUDPC values by the total duration in days of each disease epidemic (Fry 1978).

Disease assessments for Experiment 2 were made August 4, 11, 18, and 25 at the Hinds Farm (inoculum applied) and August 14, 21, and 28, and September 3 and 10 at Crawfordsville (natural infection). Disease assessments for Experiments 1 and 2 were taken until at least one entry across replications within location had 100% PLAA.

Statistical Analyses

Experiment 1—An unweighted analysis of variance was calculated for each location and combined across locations. Locations and replications were considered random effects while generations were considered fixed effects. Because of herbicide damage at the Hinds Farm, data were not collected for the crosses B98 × B99 and B99 × N192; B99 was susceptible to the herbicide that affected all generations that included B99.

The generation mean analysis was used to determine the relative importance of the genetic effects for gray leaf spot in the five crosses (Hayman 1958, 1960; Gamble 1962). Each of the generation means can be expressed as m = general mean, a = pooled additive effects, and d = pooled dominance effects. A least squares regression model was used to estimate m, a, and d (Proc. GLM, SAS/STAT 1988). Successive models were fit sequentially starting with m and then adding additional parameters. Models were considered adequate when the lack-of-fit mean square was not significant when tested against the generation × environment interaction mean square for crosses B79 × B98, B79 × N192, and B100 × MS1334 or when tested against generation × replication mean square for crosses B98 × B99 and B99 × N192. Genetic models that included epistatic effects were not used because the a and d genetic parameters accounted for more than 95% of the variation among generation means. Genetic effects for gray leaf spot infection were estimated for each cross by solving the least square regression equation

$$\beta = (X'X)^{-1}(X'Y),$$

where β is a column vector of genetic effects being estimated, X is the coefficient matrix for the genetic effects of the generations, and Y is the column vector of observed generation means. A chi-square was calculated to test for lack-of-fit for the genetic effects for each model. Standard errors (SE) for the genetic effect estimates were calculated as the diagonal elements of the solution equation as

$$\text{SE} = [(X'X)^{-1}\ \sigma^2]^{1/2},$$

where σ^2 is the error variance for each cross.

Experiment 2—An analysis of variance of 100 S_1 progenies developed from the cross of B79 × B98 was calculated for each location and combined across locations. Locations (e), replications (r), and S_1 progenies (g) were considered random effects for determining the expected mean squares for making appropriate F-tests and calculation of error variance (σ^2), S_1 progeny by location interaction variance (σ^2_{ge}), and the genotypic variance among S_1 progenies (σ^2_g). All S_1 progenies had three replications at each of the two locations. Broad-sense heritability (h^2) estimates on S_1 progeny mean basis was calculated as

$$h^2 = \hat{\sigma}^2_g/re + \hat{\sigma}^2_{ge}/e + \hat{\sigma}^2_g),$$

where r is the replications (r = 3) and e is locations (e = 2). The broad-sense heritability estimate was used to determine the expected genetic gain for the next cycle of selection as

$$\Delta_G = h^2(\bar{X}_S - \bar{X}),$$

where Δ_G is the expected genetic gain, h^2 is the broad-sense heritability estimate, $(\bar{X}_S - \bar{X})$ is the selection differential, $\bar{X}_S$ is the mean of the selected S_1 progenies at a 10% selection intensity, and $\bar{X}$ is the mean of the population of 100 S_1 progenies tested (Hallauer 1986).

RESULTS AND DISCUSSION

Experiment 1

Experiment 1 was planned to include three types of crosses between inbred lines that were either resistant (R) or susceptible (S) to gray leaf spot infection: S × S, S × R, and R × R. The choice of susceptible lines was based on the relative levels of gray leaf spot resistance reported by Coates and White (1994) and included B79, MS1334, and N192. The choice of resistant inbred lines (B98, B99, and B100) was based on observations and ratings for gray leaf spot resistance in Iowa. The planned crosses included B79 × N192 as the S × S cross, B98 × B99 as the R × R cross, and B79 × B98, B99 × N192, and B100 × MS1334 as the R × S crosses. B79, however, was either incorrectly rated in Illinois, or B79 has a different reaction to gray leaf spot infection in Illinois than in Iowa. The PLAA and AUDPC ratings for B79 were similar to those of B98,

Table 1. Generation means and standard errors (SE) for percent leaf area affected (PLAA) by gray leaf spot infection for five crosses of corn inbred lines.

	Crosses				
Generation	B100 × MS1334	B79 × B98	B79 × N192	B98 × B99	B99 × N192
P_1[a]	23.5	16.2	16.2	15.0	18.3
P_2	48.4	15.0	43.1	18.3	45.2
F_1	11.2	8.8	12.6	7.2	9.6
F_2	20.3	12.4	18.3	10.8	17.1
BC1	20.0	12.2	15.8	11.8	13.2
BC2	26.4	11.2	22.2	11.4	19.6
Average	20.6	10.4	17.8	10.5	17.2
SE	5.1[b]	1.0[b]	4.5[b]	1.6[c]	5.2[c]
LSD (0.05)	5.9[b]	2.3[b]	4.7[b]	3.1[c]	4.2[c]

[a]P_1 is the first parent of each cross
[b]Values were calculated with n = 6 (SE) and error degrees of freedom = 20 (LSD)
[c]Values were calculated with n = 3 (SE) and error degrees of freedom = 10 (LSD)

Table 2. Generation means and standard errors (SE) for area under disease progress curve (AUDPC) gray leaf spot ratings for five crosses of corn inbred lines.

