Heterotic Effects of Resistance in Maize to Helminthosporium maydis Race O

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ABSTRACT

Heterotic effects of resistance to *Helminthosporium maydis* race O in maize were estimated from a set of diallel crosses involving 10 parental inbreds [i.e., P(P+1)/2 entries]. Disease evaluations, based upon disease rating on a scale of 0-100 and lesion length, were made at mid-silk following artificial inoculation.

Variations attributed to average heterosis, by line, and specific heterosis, between lines, were highly significant. Resistance was partially dominant. Resistant inbreds exhibited resistant-line effects, with inbred Pa884p exhibiting the most resistant effect for both disease evaluations. Differences existed between average heterosis effects of the disease evaluations. Based on disease rating, resistant inbreds contributed less heterotic effects for resistance to single crosses than did susceptible inbreds. Susceptible inbred Mo19 contributed more heterotic effects for resistance to single crosses than any other parental inbreds. Data on lesion length, however, showed that resistant inbreds Pa884p and Va43 contributed greater effects of average heterosis for resistance to single crosses than other inbreds. The heterotic effect for lesion length was smaller in magnitude than that for disease rating.

Disease evaluation, generally, indicated that progeny from crosses between resistant inbreds are more resistant to race O than are those involving intermediately resistant or susceptible inbreds.

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Southern corn leaf blight (SCLB), caused by *Helminthosporium maydis* Nisikado & Miyake race O, is widespread in tropical and subtropical areas. The disease often causes a significant decrease in grain yield by killing leaf tissue (9). Prior to 1970, SCLB was a relatively minor disease in the USA Corn Belt (4).

The epiphytotic of SCLB in 1970, caused by a new race (race T), resulted in a catastrophic hybrid corn yield loss. This loss exceeded 50% in the southern states of the USA (4). The cytoplasmic susceptibility of maize having Texas male-sterile cytoplasm (*cms*-T) to race T caused normal (N) cytoplasm to be returned to hybrid corn production. Good and Horner (3) reported that there is no measurable effect of different N cytoplasms on susceptibility to either race O or T. Race O does not exhibit cytoplasmic specificity in its pathogenicity. Resistance in maize to race O is known to be of nuclear gene inheritance, and is expressed both qualitatively (1, 8) and quantitatively (7). In previous studies (5, 6), I showed that resistance to race T is mainly cytoplasmic and partially nuclear-genic. Nuclear gene resistance is partially dominant and has additive genetic effects. Reactions of N cytoplasm (inbreds and hybrids) to race O were highly correlated with reactions of the same inbreds and hybrids in *cms*-T cytoplasm to race T, indicating that similar nuclear genes are probably conditioning resistance in both N and *cms*-T cytoplasms of maize (Hooker and Lim, *unpublished*). However, expression of resistance genes in *cms*-T maize to race T is partially masked by the cytoplasmic susceptibility.

Information concerning the quantitative inheritance of resistance in maize to race O is sparse, and that concerning heterotic effects on resistance is negligible. The purpose of this research was to obtain information on the genetic variation and heterotic effects of resistance in maize to race O using the diallel analysis method.

MATERIALS AND METHODS.—Ten inbred lines were selected based on their reactions in the field to *H. maydis* race O in 1971. Inbreds Pa884p, Va43, Mo12, N610, SC375, Va71, T111, and 33-16 were selected as resistant (R) or intermediately resistant (I) to race O. Inbreds Va6 and Mo19 were selected as susceptible (S).

In 1973, the parents and one set of 45 diallel crosses [i.e., P(P+1)/2 entries] were grown in a randomized complete block arrangement with three replications at the Agronomy South Farm, Urbana, Illinois. Each plot (row) contained 12 hills spaced about 30 cm apart with two plants per hill. Plots were spaced approximately 90 cm apart.

All plants were inoculated with race O at the 7- to 9-leaf stage, and again 2 weeks later. Inoculum consisted of sorghum-grain cultures. Sorghum grain was steeped in fresh tap water for 24 hours at 24 C with several washings and changes of water. Soaked grain was placed on several layers of cheesecloth in a wire basket to drain. Grain was then placed in flasks (to one-third the flask volume), plugged flasks with cotton, or diSPo plugs, and autoclaved 40 minutes at 120 C. Flasks were cooled at 24 C and the sorghum inoculated by mixing culture blocks of the fungus on potato-dextrose agar with the grain. Fungal isolates used were isolated from infected leaf tissue from the 1972 experimental plots at Urbana, Illinois. Grain cultures were shaken every 2 days and incubated 16-20 days at 24 C. Cultures were then mixed and air dried at 24 C. Inoculations were made by placing 10-20 grains into individual leaf whorls.

