Inheritance of Resistance to *Bipolaris maydis* Race O in Crosses Derived from Nine Resistant Inbred Lines of Maize

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ABSTRACT

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The inheritance of resistance in maize (*Zea mays*) to southern corn leaf blight caused by *Bipolaris maydis* race O was studied in 1981 and 1982. In 12 families derived from crosses of nine resistant inbreds and three susceptible inbreds, additive genetic effects were highly significant, accounting for 49.0 to 96.9% of the total variation. Significant dominance genetic effects were detected in all but one family, but accounted for only 2.5 to 46.9% of the total variation. Estimates of broad-sense heritabilities ranged from 29.5 to 69.1% and the estimated numbers of effective factors ranged from 2.2 to 14.6. Pedigree and recurrent selection methods should be effective in breeding for improved resistance to southern corn leaf blight.

Additional key words: generation mean analysis, corn.

Southern corn leaf blight (SCLB) is a disease of maize (*Zea mays* L.) caused by the fungus *Bipolaris maydis* (Nisik.) Shoemaker (= *Helminthosporium maydis* Nisikado and Miyake). The disease is widely distributed in regions of the world having warm and humid climates (20). *B. maydis* has two distinct races, designated O and T, which differ in their specificity for plant cytoplasms, symptoms induced, the type and amount of toxin produced, optimum growth temperatures, and reproductive rates (7). In most years, SCLB causes little or no yield loss in the United States. In years favorable for disease development, infections by race O may reduce yields up to 25% on susceptible hybrids (4,21). Yield losses of up to 71% were recorded on susceptible hybrids in the SCLB epidemic caused in 1970 by race T of *B. maydis* (9). With the decreased use of Texas male-sterile cytoplasm in commercial hybrid seed corn production, race T is now rarely found in the United States Corn Belt (18), but race O is commonly found every year from the southern states to the central Corn Belt in the United States. The common occurrence and potential destructiveness of this disease have caused commercial seed producers to use fungicide sprays routinely in seed production fields, generally in conjunction with insecticide sprays for ear worm control.

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Bipolaris maydis race O can be controlled by fungicide sprays or by plowing to bury crop residues, but the most efficient and economical means of control is host genetic resistance. Pate and Harvey (16) studied the inheritance of resistance to *B. maydis* in a number of North Carolina inbreds and hybrids in 1954. They observed that inheritance of resistance was apparently quantitatively controlled and that resistance in single crosses was partially dominant. Lim (11) and Lim and Hooker (12) showed that additive gene action was much greater than nonadditive effects, but neither study used an experimental design or genetic model that would allow estimation of heritability, numbers of effective factors or the relative importance of additive, dominance and epistatic gene action. Recently, Thompson and Bergquist (19) concluded that resistance in inbred NC250 to *B. maydis* race O is controlled by relatively few loci, that additive genetic effects are of primary importance and that this resistance may be incorporated into susceptible inbreds by backcrossing. Information on the inheritance of quantitative resistance to *B. maydis* in families derived from other inbreds is still very limited.

The objectives of this study were to determine the inheritance of resistance to *B. maydis* in a number of families derived from crosses of nine diverse, resistant inbreds with three susceptible inbreds, determine the gene action that influences this resistance, estimate the heritability of resistance and estimate the number of effective factors (3) that control this resistance.

MATERIALS AND METHODS

More than 1,200 maize inbreds were evaluated for reaction to *B. maydis* race O in disease nurseries in 1979 and 1980. From these inbreds, twelve were selected for use in this study. Nine inbreds were chosen as resistant parents (B80, C127, CH24, CI40, FR18s, FR43, FRT8, TR110, and 75ROLL) and three were selected as susceptible parents (FR632, FRB73, and TR227). Resistant (P_1) and susceptible (P_2) inbreds were crossed to obtain F_1 hybrids. Appropriate selfs and crosses were made to obtain the F_2 and the backcrosses to the resistant (B_1) and susceptible (B_2) parents. Since resistant parents were involved in crosses with one, two or all three of the susceptible parents, the families will be designated by their F_1 hybrid pedigrees, with the resistant parent always being listed first (e.g., FR43 × FR632).