	Crosses				
Generation	B100 × MS1334	B79 × B98	B79 × N192	B98 × B99	B99 × N192
P_1[a]	21.2	14.4	14.4	13.2	16.1
P_2	42.9	13.2	37.2	16.1	37.2
F_1	10.3	7.9	11.0	6.4	8.4
F_2	17.2	11.0	16.2	9.5	14.9
BC1	17.6	10.9	13.9	10.4	11.5
BC2	23.5	10.0	19.3	10.0	17.3
Average	22.1	10.9	18.7	10.9	17.6
SE	4.6[b]	0.9[b]	3.9[b]	1.4[c]	4.1[c]
LSD (0.05)	5.6[b]	2.1[b]	4.5[b]	2.7[c]	4.3[c]

[a]P_1 is the first parent of each cross
[b]Values were calculated with n = 6 (SE) and error degrees of freedom = 20 (LSD)
[c]Values were calculated with n = 3 (SE) and error degrees of freedom = 10 (LSD)

B99, and B100, which were considered the more resistant lines (Tables 1 and 2). Data for the B98 × B99 (R × R cross) and B99 × N192 (R × S cross) crosses were limited to one environment because B99 was injured by the herbicide applied at the Hinds Farm location. Herbicide damage also occurred in the F_1, F_2, and backcross generations that included B99 and the plants either died or were severely stunted.

Except for B79, the severity of gray leaf spot infection supported the original classification of the lines: MS1334 and N192 had the greatest ratings and leaf infections, whereas B98, B99, and B100 had less incidence of gray leaf spot infection (Tables 1 and 2). The levels of gray leaf spot infection in B79 (PLAA, Table 1 and AUDPC, Table 2) was less than for B99 and B100. It seems B79 has resistance to gray leaf spot and should be included with B98, B99, and B100 in the resistant group of lines.

Highly significant ($P \leq 0.01$) differences occurred among generations for all crosses for PLAA and AUDPC (analyses are not shown). Genetic models that include the mean (m), pooled additive effects (a), and pooled dominance effects (d) were sequentially fit for PLAA and AUDPC. The lack-of-fit mean square was highly significant after fitting the m and a parameters in all crosses for PLAA and AUDPC. After the d parameter was included in the genetic model, the lack-of-fit mean square was significant ($P \leq 0.05$) in 3 of 10 instances: the AUDPC for B79 × N192 and the AUDPC and PLAA for B99 × N192. N192 (the susceptible parent) was one parent in each of the crosses that had significant lack-of-fit mean square after fitting the m, a, and d parameters. But the model that included m, a, and d accounted for 95.6 (B79 × N192 for AUDPC), 96.9 (B99 × N192 for PLAA), and 95.7% (B99 × N192) of the total variation among generations.

The PLAA and AUDPC ratings of the F_1s were less than either of the parents in all instances (Tables 1 and 2). Average PLAA ratings of the resistant parents were 17.2 vs. 45.6% for the susceptible parents and 8.3% for the five crosses (Table 1). Average PLAA ratings of the F_1s (9.9%) were 8.4% less than the average of the resistant parents (18.2%), or F_1s averaged 45.9% less PLAA than the average of resistant parents. The average AUDPC ratings of the resistant parents were 16.2 vs. 40.0 for the susceptible parents and 8.8 for five crosses (Table 2). Average AUDPC of the five crosses was 7.4 less than the average of the resistant parents, or F_1s averaged 45.7% lower AUDPC ratings than the resistant parents. If the R × R (B79 × B98 and B98 × B99) crosses are compared with the R × S (B100 × MS1334, B79 × N192, and B99 × N192) crosses, the average of the two sets of crosses is similar for PLAA (8.0% for R × R vs. 11.1% for R × S) and AUDPC (7.2 for R × R vs. 9.9 for R × R) ratings. It seems that resistance to gray leaf spot is conditioned by dominant favorable alleles and that different favorable alleles are included in the parents because the F_1s had less gray leaf spot infection than the more resistant parent. It seems single-cross hybrids can be produced with improved levels of resistance if at least one of the parents has good resistance to gray leaf spot infection.

Estimates of genetic effects that conditioned resistance to gray leaf spot indicate that additive (a) and dominance (d) effects were significant in all crosses except for the a parameter for B98 × B99 (Table 3). The estimates of a (1.4 and −1.2) were smallest for the two R × R crosses compared with the estimates of a (−11.4, −2.0, and −12.0) for the R × S crosses for PLAA; similar estimates were obtained for AUDPC. Except for B79 × B98, all estimates of a for PLAA and AUDPC were negative, indicating thar the pooled additive effects contributed to greater resistance to gray leaf spot infection.