Excluding end hills, 10 plants per plot, one from each hill, were rated for disease reactions at mid-silk using a 0-100 scale based on the degree of infection of the entire plant. On this scale 0 designated no infection and 100, infection of the entire plant. Also, the length (in millimeters) of clearly expressed lesions on the ear-leaf of

10 plants per plot was measured at mid-silk.

The methods of Gardner and Eberhart (2) were used for the diallel analysis of heterosis. Orthogonal partitioning of the total variance among entries by the method of least squares provided a test for heterosis ($h_{jj'}$) due to differences in gene frequencies in inbred lines j and j′ and to dominance in crosses. The variation due to $h_{jj'}$ effects was further partitioned to obtain additional information as follows:

$$h_{jj} = \bar{h} + h_j + h_j + s_{jj'}$$

where $\bar{h}$ is the average heterosis contributed by the particular set of lines used in crosses; h_j is the average heterosis contributed by line j in its crosses measured as a deviation from average heterosis ($\Sigma_j h_j = 0$) and $s_{jj'}$ is the specific heterosis that occurs when line j is mated to line j′. The h_j effects were calculated for each line, and $s_{jj'}$ effects were calculated for each cross.

RESULTS.—Disease ratings (DR) of parental inbreds and F_1 hybrids were highly correlated with lesion lengths (LL); $r = 0.73$, $P < 0.01$. The analyses of variance summaries for DR and LL in the diallel cross are presented in Table 1. Variations due to the inbred line effects (L_j), which are deviations from the mean of all parental inbreds involved, and $h_{jj'}$ effects were highly significant for resistance in maize to race O determined by either DR or LL. The mean squares for the $\bar{h}$ effects are attributable to nonadditive genetic effects and were large. Variations due to h_j and $s_{jj'}$ effects were also highly significant for DR and LL indicating that both additive and nonadditive genetic effects are significant in the inheritance of the disease resistance to race O in this set of diallel crosses. The coefficient of variability (C.V.) of DR was similar to that of LL thus indicating the variation in each disease evaluation was relatively similar.

Means of DR for parental inbreds and F_1 hybrids are presented in Table 2. Estimates of L_j, h_j, and $s_{jj'}$ effects are

TABLE 1. Analyses of variance for heterotic effects of disease reaction in the diallel crosses of corn inbreds to *Helminthosporium maydis* race O [i.e., P(P+1)/2 entries]

Source of variation	d.f.	Mean squares: Disease rating (0-100)[a]	Mean squares: Lesion length (mm)
Replication	2	274.70	9.46
Entries	54	397.21**[b]	16.82**
Parental lines (L_j)	9	1,141.54**	66.60**
Heterosis ($h_{jj'}$)	45	248.34**	6.86**
Average heterosis ($\bar{h}$)	1	7,644.86**	10.43**
Line heterosis (h_j)	9	88.87**	9.22**
Specific heterosis ($s_{jj'}$)	35	78.02**	6.16**
Error	108	22.23	2.44
Total	164		
C. V. (%)		14.70	13.30

[a]Scale of 0-100; 0 designates no infection and 100 designates infection of the entire plant.
[b]Indicates significant difference, $P = 0.01$.

TABLE 2. Means of disease ratings (scale of 0-100, with 100 = infection of the entire plant) for 10 parental corn inbreds and for 45 hybrids infected with *Helminthsporium maydis* race O (hybrids above the diagonal; inbreds on the diagonal), and estimates of inbred line effects (L_j), average (h_j) and specific heterosis ($s_{jj'}$) (below the diagonal)