The six generations of each of a number of families were grown at the Agronomy and Plant Pathology South Farm, Urbana, IL, in 1981 and 1982. Because of the large number of crosses and the number of growing seasons necessary to obtain seed of all crosses, not all families were grown in both years of this study (Table 1).

The generations of each family were planted in a randomized complete block design with five replications. Nonsegregating generations (P_1, P_2, and F_1) were planted in one-row plots 4.2 m long with 76-cm row spacings and a maximum of 24 plants per row. Segregating generations (B_1, B_2, and F_2) were planted using the same row length, spacing and plant populations, but the plots consisted of four rows for B_1 and B_2 and eight rows for the F_2 generation.

TABLE 1. Maize families used to study the inheritance of resistance to *Bipolaris maydis* race O in 1981 and 1982 at the Agronomy and Plant Pathology South Farm, Urbana, IL

1981	1982
	B80 × FR632
CH24 × FR632[a]	CH24 × FR632
CI40 × FR632	CI40 × FR632
FR43 × FR632	FR43 × FR632
TR110 × FR632	TR110 × FR632
75ROLL × FR632	75ROLL × FR632
	C127 × FRB73
	FR18s × FRB73
	FR43 × FRB73
	FRT8 × FRB73
	TR110 × FRB73
	FR43 × TR227

[a] In each cross, the resistant inbred is listed first, the susceptible inbred second.

Bipolaris maydis race O was isolated from diseased leaf tissue collected at Urbana, IL, in the fall of 1980. The isolate was preserved on silica gel and used to produce inoculum in 1981 and 1982. Inoculum was produced by culturing *B. maydis* on moistened sterile oat and sorghum seeds for 10–14 days. All plants were inoculated by placing approximately 50 ml of the infected seed in the leaf whorls of plants at the four- to five-leaf stage. Plots were inoculated twice in 1981 and 1982; the second inoculation was approximately 1 wk after the first. Individual plants were rated, using visual estimates of percent leaf tissue infected, beginning approximately 1 wk prior to anthesis and continuing on a weekly basis for 4 wk in 1981 and 7 wk in 1982 or until plant senescence within a family made further ratings unreliable. The data were arc sine transformed, as recommended for data expressed as decimal fractions or percentages (17). Transformed generation means over all families studied in 1981 and 1982 showed that the rating date closest to approximately 3 wk after anthesis gave the maximum separation of resistant and susceptible inbreds with minimum standard errors and no premature death of the plants. Using these criteria, the data from the rating date closest to 3 wk after anthesis were used for genetic analysis.

Analyses of the transformed data were performed as described by Carson and Hooker (1,2). The model of Hayman and Mather (6) was fit to the generation means:

$$Y = m + a_1 d + a_2 h + a_3 i + a_4 j + a_5 l$$

in which Y is the mean of the generation, m is the intercept, d is the pooled additive effect, h is the pooled dominance effect, i is the pooled additive × additive epistatic effect, j is the pooled additive × dominance epistatic effect, l is the dominance × dominance epistatic effects, and $a_1 - a_5$ are the coefficients of the equations of expectation (Table 2). The coefficients are defined as the deviations from the F_2 mean, and are the relative contributions of these effects to each generation mean (5). F-values in the analysis of variance of generation means were used to test the significance of the genetic effects and any deviations from the model. For the five populations studied over 2 yr, the significance of the generations × year mean square was tested with a pooled error term (generations × blocks within years). When the generation × year mean square was significant, it was used to test genetic effects over years.