Estimates of d were significantly negative for all crosses for PLAA and AUDPC (Table 3). Estimates of d were greater for the R × S crosses than for the R × R crosses, which also was evident in the levels of gray leaf spot resistance of the F_1s (Tables 1 and 2). The significant estimates of a and d suggest selection for greater gray leaf spot resistance would be effective and that the resistance of the lines used to produce single-cross hybrids would transmit this resistance to the hybrids. Estimated pooled dominance effects were 0.5 to 8 times greater than pooled additive effects.

Experiment 2

The combined analysis of variance of the 100 S_1 progenies developed from the F_2 population of B79 × B98 indicated highly significant differences for PLAA among the S_1 progenies and for the S_1 progeny × location interaction (analysis not shown). Differences of

Table 3. Estimates of genetic effects and standard errors (SE) for percent leaf area affected (PLAA) and area under disease progress curve (AUDPC) for gray leaf spot ratings for five crosses of corn inbred lines when the mean (m), additive (a), and dominance (d) effects are included in the genetic model.

Genetic effects[a]	Crosses				
	B100 × MS1334[b]	B79 × B98[b]	B79 × N192[b]	B98 × B99[c]	B99 × N192[c]
Percent leaf area affected					
m	22.9** ± 1.2	11.8** ± 0.2	19.9** ± 1.3	11.6* ± 0.4	18.5** ± 1.6
a	−11.4** ± 1.8	1.4* ± 0.3	−12.0** ± 1.9	−1.2 ± 0.6	−12.0** ± 2.4
d	−24.9** ± 3.3	−5.8** ± 0.5	−17.9** ± 3.6	−9.7** ± 1.1	−23.6** ± 4.5
Area under disease progress curve					
m	20.3** ± 1.1	10.5** ± 0.2	17.4** ± 1.1	10.2** ± 0.4	16.0** ± 1.1
a	−9.9** ± 1.7	1.4* ± 0.3	−10.2** ± 1.6	−1.1 ± 0.5	−9.6** ± 1.7
d	−22.4** ± 3.2	−4.9** ± 0.5	−15.5** ± 3.0	−8.4** ± 1.0	−19.3** ± 3.2

[a]The genetic effects include the mean (m), pooled additive effects (a), and pooled dominance effects (d)
[b]Estimates of genetic effects are based on data collected from three replications in two environments
[c]Estimates of genetic effects are based on data collected from three replications in one environment
* and ** indicates levels of significance at the 0.05 and 0.01 probability levels, respectively.

level of PLAA between locations could occur because plots at the Hinds Farm near Ames had gray leaf spot inoculum applied four times, whereas plots at Crawfordsville relied on natural infection. The range of PLAA means across the two locations for gray leaf spot ratings was from 10.2 to 36.4% with an average rating of 17.7%.

Estimates of components of variance for experimental error (σ^2 = 22.3 ± 4.1), S_1 progeny by location interaction (σ^2_{ge} = 4.7 ± 2.0), and S_1 progenies (σ^2_g = 16.2 ± 1.6) were calculated from the mean squares of the analysis of variance combined across locations to obtain an estimate of heritability (h^2 = 0.78) based on S_1 progeny means. The estimate of heritability was used to predict future genetic gain (Δ_G) if the best 10% were used in future selection: Δ_G = 5.1%, indicating a 5.1% reduction in PLAA from the selected 10 S_1 progenies. The estimates of h^2 and Δ_G were greater than expected after it was determined that B79 had greater resistance than reported by Coates and White (1994) (Tables 1 and 2). The original intent was to study the variation among S_1 progenies developed from a cross that included susceptible and resistant parents. But B79 was as resistant to gray leaf spot as B98. The relatively high estimate of heritability (h^2 = 0.78) from the cross of resistant parents suggests different alleles were conditioning resistance in B79 and B98. B79 and B98 were developed from two different source populations. B79 was developed from BS10(FR)C0, whereas B98 was developed from BS11(FR)C5 (Russell and Hallauer 1976, Hallauer et al. 1994). The divergent source populations provided different alleles for gray leaf spot resistance in B79 and B98 and is consistent with the mean gray leaf spot resistance of the F_1s in Tables 1 and 2. Resistance to gray leaf spot of the F_1 generations of B79 × B98 and B98 × B99 was greater than either of the parents of the two crosses.

Information from Experiments 1 and 2 supports the suggestion that selection should be effective to increase the levels of gray leaf spot resistance. Genetic variation was adequate to expect response to selection, including both additive and dominance genetic effects. If good levels of resistance are not present in both parents of a hybrid, the level of resistance of the hybrid can be increased nearly 50% or more if one parent has a good level of resistance. Gray leaf spot is a common disease of corn in Iowa and will continue to be with the necessary tillage practices needed to reduce wind and water erosion. Although gray leaf spot resistance does not seem to be controlled by a few major genes, selection methods and germplasm are available to enhance the levels of resistance to gray leaf spot for corn grown in Iowa.

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