	Pa884p	Va43	Mo12	N610	Va6	Mo19	SC375	Va71	T111	33-16	Mean of crosses
Pa884p	23.3	20.0	18.3	23.3	25.0	21.7	25.0	18.3	18.3	26.7	21.84
Va43	6.69	30.0	20.0	18.3	30.0	26.7	20.0	16.7	20.0	26.7	22.04
Mo12	− 1.23	0.23	38.3	28.3	35.0	40.0	31.7	26.7	23.3	25.0	27.58
N610	3.56	− 1.85	1.90	46.7	26.7	48.3	30.0	20.0	23.3	33.3	27.94
Va6	− 3.10	1.69	0.44	− 8.31	61.7	33.3	36.7	38.3	41.7	50.0	35.18
Mo19	− 5.18	− 0.39	6.69	14.61	− 8.52	71.7	36.7	31.7	30.0	38.3	34.07
SC375	2.31	− 2.89	2.52	0.44	− 1.02	0.23	46.7	31.7	26.7	35.0	30.38
Va71	− 1.43	− 3.31	0.44	− 6.64	3.56	− 1.85	2.31	48.3	35.0	31.7	27.78
T111	− 1.85	− 0.39	− 3.31	− 3.73	6.48	− 3.93	− 3.10	8.15	50.0	35.0	28.14
33-16	0.44	0.23	− 7.68	0.23	8.77	− 1.64	− 0.81	− 1.23	1.69	48.3	33.52
L_j effects	23.17	−16.50	− 8.17	0.17	15.17	25.17	0.17	1.83	3.50	1.83	
h_j effects	3.71	0.58	2.67	− 1.08	− 0.46	− 6.71	1.62	− 2.12	− 2.54	4.33	

$SE^a = 0.86$; $SE^b = 1.22$

[a]SE = standard error of any treatment mean.
[b]SE = standard error of the difference between two treatment means.

also included. A negative (−) effect implies effects for resistance and a positive (+) effect implies effects for susceptibility. Significant differences among inbreds and hybrids were found for DR. The correlation coefficient (r) between parental means and hybrid means was highly significant ($r = 0.89$, $P < 0.01$) for DR. Resistant inbreds Pa884p, Va43 and Mo12 all had significantly lower DR than other inbreds. This was also reflected in crosses involving these inbreds. They all exhibited negative L_j effects. Inbred Pa884p was the most R parent. Negative h_j effects were found from rather I or S inbreds. Inbred Mo19 was the most susceptible parent, and had the greatest negative estimate of h_j effect. The greatest negative $s_{jj'}$ effect was found in the cross Mo19 × Va6 (S × S). Crosses of N610 × Va6 (I × S), Mo12 × 33-16 (R × I), Va71 × N610 (I × I) and Pa884p × Mo19 (R × S) all exhibited large negative $s_{jj'}$ effects.

LL means, estimates of L_j, h_j, and $s_{jj'}$ for LL are given in Table 3. Significant differences in LL were found among inbreds and hybrids. Inbreds Pa884p, Va43, and Mo12 were resistant parents and had shorter LL than other inbreds. The r value between parental means and hybrid means for LL was highly significant ($r = 0.81$, $P < 0.01$). Estimates of L_j effects are similar to those for DR. Inbred Pa884p had the greatest negative L_j effect. The r value between L_j effects of DR and LL was highly significant ($r = 0.87$, $P < 0.01$). For LL, however, resistant inbreds Pa884p and Va43 exhibited negative h_j effects. Intermediately resistant inbreds N610 and Va71 also showed negative h_j effects but to a lesser extent than resistant inbreds. No susceptible inbred exhibited a negative h_j effect for LL. The r value between h_j effects for DR and LL was not significant ($r = 0.17$). The cross Pa884p × Va6 (R × S) had the greatest negative $s_{jj'}$ effect. Large negative $s_{jj'}$ effects were also estimated from crosses of Mo12 × 33-16 (R × I), Mo19 × Va6 (S × S), Mo 12 × T111 (R × I), Va81 × 33-16 (I × I), and Pa884p × T111 (R × I). The several incidences of greater negative than positive $s_{jj'}$ effects for both DR and LL, indicated the presence of nonadditive genetic effects and partial dominance of resistance.

DISCUSSION.—The highly significant h_j and $s_{jj'}$ effects in this experiment indicate that both additive and nonadditive genetic effects are important in SCLB resistance.