Second order statistics were used to estimate genetic and environmental variances directly. Within-plot variances of each generation were obtained by pooling sums of squares and degrees of freedom of all plots containing the same generation. Additive genetic variances were calculated (2) by:

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

in which V_{F_2}, V_{B_1}, and V_{B_2} are the respective variances of the F_2, B_1, and B_2 generations. Dominance genetic variances were then directly estimated (2) by:

TABLE 2. Coefficients of terms in the equations of expectation for generation means in terms of additive (d), dominance (h), additive × additive (i), additive × dominance (j), and dominance × dominance (l) genetic effects in the model $Y = m + a_1 d + a_2 h + a_3 i + a_4 j + a_5 l$

Y	a_1	a_2	a_3	a_4	a_5
P_1	1	−1/2	1	−1	1/4
P_2	−1	−1/2	1	1	1/4
F_1	0	1/2	0	0	1/4
F_2	0	0	0	0	0
B_1	1/2	0	1/4	0	0
B_2	−1/2	0	1/4	0	0

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

in which σ^2_E is the environmental variance obtained by pooling the within-plot variance of the P_1, P_2, and F_1 generations. These estimates of additive and dominance genetic variance were used to calculate broad-sense heritability values:

$$h^2_b = (\sigma^2_A + \sigma^2_D)/V_{F_2} \times 100.$$

The number of effective factors was calculated by the Castle-Wright formula (3,22) as used by Carson and Hooker (1):

$$K_1 = (\text{phenotypic range in the } F_2)^2/8(\sigma^2_{F_2} - \sigma^2_E)$$

in which K_1 is the number of effective factors and the variances are equivalent to those described above.

RESULTS

Generation means for disease reaction to SCLB in 1981 and 1982 showed disease severity of the resistant inbred was always lower than the disease severity of the corresponding susceptible inbred (Table 3). Significant negative deviations of the F_1 mean from the midparent value were found in all families in 1981 and 1982. The F_1 means were significantly lower than the F_2 means in all families in both years except in the 75ROLL × FR632 family in 1981 and in the B80 × FR632, FRT8 × FRB73, and TR110 × FRB73 families in 1982. The B_1 means were all less than the F_2 means, but the differences were not significant in the 75ROLL × FR632 family in 1981 and in the B80 × FR632 and CI40 × FR632 families in 1982. The B_2 means were significantly higher than the F_2 means in all families in 1981. The 1982 B_2 means were higher than the F_2 means except in the B80 × FR632 family, but the differences were not significant in the C127 × FRB73, CI40 × FR632, FRT8 × FRB73, FR43 × FR632, and FR43 × TR227 families. The B_2 means were significantly greater than the B_1 means in all families in both years except in the CI40 × FR632 and B80 × FR632 families in 1982.

The frequency distribution in F_2 populations showed transgressive segregation for resistance in the 75ROLL × FR632 and CI40 × FR632 families in 1982. Transgressive segregation for susceptibility was observed in the CH24 × FR632 family in 1981 where the B_2 segregates exceeded the susceptible parent in disease severity. The B_1 frequency distribution in all families was highly skewed towards increased resistance.

When the Hayman and Mather model was fit to the data, highly significant additive genetic effects were evident in the families studied in 1981 and 1982 (Table 4) and in the families studied only in 1982 (Table 5). Highly significant dominance genetic effects also were detected in all but two families. In the TR110 × FRB73 family, the dominance genetic effect was significant at $P = 0.05$. No significant dominance effect was detected in the FRT8 × FRB73 family. Significant deviations from the model, indicating epistasis, were found in 1981 in the 75ROLL × FR632 family. No significant deviations were observed in this family in 1982. Significant deviations were found in 1982 in the CH24 × FR632 and FRT8 and FRB73 families. Highly significant deviations were seen in 1982 in the C127 × FRB73, B80 × FR632, and CI40 × FR632 families. The analysis of variance of data combined over 1981 and 1982 (Table 4), showed no significant deviations in the CH24 × FR632, CI40 × FR632, or 75ROLL × FR632 families but showed highly significant differences between years. The generation × year interactions were highly significant in the CH24 × FR632, CI40 × FRB73, and 75ROLL × FR632 families. In the FR43 × FR632 and TR110 × FR632 families, the lack of significant interaction between generations and years allowed the use of a pooled error term (generations × blocks within years).

