Inheritance of Resistance to Stalk Rot of Corn Caused by *Colletotrichum graminicola*

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ABSTRACT

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The inheritance of resistance in corn (*Zea mays* L.) to anthracnose stalk rot (ASR) caused by *Colletotrichum graminicola* was studied in progeny from five sets of crosses involving four resistant inbred lines—A556, A638, Oh43, and R177—and two susceptible inbreds—C123 and B73. In 1977 and 1978, populations consisted of the parental inbred lines, F_1, F_2, and backcross generations. In 1979, the study was expanded to include second backcross (B_{11} and B_{22}), backcross-selfed (B_{1S} and B_{2S}), and F_3 generations. Analysis of generation means over years indicated that additive genetic effects accounted for more than 90% of the total variation among generation means in all populations. Estimates of genetic and environmental variances were apparently biased in some populations. Estimates of heritability, the largely additive gene action involved, and the relatively high frequency of F_3 families with high levels of resistance in all populations indicate that the pedigree method and recurrent selection schemes would be effective ways to increase ASR resistance in corn populations and inbred lines developed from them.

Additional key words: generation mean analysis, maize.

Anthracnose stalk rot (ASR), caused by *Colletotrichum graminicola* (Ces.) G. W. Wils., has become a major stalk rot problem of corn (*Zea mays* L.) in the United States (4,13,18,27,30,32,34,35). Once considered a minor leaf spot disease in the United States (31), anthracnose has been shown to cause serious damage to corn in other areas of the world (21,29). The increasing prevalence of *C. graminicola* in cornfields and the pathogen's demonstrated ability to reduce yields in dent corn concern U.S. corn producers, breeders, and seedsmen.

Williams and Willis (35) reported isolating *C. graminicola* from 50% of infected stalks in plots at the Ohio Agricultural Experiment Station in 1961. Dale (4) reported in 1963 that anthracnose was common in Arkansas cornfields. However, yield losses or increased stalk lodging caused by ASR could not be demonstrated in two hybrids when plots of inoculated and uninoculated plants were compared. Sweet corn fields in Benton County, IN, were reported as total losses in 1972 because of damage by *C. graminicola* (32).

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Anthracnose was one of the most devastating diseases of corn in North Carolina in 1972 and 1973 (18). Affected fields showed increased lodging and premature plant death. The greater prevalence of corn anthracnose was attributed to the increased use of susceptible hybrids. *C. graminicola* was associated with rotted stalks from 78% of fields examined in Illinois in 1975 (13).

Yield losses from anthracnose leaf blight as high as 19% in certain hybrids have been documented when plot yields of inoculated and uninoculated plants were compared (30). Inbred lines sustained losses up to 28% in the same study. Natural ASR infection was reported to cause yield losses as high as 17.2% compared with healthy plants (27). Losses were caused primarily by premature plant death during grain filling. White et al (34) reported a 40% yield loss from ASR when yields of uninoculated plants of a susceptible hybrid were compared with yields from plants inoculated at anthesis.

Genetic resistance is the only economically feasible way to control losses from corn stalk rots. Inheritance studies indicate that resistance to stalk rot caused by *Diplodia maydis* (Berk.) Sacc. is polygenic and mostly additive in gene action and that many factors are involved (16). Selection for increased stalk strength and resistance to stalk rot has resulted in inbred lines and hybrids highly resistant to Diplodia stalk rot. Although reactions of corn inbreds

to stalk rot caused by *D. maydis* are highly correlated with reactions to stalk rot caused by *Gibberella zeae* (Schw.) Petch. (12), they are not well correlated with reactions to ASR (33). Miles et al (22), however, reported positive, significant genetic correlations between Diplodia stalk rot and ASR reactions in two corn populations.

Genetic information relating to ASR resistance in corn is limited. Resistance to sorghum red rot, also caused by *C. graminicola*, is controlled by a single dominant gene linked to another single dominant gene controlling resistance to foliar anthracnose (3). Humy (15) found that corn inbred lines reacted very differently to both foliar and stalk infection and that the two traits were not correlated. A diallel analysis of 10 corn inbred lines revealed that both additive and nonadditive genetic effects were important in resistance to ASR, although additive effects were more important (19). Significant heterotic effects indicated partial dominance for resistance or susceptibility at some loci. The authors also concluded that resistance to the stalk rot and leaf blight phases is inherited independently. Miles et al (22), in constructing selection indexes for yield and resistance to foliar diseases and stalk rots in two corn populations, concluded that selection for resistance to stalk rots (including ASR) on a single-plant basis would be as effective as selection based on S_1 progeny evaluations in terms of gain per year. Resistance to ASR was not genetically correlated with yield, so selection for ASR resistance should not produce a negative correlated response in yield.

