The Inheritance of Resistance in Corn to *Cochliobolus carbonum* Race 3

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ABSTRACT

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Diallel cross analyses were used to study the nature of gene action governing resistance in corn (*Zea mays*) to an isolate of *Cochliobolus carbonum* (=*Helminthosporium carbonum*) race 3. Disease efficiency, lesion length, and sporulation capacity were used as resistance parameters when seedlings were screened for resistance in growth chamber studies while lesion length only was used in field studies. A total of 134 F_1 and selfed inbred parents were tested for resistance against a virulent isolate of the fungus. Additive gene action was much more important than nonadditive gene action in controlling resistance for all the three traits used in the study. Average heterosis, line heterosis, specific combining ability effects, and maternal effects were occasionally significant, but were always much smaller than the additive effects.

Additional key words: epidemiology, horizontal resistance, maize, quantitative inheritance.

Helminthosporium leaf spot of corn (*Zea mays* L.) caused by *Cochliobolus carbonum* Nelson (=*Helminthosporium carbonum* Ullstrup) race 3 has become increasingly important in most corn-growing areas in Pennsylvania and the neighboring states. Resistance to race 1 of *C. carbonum* is conditioned by a single dominant gene (16), whereas resistance to race 2 and race 3 may be under polygenic control (3,12).

The presence of *C. carbonum* race 3 was known as early as 1971 and since that time its prevalence has increased. The high severity of *C. carbonum* race 3 on susceptible corn inbreds and hybrids prompted a study on the inheritance of factors governing resistance of corn inbred lines to this pathogen.

Various types of mating designs have been developed to carry out biometrical genetic analyses. When several inbred lines varying in

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disease reaction are available, the diallel crossing technique is an efficient means of analyzing quantitative inheritance (4,8–11,13).

The objective of this research was to study, in the growth chamber and in the field, the nature of gene action governing resistance in corn to an isolate of *C. carbonum* race 3. Previous research (6,7) identified three parameters that could be utilized to estimate resistance. These are disease efficiency (DE), the number of lesions resulting from a given amount of inoculum; lesion length (LL), the length in millimeters of lesions formed on inoculated plants; and sporulation capacity (SC), the number of spores per square centimeter of diseased tissue.

MATERIALS AND METHODS

Genetic material. Inbreds were selected that represented a wide range of reactions to *C. carbonum* race 3 (2).

Three sets of diallel crosses were made because of the space limitations for the growth chamber studies. In set A, inbred lines Pa419P, W438, PaB8B, Va26, B37, H95, and W153R were used and in set B, Pa33A, W64A, Pa33, MS72, Wf9, Pa32, and A495 were used. Three inbred lines from set A (W438, H95, and W153R) and three from set B (W64A, Pa33, and Pa32) were used as parental inbreds for diallel cross set C. Seed from ears of identical crosses were bulked to represent one progeny family.

Growth chamber studies. A total of 134 progeny families (F_1s reciprocal F_1's, and inbred parents) from the three diallel crosses were screened in a growth chamber for disease resistance as measured by DE, LL, and SC. Detailed procedures for obtaining estimates of DE, LL, and SC were described previously (7). Briefly, inoculum of a highly virulent isolate (7), RS419-3, of *C. carbonum* race 3 was prepared by incubating sections of the stock cultures (greenhouse-grown corn leaf material infected with the isolate) in petri dishes containing moistened filter paper at room temperature for 5 days to induce sporulation. Spores were washed from the leaf tissue with 0.05% water agar solution and the volume was adjusted to provide $1.5–3.0 \times 10^3$ spores per milliliter.

Assessments of DE were done by placing the spore suspension on the middle of the fifth leaf of corn seedlings at the five-leaf stage with a quantitative inoculator (15). Inoculated plants were placed in a dew chamber (model DC20, Percival Refrigeration and Mfg. Co., Boone, IA 50036) for 15 hr at 21 C and then were maintained in a locally built growth chamber at 23 C with the lights on for 12 hr per day (135 $\mu E \cdot m^{-2} \cdot sec^{-1}$). The numbers of lesions produced from a quantified viable inoculum, 5 days after inoculation, were recorded. The length (in milliliters) of 10 lesions on the fifth leaf was determined 14 days after inoculation and SC was then measured after plants had been incubated in a dew chamber for 4 days at 21 C under a 12-hr light/dark regime (195 $\mu E \cdot m^{-2} \cdot sec^{-1}$).

For each diallel cross, a randomized complete block design with three replications was used. Replications were carried out over time with new inoculum prepared each time.

Field studies. Plants from the three diallel sets were evaluated for LL at the Plant Pathology Research Farm, Rock Springs, PA. A randomized complete block design with three replications was used for each diallel set. Experimental units were individual rows 7.09 m in length planted on 0.76-m centers. Seedlings were thinned to approximately 30 plants per row 1 mo after emergence. Plants were inoculated at the eight- to 10-leaf stage with isolate RS419-3 of *C. carbonum* race 3.

Inoculum was prepared by macerating 5-day-old cultures of the fungus that had been grown on 2% potato-dextrose agar and straining the suspension through four layers of cheesecloth. The spore suspension was diluted with distilled water to approximately 5.0×10^3 spores per milliliter. All plants of each row were inoculated at dusk using a hand-held pressurized sprayer. Three weeks after inoculation, ear-leaf samples were collected from 10 plants in the center of each test row and dried in a plant press for 1 wk. The length of the first four lesions, typical of *C. carbonum* race 3 reaction, nearest the tip of each leaf was determined in millimeters.