The percent of variation in disease reaction among transformed generation means accounted for by the different genetic effects in the families studied in 1981, 1982 and combined over both years are given in Table 6. Similar data are given for those families studied only in 1982 in Table 7.

Direct estimates of the additive, dominance and environmental variances are given in Table 8. Broad sense heritabilities and numbers of effective factors calculated from these estimates are given in Table 9.

TABLE 3. Mean southern corn leaf blight disease severities (percent leaf tissue infected) of six generations and deviations of the F_1 mean from the midparent value 3 wk after anthesis in five maize families in 1981 and 12 maize families in 1982

Family	Year	P_1	B_1	F_1	F_2	B_2	P_2	FLSD[a]	F_1-MP[b]
CH24 × FR632	1981	19.5	19.0	17.5	22.0	29.6	35.9	3.00	−10.2*[c]
	1982	7.4	9.1	9.2	11.8	13.4	21.9	1.40	−5.45*
CI40 × FR632	1981	17.8	15.5	16.3	22.4	27.6	39.0	3.30	−12.1*
	1982	12.3	13.3	11.0	15.4	16.3	29.1	3.22	−9.70*
FR43 × FR632	1981	13.5	14.4	15.7	18.5	23.1	31.3	2.61	−6.70*
	1982	10.9	11.3	10.8	14.2	16.3	26.1	2.76	−7.70*
TR110 × FR632	1981	13.5	13.8	13.2	18.4	21.7	28.5	2.88	−7.80*
	1982	9.4	11.8	11.9	14.6	18.3	26.1	2.33	−5.85*
75ROLL × FR632	1981	22.4	20.7[d]	19.6	21.0	24.1	30.1	2.11	−6.65*
	1982	10.2	10.4	10.0	12.9	15.5	24.6	1.82	−7.40*
B80 × FR632	1982	10.1	10.4	12.4	13.2	12.0	26.6	3.11	−5.95*
C127 × FRB73	1982	15.6	18.6	15.2	24.6	26.0	27.6	2.62	−6.40*
FRT8 × FRB73	1982	9.7	9.0	16.3	17.4	18.7	27.4	4.07	−2.25*
FR18s × FRB73	1982	8.8	10.8	11.6	15.4	22.1	30.7	2.09	−8.15*
FR43 × FRB73	1982	10.5	11.7	11.4	15.2	18.1	27.5	2.63	−7.60*
FR43 × TR227	1982	10.5	10.6	9.7	13.3	14.2	21.2	2.57	−6.15*
TR110 × FRB73	1982	10.6	13.9	17.8	19.4	26.3	31.7	2.04	−3.35*

[a] Fisher's Least Significant Difference at the $P = 0.05$ level of probability.
[b] Mid-parent value = $(P_1 + P_2)/2$.
[c] Asterisk (*) indicates statistical significance at the $P = 0.05$ level of probability.
[d] Disease severity based on only one replication.

TABLE 4. Analysis of variance of arc sine-transformed generation means for reaction of five maize families to *Bipolaris maydis* race O 3 wk after anthesis in 1981, 1982, and combined over both years

Year	Source of variation	Means squares for families[a]				
		CH24	CI40	FR43	TR110	75ROLL
1981						
	Block	1.33	0.14	0.81	0.94	0.47
	Generations					
	Additive	101.61**[b]	161.65**	103.97**	74.77**	19.21**
	Dominance	37.25**	58.00**	17.87**	21.10**	18.37**
	Deviations	1.22	0.30	0.05	0.21	0.53*
	Error	0.57	0.69	0.41	0.50	0.27
1982						
	Block	0.73*	0.17	1.29	0.31	0.60*
	Generations					
	Additive	57.07**	70.31**	64.74**	82.44**	59.52**
	Dominance	11.81**	37.32**	23.47**	12.44**	21.73**
	Deviations	0.73*	2.31**	0.63	0.28	0.44
	Error	0.11	0.63	0.45	0.32	0.20
Combined						
	Block (year)	0.21	0.45	0.38	0.56	0.86
	Year	217.64**	74.94**	31.05**	12.32**	127.20**
	Generations					
	Additive	155.49**	222.59**	166.40**	157.11**	73.17**
	Dominance	45.51**	94.18**	41.15**	32.97**	40.03**
	Deviations	0.21	1.00	0.46	0.18	0.77
	Gen × Year	2.39**	3.06**	0.63	0.32	1.25**
	Pooled Error	0.34	0.66	0.43	0.41	0.23