Data from both DR and LL suggest that an evaluation of the inbreds per se would give a good prediction of the resistance of hybrids from these inbreds. This is indicated by the highly significant correlation coefficients between parental means and F_1 hybrid means. Resistant inbreds all showed negative L_j effects and Pa884p exhibited the most negative effect of all inbreds for both DR and LL. However, results of DR showed that resistant inbreds did not contribute any more resistance to single crosses than did either intermediately resistant or susceptible parental inbreds having negative h_j effects. Susceptible inbred Mo19 contributed more heterotic effects for resistance to its single crosses than any other parental inbred. The resistant (−) h_j effect for Mo19, however, was not predicted from the DR of the inbred per se and was caused probably by a highly negative $s_{jj'}$ effect for the single cross Mo19 × Va6. The high degree of resistance in single crosses between susceptible inbreds indicates that resistance is highly associated with heterosis. The occurrence of highly negative $s_{jj'}$ effects suggests that the DR of some single crosses are lower than that predicted by their parental means. Results of LL showed that resistant inbreds Pa884p and Va43 exhibited negative h_j effects, whereas susceptible inbreds Mo19 and Va6 had positive effects. The $s_{jj'}$ effects for LL among parental inbreds were generally similar to those for DR. However, the $s_{jj'}$ effects for LL were smaller in magnitude than those obtained for DR. Single crosses among susceptible inbreds did not exhibit a high degree of heterosis for resistance.

TABLE 3. Means of lesion length (mm) for 10 parental corn inbreds and for 45 hybrids infected with *Helminthosporium maydis*[•] race O (hybrids above the diagonal; inbreds on the diagonal), and estimates of inbred line effects (L_j), average (h_j) and specific ($s_{jj'}$) heterosis (below the diagonal)

	Pa884p	Va43	Mo12	N610	Va6	Mo19	SC375	Va71	T111	33-16	Mean of crosses
Pa884p	9.7	8.3	8.7	9.0	8.0	10.0	10.3	8.3	7.7	11.0	9.03
Va43	1.85	10.3	9.3	8.3	12.7	10.7	8.7	8.0	9.3	11.3	9.62
Mo12	− 0.06	− 0.06	10.7	12.0	15.7	14.7	12.0	11.0	10.0	11.3	11.63
N610	1.23	− 0.11	1.31	12.3	10.7	14.7	11.3	9.0	10.0	12.0	10.77
Va6	− 2.73	1.27	2.02	− 2.02	14.7	13.3	13.3	14.3	16.0	16.7	13.41
Mo19	− 0.40	− 0.40	1.35	2.31	− 1.98	14.7	14.3	12.7	12.3	15.3	13.11
SC375	0.89	− 1.44	− 0.36	− 0.06	− 1.02	0.31	11.3	13.3	12.3	14.7	12.24
Va71	− 0.06	− 1.06	− 0.31	− 1.36	1.02	− 0.31	1.31	13.3	13.7	11.7	11.33
T111	− 1.23	− 0.23	− 1.81	− 0.86	2.18	− 1.15	− 0.19	2.19	12.3	14.7	11.77
33-16	0.52	0.18	− 2.06	− 0.44	1.27	0.27	0.56	− 1.40	1.10	13.3	13.18
L_j effects	− 2.60	− 1.93	− 1.60	0.07	2.40	2.40	− 0.93	1.07	0.07	1.07	
h_j effects	− 1.60	− 1.27	0.82	− 0.97	0.82	0.48	1.19	− 0.85	0.15	1.23	

$SE^a = 0.28$; $SE^b = 0.40$

[a]SE = standard error of any treatment mean.
[b]SE = standard error of the difference between two treatment means.

The h_j effects obtained from DR and LL contain useful information. No resistant inbred exhibited a negative h_j effect for DR, but some of them exhibited a negative h_j effect for LL. The discrepancy between h_j effects of DR and LL are possibly attributable to (i) variations between the two methods of determining disease reaction, or (ii) the amount of infection and LL are each influenced by separate genetic systems in maize. In addition, because inbreds and hybrids were randomized within the same block, differences in DR between inbreds and hybrids are confounded with competition effects that may have resulted in higher DR for inbreds than if each inbred had been grown in pure stands.

Data from both DR and LL showed that resistant inbreds all exhibit negative L_j effects, and that crosses involving these inbreds were the most resistant to *H. maydis* race O. This was true even though contributions of heterotic effects for resistance were smaller for single crosses of resistant than of susceptible inbreds. Previously, I (6) indicated that the nuclear gene resistance in both *cms*-T- and N-cytoplasm corn to race T was associated with highly significant additive genetic effects and was partially dominant. A highly resistant inbred always exhibited much greater additive than nonadditive genetic effects regardless of the methods used to determine disease reactions. It should be emphasized that genetic interpretations from my study with a fixed diallel set of 10 parental inbreds are valid for this set only. Different genetic populations may exhibit different types of gene action.

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