We examined the inheritance of ASR reactions in several adapted corn inbred lines and generations derived from crosses between them and attempted to determine the importance of genetic and environmental effects in ASR reaction and the implications for breeding for ASR resistance.

MATERIALS AND METHODS

Six corn inbred lines were chosen based on prior evaluations of their reactions to ASR (listed in order of increasing susceptibility): A556, A638, R177, Oh43, B73, and C123. Four populations were created by crossing A556, A638, R177, and Oh43 with C123. A fifth population was formed by crossing A556 with B73. The study was conducted in 1977, 1978, and 1979 on the Agronomy South Farm, Urbana, IL.

In 1977 and 1978, the reactions of the parent inbred lines and the F_1, F_2, and first backcross generations (B_1 and B_2) to ASR were studied. Each population was grown in a randomized block with four replicates. Each replicate consisted of the following number of 12-plant, single-row plots per generation: P_1, P_2, and F_1, one plot each; F_2, six plots; and B_1 and B_2, three plots each. Plants were spaced about 38 cm apart in a row, with rows 76 cm apart. Actual numbers of plants per plot varied somewhat because of loss from poor germination and cultivator injury.

In 1979, the study was expanded to include the F_3, the first backcross-selfed (B_{1S} and B_{2S}), and second backcross (B_{11} and B_{22}) generations of each of the five populations. F_3 families were produced by selfing a random sample of F_2 plants. B_{1S} and B_{2S} generations were produced by selfing a random sample of plants in the B_1 and B_2 generations, respectively. B_{11} and B_{22} generations were produced by crossing individual plants in the B_1 and B_2 generations with P_1 and P_2 parental inbreds, respectively. Table 1 lists the number of F_2, B_1, and B_2 plants sampled to form the F_3, B_{11} and B_{1S}, and B_{22} and B_{2S} generations, respectively. F_3 families were kept separate, while the B_{1S}, B_{2S}, B_{11}, and B_{22} generations were composites formed by bulking equal numbers of seeds from each plant sampled in the parental generations. Field plot design in 1979 was identical to that used in the previous years with the addition of the following number of 12-plant, single-row plots and generations to each replicate: F_3 families, one plot each; B_{1S}, B_{2S}, B_{11}, and B_{22} generations, three plots each.

Plants were inoculated 1–2 wk after midsilk by injecting 2 ml of a conidial suspension of *C. graminicola* into the first elongated internode above the brace roots with a 50-cm^3 Vaco Pistol Grip rubber plunger syringe (Ideal Instruments, Inc., Chicago, IL 60612) fitted with a stainless steel needle similar to that described by Koehler (17).

Inoculum was produced by washing conidia from the surface of oatmeal agar cultures of *C. graminicola*, filtering the suspension through a double thickness of cheesecloth, and adjusting the final concentration to 2.0×10^5 conidia per milliliter. The inoculum used each year was a mixture of isolates obtained from infected stalk tissue collected in the nursery the previous year.

Four weeks after inoculation, plants were cut off above the ear, the stalk was split longitudinally to ground level, and the total number of discolored internodes and internodes with more than 75% rotting was recorded. The disease rating scale for ASR was based on disease spread and severity in the inoculated internode and the five internodes above it. Disease scores of individual plants equaled the number of the six internodes showing discoloration plus the number with more than 75% rotted tissue.

The data from all plots containing the same generation within a replicate were pooled to obtain mean ASR reactions of each generation. Means were then fitted by both weighted and unweighted regression to the genetic model of Mather and Jinks (20):

$$\bar{Y} = m + a_1 d + a_2 h + a_3 i + a_4 j + a_5 l,$$

where $\bar{Y}$ is the mean of a given generation; m is the intercept; d is the pooled additive genetic effect; h is the pooled dominance genetic effect; i, j, and l are the pooled digenic, epistatic genetic effects; and a_1–a_5 are the respective coefficients of these effects in the equations of expectation of generation means (Table 2). Genetic effects were tested for significance by F tests in the analyses of variance of generation means.