[a] FR632 was the susceptible parent in these five families.
[b] Asterisks (*) and (**) indicate statistical significance at $P = 0.05$ and 0.01, respectively.

TABLE 5. Analysis of variance of arc sine-transformed generation means for reaction to *Bipolaris maydis* race O 3 wk after anthesis in seven maize families studied only in 1982

Source	Families					
	B80[a]		C127[b]		FR18s[b]	
	df	ms	df	ms	df	ms
Block	4	1.33	4	0.54	4	1.05*[c]
Generations	5		5		5	
Additive	1	64.24**	1	51.75**	1	158.24**
Dominance	1	19.01**	1	8.43**	1	24.46**
Deviations	3	6.48**	3	5.97**	3	0.18
Error	20	0.58	20	0.41	20	0.26

	FR43[b]		FR43[d]		FRT8[b]	
	df	ms	df	ms	df	ms
Block	4	0.12	4	0.74	4	0.57
Generations	5		5		5	
Additive	1	84.72**	1	31.89**	1	106.22**
Dominance	1	21.77**	1	14.07**	1	3.84
Deviations	3	0.35	3	0.33	3	2.85*
Error	20	0.42	20	0.39	20	0.95

	TR110[b]	
	df	ms
Block	4	1.58*
Generations	5	
Additive	1	156.26**
Dominance	1	4.09*
Deviations	3	0.31
Error	20	0.25

[a] FR632 was used as the susceptible parent.
[b] FRB73 was used as the susceptible parent.
[c] Asterisks (*) and (**) indicate statistical significance at $P = 0.05$ and 0.01, respectively.
[d] TR227 was used as the susceptible parent.

TABLE 6. Percentages of variation in reaction to *Bipolaris maydis* race O among arc sine-transformed generation means accounted for by additive and dominance genetic effects and deviations from the additive-dominance model in 1981, 1982, and combined over both years

	Family[a]				
	CH24	CI40	FR43	TR110	75ROLL
1981 Effect					
Additive	70.9	73.3	85.2	77.5	49.0
Dominance	26.0	26.3	14.7	21.9	46.9
Deviations	3.1	0.4	0.1	0.6	4.1
1982 Effect					
Additive	80.3	61.4	71.8	86.1	72.1
Dominance	16.6	35.6	26.1	13.0	26.3
Deviations	3.1	6.0	2.1	0.9	1.6
Combined effect					
Additive	77.1	69.6	79.6	82.4	63.3
Dominance	22.6	29.6	19.7	17.3	34.7
Deviations	0.3	1.0	0.7	0.3	2.0

[a] FR632 was used as the susceptible parent in these families.

TABLE 7. Percentages of variation in reaction to *Bipolaris maydis* race O among arc sine-transformed generation means accounted for by additive and dominance genetic effects and deviations from the additive-dominance model in maize families studied in 1982 only

Family	Genetic effect		
	Additive	Dominance	Deviations
B80 × FR632	62.6	18.5	18.9
C127 × FRB73	66.3	10.8	22.9
FR18s × FRB73	86.4	13.3	0.3
FR43 × FRB73	78.8	20.2	1.0
FRT8 × FRB73	89.6	3.2	7.2
TR110 × FRB73	96.9	2.5	0.6
FR43 × TR227	67.9	30.0	2.1

TABLE 8. Direct estimates of the additive genetic variance (σ^2_A), dominance genetic variance (σ^2_D), the environmental variance (σ^2_E) and their standard errors for reaction to *Bipolaris maydis* race O after artificial inoculations in 12 maize families based on data collected 3 wk after anthesis