The additive plus dominance model ($\bar{Y} = m + a_1 d + a_2 h$) was fitted to 1977 and 1978 data, while the complete model allowing for digenic interactions ($\bar{Y} = m + a_1 d + a_2 h + a_3 i + a_4 j + a_5 l$) was fitted to 1979 data. In the combined analysis over years, the generation × years mean square, when significant, was used to test the significance of additive and dominance genetic effects and residuals from fitting the $\bar{Y} = m + a_1 d + a_2 h$ model. Weights used in the

TABLE 1. Numbers of F_2 plants sampled to form F_3 families, B_1 plants sampled to form B_{11} and B_{1S} generations, and B_2 plants sampled to form B_{22} and B_{2S} generations in five corn populations studied in 1979 for resistance to stalk rot caused by *Colletotrichum graminicola*

	Number of plants in sampled generation/tested generation				
Population	F_2 F_3	B_1 B_{11}	B_2 B_{22}	B_1 B_{1S}	B_2 B_{2S}
A556 × C123	40	23	16	23	17
A556 × B73	40	23	15	24	21
A638 × C123	40	20	23	22	24
Oh43 × C123	30	24	18	24	24
R177 × C123	30	24	18	23	14

TABLE 2. Equations of expectation for generation means for anthracnose stalk rot resistance in corn in terms of additive (d), dominance (h), additive × additive (i), additive × dominance (j), and dominance × dominance (l) genetic effects in the model, $\bar{Y} = m + a_1 d + a_2 h + a_3 i + a_4 j + a_5 l$

	Coefficients				
Generation mean	a_1	a_2	a_3	a_4	a_5
$\bar{P}_1$	1	0	1	0	0
$\bar{P}_2$	−1	0	1	0	0
$\bar{F}_1$	0	1	0	0	1
$\bar{F}_2$	0	1/2	0	0	1/4
$\bar{B}_1$	1/2	1/2	1/4	1/4	1/4
$\bar{B}_2$	−1/2	1/2	1/4	−1/4	1/4
$\bar{B}_{11}$	3/4	1/4	9/16	3/16	1/16
$\bar{B}_{22}$	−3/4	1/4	9/16	−3/16	1/16
$\bar{B}_{1S}$	1/2	1/4	1/4	1/8	1/16
$\bar{B}_{2S}$	−1/2	1/4	1/4	−1/8	1/16
$\bar{F}_3$	0	1/4	0	0	1/16

weighted regression procedure were the reciprocals of the variances of generation means.

Second-order statistics were obtained as follows to allow the estimation of genetic and environmental variances: within-plot variances of each generation were obtained by pooling sums of squares and degrees of freedom of all plots containing the same generation within replicates. With the 1977 and 1978 data, only direct estimation of genetic and environmental variances was possible. Additive genetic variances (σ^2_A) were obtained by the formula:

$$\sigma^2_A = 2V_{F_2} - V_{B_1} - V_{B_2},$$

where V_{F_2}, V_{B_1}, and V_{B_2} are the variances of the F_2, B_1, and B_2 generations, respectively. Similarly, dominance genetic variances (σ^2_D) were estimated by:

$$\sigma^2_D = V_{B_1} + V_{B_2} - V_{F_2} - \sigma^2_E.$$

Environmental variances (σ^2_E) were estimated directly from the pooled within-plot variances of the parental inbred lines and F_1 generations. Standard errors of the estimates were obtained in the manner of Powers (28); eg,

$$V_{\sigma^2_A} = 4V_{V_{F_2}} + V_{V_{B_1}} + V_{V_{B_2}}.$$

The variances obtained with 1979 data and their equations of expectation in terms of genetic and environmental variances are listed in Table 3. The variances of F_3 family means ($\sigma^2_{\bar{F}_3}$) were adjusted for genetic sampling (25) and for E_2, the best estimate of which was the error term from the analysis of variance of F_3 means (14).

Three methods were used to estimate genetic and environmental variances and their standard errors in 1979. Ordinary least squares (OLS) regression was used to fit the data to the genetic model:

$$\sigma^2_P = C_1\sigma^2_A + C_2\sigma^2_D + C_3\sigma^2_E,$$

where σ^2_P is the phenotypic variation for individual ASR reactions in a given generation and C_1–C_3 are the coefficients of the genetic and environmental variances in the equations of expectation. Hayman (10) and Nelder (25) have both proposed alternatives to OLS estimation of variance components, which is considered inefficient in the estimation of genetic variances and biased in the estimation of their standard errors, because of the inequality of variances and the correlations among the variances used in the estimation. Nelder proposed that least-squares be used to fit the genetic model to data within each replicate and that these estimates be used to obtain mean estimates and their standard errors by the direct, empirical method. Hayman proposed a maximum likelihood estimation (MLE) procedure, which reduces to iterative weighted least-squares regression. Weights used in this procedure were reciprocals of the maximum likelihood expectations for the variances of the variances, $N/2V^2$, where N is the degrees of freedom associated with the variance V. The estimated genetic and environmental variances from the weighted regression are then used to calculate expected variances, which in turn are used to calculate weights for the next iteration. Four iterations were used in the analysis of 1979 variances.