Family	Year	σ^2_A	σ^2_D	σ^2_E
CH24 × FR632	1981	−1.0818 ± 0.1468	1.8507 ± 0.1430	1.4215 ± 0.1201
	1982	0.966 ± 0.0611	0.1006 ± 0.0437	0.964 ± 0.0877
CI40 × FR632	1981	2.4825 ± 0.4139	−0.1959 ± 0.2678	2.6527 ± 0.6325
	1982	0.9229 ± 0.1343	−0.094 ± 0.1019	1.9838 ± 0.2865
FR43 × FR632	1981	−1.0974 ± 0.1201	0.6623 ± 0.1365	2.4713 ± 0.5298
	1982	1.1652 ± 0.1267	−0.1566 ± 0.0928	1.7017 ± 0.2345
TR110 × FR632	1981	−0.4761 ± 0.0715	0.1472 ± 0.0895	1.9971 ± 0.4258
	1982	0.6367 ± 0.1036	0.0709 ± 0.0827	1.6207 ± 0.1485
75ROLL × FR632	1981	...[a]	...	...
	1982	1.3844 ± 0.1164	−0.5242 ± 0.0871	1.5048 ± 0.1851
B80 × FR632	1982	−3.0759 ± 0.0233	−1.2958 ± 0.0233	1.097 ± 0.1222
C127 × FRB73	1982	2.1406 ± 4.0403	−0.4843 ± 1.1263	2.2919 ± 0.4763
FRT8 × FRB73	1982	2.6951 ± 0.0980	−1.6921 ± 0.0685	1.7089 ± 0.2634
FR18s × FRB73	1982	2.081 ± 0.1394	0.0767 ± 0.0766	0.9532 ± 0.0511
FR43 × FRB73	1982	1.755 ± 0.1053	−0.5098 ± 0.0670	1.4684 ± 0.1568
FR43 × TR227	1982	0.8837 ± 0.0825	−0.0194 ± 0.1606	1.2922 ± 0.1434
TR110 × FRB73	1982	2.0016 ± 0.1596	0.0931 ± 0.0867	1.1901 ± 0.0895

[a] B_1 generation had missing replications so no estimate can be made.

DISCUSSION

Resistance to *B. maydis* race O was predominantly due to additive genetic effects in all families studied. Dominance genetic effects, while significant in all but one family, accounted for a relatively small percentage of the total genetic variation. Epistasis, measured as deviations from the model, seemed to be of minor importance. These conclusions were similar to those of Lim (11) who found that crosses with highly resistant inbreds always had much greater additive than nonadditive genetic effects and to those of Lim and Hooker (12), who concluded that additive gene action was of major importance in a set of diallel crosses. Generation means analysis may, however, underestimate the importance of genetic dominance (1,13). Significant negative deviations of the F_1 mean from the midparent were observed in all families in this study. Skewness of the B_1 towards resistance and the generally smaller variances of the B_1 in comparison to the B_2 both provide additional support for the potential significance of genetic dominance (1,13).

Epidemics of SCLB are highly influenced by environmental conditions. Both 1981 and 1982 had warm and wet growing conditions favorable for disease development at Urbana, yet the combined analysis of variance for the five families studied in these years showed a highly significant difference between years. The generation × year interaction term, which can be interpreted as being the genotype × environment interaction, was significant in three of the five families (75ROLL × FR632, CI40 × FR632, and CH24 × FR632) studied in both years. These results suggest that evaluation of genetic materials for resistance to *B. maydis* race O should take place over a number of different environments in a number of years.

The presence of transgressive segregation suggests that the susceptible inbred parents carry some genes for resistance and that by crossing inbreds intermediate in their reaction to *B. maydis* race O, hybrids with greater disease resistance than the more resistant parent may be produced. Similar transgressive segregation has been reported by Carson and Hooker (1) for anthracnose leaf blight of corn. The significance of transgressive segregation in this study cannot easily be determined.