TABLE 3. Equations of expectation for variances obtained with 1979 data in terms of genetic and environmental variances

Variance	Coefficient of: σ^2_A	σ^2_D	σ^2_E
$\sigma^2_{P_1}$, $\sigma^2_{P_2}$, $\sigma^2_{F_1}$ (pooled)	0	0	1
$\sigma^2_{F_2}$	1	1	1
$\sigma^2_{B_1} + \sigma^2_{B_2}$	1	2	2
$\sigma^2_{\bar{F}_3}$	1	1/4	0
$\sigma^2_{F_3}$	1/2	1/2	1

TABLE 4. Mean anthracnose stalk rot reactions[a] of generations and deviations of the F_1 mean from midparent (MP) values in five corn populations in 1977 and 1978

Year Generation	Population A556 × C123	A556 × B73	A638 × C123	Oh43 × C123	R177 × C123
1977					
$\bar{P}_1$	1.06	1.04	4.35	3.83	3.48
$\bar{B}_1$	2.29	2.81	5.02	4.10	5.50
$\bar{F}_1$	4.48	4.57	6.49	4.35	6.62
$\bar{F}_2$	5.03	4.21	7.03	5.69	6.39
$\bar{B}_2$	7.13	6.55	8.38	8.07	7.76
$\bar{P}_2$	8.79	7.40	8.54	9.07	8.50
FLSD (0.05)[b]	1.23	0.93	0.88	0.89	0.86
$\bar{F}_1$ – MP	−0.45	0.35	0.04	−2.10**[c]	0.63
1978					
$\bar{P}_1$	···	1.04	3.13	2.97	2.86
$\bar{B}_1$	···	1.43	3.33	3.02	3.45
$\bar{F}_1$	···	2.38	5.79	6.02	4.02
$\bar{F}_2$	···	3.07	3.39	4.61	3.88
$\bar{B}_2$	···	3.76	6.33	7.42	6.32
$\bar{P}_2$	···	6.39	8.95	8.69	7.78
FLSD (0.05)[b]	···	1.01	1.26	1.11	0.64
$\bar{F}_1$ – MP	···	−1.34**	−0.25	0.19	−1.30**

[a] Rating scale of 1–12; 1 = most resistant, 12 = most susceptible.
[b] Fisher's least significant difference ($P = 0.05$).
[c] ** Indicates that the difference is statistically different from zero at the 0.01 level of probability.

RESULTS

Data from the A556 × C123 population in 1978 were discarded because a poor stand was obtained, and a replicate of F_3 families in the A638 × C123 population in 1979 was lost because of an error in inoculating the plants.

Mean reactions of parental inbred lines to ASR varied widely (Table 4). A556 was the most resistant of the four resistant parents,

TABLE 5. Mean anthracnose stalk rot reactions[a] of generations and deviations of the F_1 mean from midparent (MP) values in five corn populations in 1979 and combined means for 1977, 1978, and 1979

Year Generation	Population A556 × C123	A556 × B73	A638 × C123	Oh43 × C123	R177 × C123
1979					
$\bar{P}_1$	1.34	1.17	2.49	5.56	4.13
$\bar{B}_{1_1}$	2.37	1.89	4.45	4.49	4.28
$\bar{B}_{1_S}$	2.98	1.93	4.09	5.51	4.36
$\bar{B}_1$	2.58	2.19	5.31	6.32	4.22
$\bar{F}_1$	4.17	5.24	7.81	6.04	7.02
$\bar{F}_2$	6.09	4.03	6.94	6.91	5.25
$\bar{F}_3$	5.93	4.44	3.88	6.37	4.85
$\bar{B}_2$	8.40	6.22	8.21	8.80	7.20
$\bar{B}_{2_S}$	9.33	5.30	7.54	8.00	6.06
$\bar{B}_{2_2}$	9.68	6.88	9.35	9.18	7.17
$\bar{P}_2$	11.57	7.90	11.28	10.78	10.61
FLSD (0.05)[b]	0.79	0.66	1.28	1.13	0.92
$\bar{F}_1$ – MP	−2.29**[c]	0.71*	0.93	−2.13**	−0.34
Combined data					
$\bar{P}_1$	1.20	1.08	3.32	4.22	3.49
$\bar{B}_1$	2.43	2.15	4.51	4.48	4.39
$\bar{F}_1$	4.32	4.06	6.70	5.47	5.89
$\bar{F}_2$	5.58	3.77	5.78	5.74	5.17
$\bar{B}_2$	7.98	5.41	7.64	8.10	7.09
$\bar{P}_2$	10.18	7.23	9.48	9.68	9.07
FLSD (0.05)[b]	1.94	1.10	1.79	1.24	1.36
$\bar{F}_1$ – MP	−1.37	−1.10	0.29	−1.48*	−0.39

[a] Rating scale of 1–12; 1 = most resistant, 12 = most susceptible.
[b] Fisher's least significant difference ($P = 0.05$).
[c] * and ** indicate that the difference is statistically different from zero at the 0.05 and 0.01 levels of probability, respectively.

averaging slightly more than one discolored internode and never having internodes more than 75% rotted. The other three resistant parents—A638, Oh43, and R177— varied more in ASR reaction, both among plants within a year and among years.

Of the two susceptible parents used, B73 was less susceptible than C123. Both parents showed the same amount of disease spread within the stalk, as measured by the total number of discolored internodes, but plants of C123 usually had more internodes with 75% or more rot than B73. In the field, plants of C123 were usually killed prematurely by ASR within 4 wk of inoculation.

Reactions of F_1 generations to ASR varied greatly (Tables 4 and 5). Significant deviations of the mean F_1 reaction from midparent values were detected within years in the A556 × C123, A556 × B73, Oh43 × C123, and R177 × C123 populations. All of the significant deviations were negative (toward a lower disease severity) except the A556 × B73 population in 1979. In the combined analysis over years, only the Oh43 × C123 F_1 mean deviated significantly from the midparent value.

Several generalizations can be made about the means of segregating generations in this study. Mean ASR reactions of backcross generations approached those of the recurrent parent. Second backcross generation means, $\bar{B}_{11}$ and $\bar{B}_{22}$, were generally closer to the reactions of the recurrent parents than those of the B_1 and B_2 generations. F_3 generation means were generally very close to the F_2 means in all populations in 1979 except the A638 × C123 population, where the F_3 mean ASR severity was significantly below that of the F_2. In the combined analysis, F_2 means did not differ significantly from F_1 generation means.

The analysis of variance of generation means over years (Table 6) reveals that year and generation × year effects were important in ASR reactions. Year effects were significant in all but the A556 × C123 population. In general, generation means were lower in 1978 than in either 1977 or 1979, indicating that the environment in 1978 was less favorable for ASR development. Generation × year mean squares, though significant, were much smaller than generation mean squares.

Generation effects were highly significant in all populations in all years and in the combined analysis (Table 6). Additive genetic effects were always highly significant, accounting for more than 90% of the total variation among generation means in all populations with the unweighted combined analysis (Table 7). Dominance genetic effects were significant only in the Oh43 × C123 population in the combined analysis. Although some populations deviated significantly from the $\bar{Y} = m + a_1 d + a_2 h$ model in some years, these deviations became insignificant in the combined analysis when the generation × year mean square was used as an error term.

Results from the weighted analyses of generation means were similar to those obtained from unweighted analyses (Table 7). Additive genetic effects accounted for 92% or more of the variation among generation means in the combined weighted analyses of all populations.

TABLE 6. Analyses of variance of generation means for anthracnose stalk rot reaction after artificial inoculation in five corn populations in 1977, 1978, and 1979

Year	Source of variation	Mean squares for populations[a]				
		A556 × C123	A556 × B73	A638 × C123	Oh43 × C123	R177 × C123
1977	Block	0.93	1.04	3.34**	0.36	2.97**
	Generations					
	Additive	153.18**	103.81**	52.51**	83.67**	60.52**
	Dominance	0.52	0.39	0.09	10.28**	1.46
	Residual	1.36	1.08	1.13*	1.70*	0.20
	Error	0.65	0.37	0.34	0.35	0.32
1978	Block	...	1.31	0.92	3.56**	0.10
	Generations					
	Additive	...	67.94**	71.68**	72.94**	55.30**
	Dominance	...	5.87**	1.31	0.00	4.78**
	Residual	...	0.43	6.10**	1.71	1.68**
	Error	...	0.45	0.70	0.52	0.18
1979	Block	0.41	0.49	0.80	6.72**	0.52
	Generations					
	Additive	460.57**	194.76**	215.15**	122.17**	117.16**
	Dominance	14.66**	1.18*	6.14**	6.55**	0.15
	Epistasis	0.51	1.44**	9.21**	2.02*	11.12**
	Residual	0.85*	1.64*	3.64*	1.51	1.28*
	Error	0.30	0.21	0.78	0.62	0.40
Combined	Block (year)	0.95	0.78	1.59	2.02	1.52
	Year	8.50	15.98**	21.95*	23.94**	21.57**
	Generations					
	Additive	426.72**	288.32**	262.27**	226.82**	220.61**
	Dominance	9.12	0.39	0.11	18.42*	2.67
	Residual	1.70	0.68	2.02	2.11	2.45
	Generation × Year	2.29**	1.47**	3.88**	1.87**	2.24**
	Pooled error	0.51	0.37	0.67	0.57	0.28