The genetic model used in generation mean analysis has been used in several studies on the inheritance of quantitative resistance to plant disease (1,2,8,10,14). As with all genetic models, the use of this one requires that certain statistical and genetic assumptions be made. Statistical assumptions are those of analysis of variance (17). Genetic assumptions are: no selection which favors particular gametes, no linkage between interacting loci, normal Mendelian segregation of alleles, the absence of mutation and the

TABLE 9. Broad-sense heritability (h^2_b) estimates and the number of effective factors (K_1) controlling resistance to *Bipolaris maydis* race O based on arc sine-transformed data 3 wk after anthesis in 12 maize families

Family	Year	h^2_b	K_1
CH24 × FR632	1981	35.1	14.6
	1982	47.3	9.0
CI40 × FR632	1981	45.7	2.2
	1982	29.5	9.4
FR43 × FR632	1981	†[a]	†
	1982	37.2	7.7
TR110 × FR632	1981	†	†
	1982	30.4	11.0
75ROLL × FR632	1981	††[b]	††
	1982	36.4	9.1
B80 × FR632	1982	†	6.2
C127 × FRB73	1982	41.9	6.8
FRT8 × FRB73	1982	36.1	11.3
FR18s × FRB73	1982	69.1	7.1
FR43 × FRB73	1982	45.9	6.3
FR43 × TR227	1982	40.1	3.2
TR110 × FRB73	1982	63.8	2.4

[a] † Negative estimates were obtained in these families.
[b] †† B_1 generation had missing replications so no estimate can be made.

isodirectional distribution of genes for resistance and susceptibility between the two parental lines. This last assumption is one which may pose the greatest difficulty. The presence of transgressive segregation for resistance in two families indicates that this assumption may be false in other families as well. The lack of isodirectional distribution of genes between the parental lines could result in significant deviations from the additive dominance model. These deviations, which would indicate digenic or higher order epistasis, would result in biased estimates of the additive and dominance genetic variances. This is a major limitation to the use of generation means analysis. In this study, epistasis was detected in the 75ROLL × FR632 family in 1981 and in the B80 × FR632, C127 × FRB73, CH24 × FR632, CI40 × FR632, and FRT8 × FRB73 families in 1982. If families within years were examined separately, epistasis was detected in six of 17 family-year combinations. When the analysis was combined over 2 yr in the 75ROLL × FR632, CI40 × FR632, and CH24 × FR632 families in which epistasis was detected, epistatic effects were no longer significant. Apparently epistasis was responsible for bias in some of the direct estimates of the additive and dominance genetic variances. Since epistasis may have been present at undetectable levels in other families, only broad-sense heritabilities were calculated.

Direct estimates of the additive and dominance genetic variances were apparently biased by factors other than epistatic effects in some families, particularly in families for which negative estimates of the additive genetic variance were obtained. This bias was probably due to the effects of linkage, which cannot be detected in generation means in the absence of epistasis. Assuming no significant epistatic effects in this study, the effects of linkage on reaction to *B. maydis* cannot be determined. Another limitation of generation means is that the cancelling effects of genes for resistance and susceptibility may prevent real genetic effects from being detected. The magnitude of this problem was impossible to determine from this study.

Breeding for improved resistance to SCLB can be implemented without major modification of most breeding programs. Numerous sources of resistance have been identified and many appear to be quantitatively inherited. For the inbreds used in this study, heritabilities ranged from low to high and resistance was largely due to additive genetic effects. The predominance of additive genetic variability in this study provides data that support the observation of Pate and Harvey (16) that resistance to *B. maydis* race O is fixed relatively early in the selfing process in the development of inbreds, generally by the S_2 or S_3 generations. A pedigree breeding program with intensive disease screening of S_2 and S_3 lines over a number of environments should allow selection of lines with relatively high levels of resistance, as well as other agronomically desirable traits. Recurrent selection techniques should also be useful in the development of highly resistant populations which could be used to develop high-yielding, resistant inbreds and hybrids (15).

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