[a] * and ** indicate that the difference is statistically different from zero at the 0.05 and 0.01 levels of probability, respectively.

TABLE 7. Percentage of variation in anthracnose stalk rot reaction among generation means of five populations accounted for (R^2) by fitting genetic effects in the combined weighted and unweighted regression analyses for 3 yr

Population	Unweighted analysis			Weighted analysis		
	Additive	Dominance	Residual	Additive	Dominance	Residual
A556 × C123	96.8	2.1	1.1	98.0	1.7	0.3
A556 × B73	99.2	0.1	0.7	97.7	1.7	0.6
A638 × C123	97.7	0.0	2.3	92.0	0.9	7.1
Oh43 × C123	90.2	7.3	2.5	92.8	3.7	3.5
R177 × C123	95.6	1.2	3.2	93.3	4.1	2.6

Direct estimates of genetic and environmental variances in the five corn populations grown in 1977 and 1978 are presented in Table 8. Estimates of additive genetic variance were significantly greater than zero in the A556 × C123, A638 × C123, and Oh43 × C123 populations in 1977 and in the Oh43 × C123 population in 1978. Significant positive estimates of dominance genetic variance were found in the R177 × C123 population in both years and in the A638 × C123 population in 1978. Significant negative estimates of dominance genetic variance were found in the Oh43 × C123 population in both years. All estimates of environmental variance were significantly greater than zero.

The data obtained in 1979 enabled us to use and compare three different methods of estimating genetic and environmental variances and their standard errors (Table 9). Estimates of σ^2_A by all three methods were significant in the A556 × C123, A556 × B73, and Oh43 × C123 populations. Estimates of σ^2_D were or were not significantly different from zero, depending on the method.

As expected (23), estimates from OLS and Nelder's empirical method were identical, except for the A638 × C123 population, in which data were missing in one replicate. Differences between the two procedures in the estimation of standard errors resulted in differences in the statistical significance of the estimates. In general, standard errors were lower with the empirical method than with OLS. The estimate of σ^2_D in the A556 × C123 population was not significant with OLS but was highly significant with Nelder's method. Similarly, estimates of σ^2_E were not significantly different from zero in two populations with OLS but were highly significant with Nelder's empirical method.

The MLE method gave lower estimates of σ^2_A when they were significant and tended to increase estimates of σ^2_D compared with OLS. Estimates of σ^2_E from MLE did not differ significantly from those obtained by OLS.

Narrow-sense heritabilities, where estimable, varied widely from year to year and among populations (Table 10). Unusually high estimates (greater than 90%) were obtained whenever estimates of σ^2_D were negative. When estimates of genetic and environmental variances were all positive, however, heritabilities ranged from 26 to 70%.

TABLE 8. Direct estimates of additive genetic variance (σ^2_A), dominance genetic variance (σ^2_D), and environmental variance (σ^2_E) of anthracnose stalk rot reactions after artificial inoculation in five corn populations

Population	Year	Variance[a]		
		σ^2_A	σ^2_D	σ^2_E
A556 × C123	1977	8.08**	−1.43	1.75**
A556 × B73	1977	2.56	1.70	1.02**
	1978	1.01	1.94	0.91**
A638 × C123	1977	4.25**	−0.27	2.13**
	1978	−5.13**	4.67**	3.35**
Oh43 × C123	1977	8.17**	−3.03*	3.04**
	1978	7.27**	−2.77*	2.37**
R177 × C123	1977	1.39	2.19*	1.45**
	1978	−1.32	3.60**	1.55**

[a] * and ** indicate that the estimate is significantly different from zero at the 0.05 and 0.01 levels of probability, respectively.

TABLE 9. Estimates of additive genetic variance (σ^2_A), dominance genetic variance (σ^2_D), and environmental variance (σ^2_E) of anthracnose stalk rot reactions after artificial inoculation in five corn populations in 1979

Population	Estimated variance	Method of estimation		
		OLS[a]	Empirical[b]	MLE[c]
A556 × C123	σ^2_A	6.03**[d]	6.03**	5.82**
	σ^2_D	1.45	1.45**	2.25
	σ^2_E	2.071**	2.071**	1.84**
A556 × B73	σ^2_A	2.28**	2.28**	1.47*
	σ^2_D	0.89	0.89	2.92*
	σ^2_E	1.92	1.92**	1.93*
A638 × C123	σ^2_A	−1.08	0.59	−0.55
	σ^2_D	11.04**	10.03**	7.96**
	σ^2_E	1.31	1.35	1.61**
Oh43 × C123	σ^2_A	3.39**	3.39**	2.37**
	σ^2_D	2.71*	2.71*	4.54*
	σ^2_E	3.45**	3.45**	3.33**
R177 × C123	σ^2_A	−0.06	−0.06	0.75
	σ^2_D	2.91**	2.91*	1.86
	σ^2_E	2.70	2.70**	2.55**

[a] Ordinary least squares estimation.
[b] This procedure is described by Nelder (25).
[c] Maximum likelihood estimation as described by Hayman (8).
[d] * and ** indicate that the estimate is significantly different from zero at the 0.05 and 0.01 levels of probability, respectively.

DISCUSSION

Generation mean analysis indicates that resistance to ASR in the corn populations studied is mostly additive in inheritance, with important dominance genetic effects in some populations. These findings are similar to results others have obtained using generation mean analysis to study the quantitative inheritance of disease resistance (7,14,16,23). The results are also in general agreement with those obtained by Lim and White (19) in a diallel analysis of ASR reaction. Their study indicated that partial dominance may also be important in ASR resistance, although their conclusions were based on single-cross hybrids and data from only 1 yr. The analysis indicated that genotype × environment interactions are a significant factor in ASR reactions. In this study, results from single-year analyses would indicate significant dominance genetic effects and deviations from the $\overline{Y} = m + a_1 d + a_2 h$ model. These effects were not significant in the combined analyses over years when the generation × year mean square was used as an error term, except in the Oh43 × C123 population, where significant, though small, dominance genetic effects were detected.

Weighted least squares have been suggested to ensure that assumptions about the equality of variances of generation means are met (20). Our results suggest that little information can be gained this way. Although the weights given to different generation means differed by 10-fold or more, differences in the percentage of variation among generation means accounted for by genetic effects in the weighted and unweighted analyses were negligible. These results resemble those obtained by others (22) and indicate that concerns about the appropriateness of unweighted least squares for generation mean analysis are unwarranted.

Assumptions are inherent in the use of any genetic and/or statistical model, including the model used in generation mean analyses. Besides the usual assumptions in least-squares models about normalized and homogeneous variances, certain assumptions must also be made about the genetic materials used in the study. Normal Mendelian segregation of alleles, the absence of selection favoring certain gametes or zygotes, and the absence of mutation are usually assumed in all quantitative genetic models (20). The lack of isodirectional distribution of alleles between the two parental lines alters the genetic expectations of the means of parents and backcross generations (20). These effects will be confounded with epistatic effects. Further, digenic or higher-order epistasis biases estimates of additive and dominance genetic effects

TABLE 10. Narrow-sense heritability estimates (percentages) of anthracnose stalk rot reaction in five corn populations

Population	Year		
	1977	1978	1979[a]
A556 × C123	96.2	···	63.2
A556 × B73	48.5	26.2	44.7
A638 × C123	69.6	++[b]	++
Oh43 × C123	99.9	105.8	35.5
R177 × C123	27.6	++	++

[a] Ordinary least squares estimates of variances were used to estimate heritability in 1979.
[b] ++ = No estimate of narrow-sense heritability was possible because estimates of additive genetic variance were negative.

(8,9,11,20). Because no significant deviations from the $\bar{Y} = m + a_1d + a_2h$ model were detected in the combined analyses of generation means in all five populations, neither epistasis nor noniso-directional distribution of alleles was implicated in ASR reactions in these populations. The limitations of means in detecting certain types of genetic situations should not be overlooked, however. Linkage cannot be detected in generation means in the absence of epistasis (20). Further, canceling effects of genes for resistance and susceptibility may prevent real genetic effects from being detected in generation means (16,20).

Direct estimation of σ^2_A and σ^2_D in 1977 and 1978 was apparently unreliable in some populations. Significant negative estimates of genetic variances in the A638 × C123 and Oh43 × C123 populations suggest that the estimates were biased. Estimates from 1979 data were more reasonable; none of the negative estimates of variances were significant by any of the three methods.

The highly significant estimates of σ^2_D and insignificant estimates of σ^2_A in the A638 × C123 population in 1979 suggest that these estimates may have been biased also. Tests of residuals from fitting the $\sigma^2_P = C_1\sigma^2_A + C_2\sigma^2_D + C_3\sigma^2_E$ model to the 1979 data were significant in the A638 × C123 and Oh43 × C123 populations and were nearly so in the R177 × C123 population. Linkage and epistasis can both lead to bias in the estimation of genetic variances by altering the equations of expectation for certain generations (20,26). Although methods exist for detecting these effects on variances (26), the 1979 data were insufficient to allow tests to be made.

Although results from generation mean analysis are not directly comparable to results of variance component analysis, the lack of significant deviations from the $\bar{Y} = m + a_1d + a_2h$ model in generation mean analysis suggests that linkage is probably the cause of bias in the variance component estimation, because linkage in the absence of epistasis would not be detected in generation mean analysis but would bias variance estimates. Epistatic effects not detected in the generation mean analyses may also have introduced bias.

The relatively large, highly significant estimates of plant-to-plant variation (σ^2_E) in all populations indicate the importance of environment on ASR reactions. Variation in competition among plants caused by a loss of stand in some plots is a source of variation in these studies. The increasing competition as plant population densities rise has been shown to increase incidence and severity of corn stalk rots, until the point where plants have reduced seed set or become barren, reducing stalk rot (24). The relatively low overall plant densities in these studies, however, indicate a small effect of competition among plants.

Another possible source of extraneous variability in the study was the severe infestation of European corn borers (*Ostrinia nubilalis* Hbn.) in all plots in 1978. Corn borer infestations can increase the incidence and severity of corn stalk rots, including ASR (1,2,21). This effect was not apparent in our studies, however; the mean ASR reactions were generally lower in 1978 than in either 1977 or 1979.

Highly heterozygous genotypes such as F_1 hybrids are usually thought to be less subject to fluctuations of the environment than are highly inbred genotypes (20). Evidence from this study indicates that the opposite is true in the case of ASR reaction in corn. In all populations in all years, the within-plot, plant-to-plant variation in ASR reaction was greater in the F_1 generation than in either inbred parent. Because stalk rot severity in individual plants is influenced by the grain yield produced by that plant (5,6,25), and because plant-to-plant variation in F_1 hybrid corn yield is high, this should not be unexpected. Because inbred lines lack the yield potential of F_1 hybrids, the effects of carbohydrate translocation stresses, caused by high grain yields, on stalk rot development could conceivably be less important in inbred line disease reactions than in hybrids. This could account for the greater variation in stalk rot reactions in hybrids than in inbreds.

Several observations can be made when comparing the three methods used to estimate genetic and environmental variances in 1979. First, the only apparent advantage of Nelder's empirical method over OLS is that standard errors of estimates are generally lower and unbiased. Second, the results from MLE resembled those from OLS with two disturbing exceptions: in the A556 × B73 population, the OLS estimate of σ^2_D was small and statistically insignificant, whereas with MLE, the estimate was significant and about twice as large as the estimate of σ^2_A; and MLE estimates of genetic variances were all insignificant in the R177 × C123 population. MLE assumes that individual data are normally distributed (10). Deviations from normality lead to bias in the calculation of weights in MLE and add extraneous variability to the data. Because of the uncertainties about meeting this assumption of normality, and because of the added time and effort required for MLE, the use of the MLE procedure cannot be recommended.

The mostly additive nature of inheritance and the medium-to-high estimates of heritability in the five populations studied suggest that selection for ASR resistance in corn is practical. Mass selection, as others have suggested (22), should be an adequate way to improve populations for ASR reaction. These data also indicate that pedigree selection would also be an effective way to select for ASR resistance. Even with limited sampling of F_3 families from the five F_2 populations (30 or 40 per population), F_3 progenies with low ASR reactions and little within-plot variation could be located, which indicates that ASR reactions can be fixed early in the inbreeding process. If the objective of pedigree breeding, however, is an inbred line that has high levels of ASR resistance and general combining ability for grain yield, then many progenies would have to be evaluated to find lines with acceptable levels of both traits. Because of genotype × environment interactions and the relatively large magnitude of σ^2_E, selection based on some sort of replicated progeny test may be necessary to obtain the highest levels of resistance. Yield loss studies (34) have indicated that very high levels of resistance to ASR may not be necessary to prevent losses, although the exact level of resistance needed is not clear. Final hybrid combinations should be tested in several environments for ASR reaction and lodging resistance to ensure an adequate level of resistance.

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