Statistical analysis of diallel crosses. The procedures described by Mather and Jinks (14) were followed in the analysis of each trait in each set. A number of assumptions are required for valid use of diallel analysis methods and Baker (1) pointed out that these assumptions are seldom met. As a check on deviations from the required assumptions, scaling tests, based on the variation over arrays, in the difference between the parent-offspring covariance and the array variance (Wr-Vr) were computed before the diallel analysis was attempted. If the (Wr-Vr) values were found to be heterogenous over arrays, parents with the lowest and/or highest (Wr-Vr) values were eliminated and the scaling test repeated until a set of parents with homogenous values was identified. The elimination of parents with uncharacteristically high or low (Wr-Vr) values does not insure that the assumptions required for diallel analysis are met, but it does provide a means for including only parents for which there is no evidence of deviations from the required assumptions. We were unable to identify the causes of uncharacteristically high or low (Wr-Vr) values.

Once a set of parents with homogenous (Wr-Vr) values was identified for each diallel set and for each resistance parameter, the diallel analysis was completed. Additive (D) and dominance (H_1) effects, asymmetry of positive and negative effects of genes (H_2), and the covariance of additive and dominance effects (F) were estimated by using procedures described by Mather and Jinks (14). Our procedures and notation follow those procedures so closely that details are not presented here. Supplementary analysis of each diallel cross was carried out by using the Griffing method 1, model I (5).

RESULTS

Mean values for DE, LL, and SC from the growth chamber studies and for LL from the field study are shown in Table 1. The number of observations vary because of the number of diallel sets in which a particular inbred appeared. The correlation ($r = 0.79$) of LL means of these inbreds from growth chamber and field studies was significant at $P = 0.01$.

Growth chamber studies. Reaction to *C. carbonum* race 3 for F_1 progenies, as measured by DE, LL, and SC, was intermediate of the two parent inbreds. The mean values of DE, LL, and SC for F_1 progeny families were generally lower than the averages of the parent lines.

Preliminary scaling tests of the (Wr-Vr) values indicated that the values were not homogenous within a diallel set. Parents W438 and W153R in set A, Wf9 in set B, and Pa32 in set C were identified as parents with uncharacteristic (Wr-Vr) values and were eliminated from subsequent analyses. With these parents eliminated from the

TABLE 1. Mean disease efficiency (DE), lesion length (LL), and sporulation capacity (SC) from a growth chamber study and mean LL from a field study of corn inbreds inoculated with *Cochliobolus carbonum* race 3 and used in three sets of diallel crosses

	Growth chamber				Field	
Inbred	DE[a]	LL[b]	SC[c]	No. of observations	LL[b]	No. of observations
Pa419P	6.4	7.6	237	3	12.4	2
W438	12.1	4.7	336	6	6.2	5
PaB8B	10.9	3.5	264	3	9.3	2
Va26	8.7	5.1	448	3	9.3	2
B37	6.9	7.5	305	3	7.3	2
H95	4.9	3.4	437	6	6.0	5
W153R	14.6	6.6	513	6	3.6	5
Pa33A	17.1	10.5	285	3	27.6	3
W64A	8.3	4.8	225	6	12.1	6
Pa33	13.5	10.6	267	6	22.5	6
MS72	7.9	5.7	365	3	6.4	3
Wf9	15.7	5.3	170	3	5.6	3
Pa32	5.0	4.5	155	6	7.5	6
A495	5.1	4.8	250	3	4.7	3

[a] The number of lesions resulting from a given amount of inoculum and expressed as percent.
[b] The length in millimeters of lesions formed on inoculated plants.
[c] The number of spores per square centimeter of diseased tissue.

analysis, variation in (Wr-Vr) values over arrays was not significant. Furthermore, regression of Wr on Vr gave regression coefficients not significantly different from 1.0 indicating that any nonadditive variation present was in the form of dominance.

Mean squares associated with additives effects (A) were highly significant for each trait in each diallel (Table 2). Mean squares for average heterosis (B_1), line heterosis (B_2), specific heterosis (B_3), maternal (C), and reciprocal (D) effects were significant in some, but not all, of the analyses. Except for lesion length in set B, the mean squares for nonadditive effects were much smaller than those for additive effects. The frequency of significant mean squares for nonadditive effects was greater for lesion length and sporulation capacity than for disease efficiency.

Estimates of the genetic variances (14) indicated that the additive variance (D) and the environmental variance (E) were the only components that exceeded twice their standard errors (Table 3), which was used as a measure of significance. None of the estimates of the nonadditive variances (H_1, H_2, or F) exceeded twice their standard errors, despite the fact that tests of the mean squares from the analysis of variance indicated the presence of small amounts of nonadditive genetic variability. The estimated variances were used in equations for narrow-sense heritability as presented by Mather and Jinks (14) and provided narrow-sense heritability estimates of 0.83, 0.60, and 0.46 for DE, SC, and LL, respectively.

The diallel analysis of Griffing (5) was performed for each diallel set without testing for homogeneity of (Wr-Vr) and with all the parents included to confirm the results obtained from the Mather and Jinks (14) analysis. General combining ability (GCA) was the most important source of variation for each of the traits in each diallel (Table 4). Specific combining ability (SCA) was significant in some, but not all, analyses. The significance of SCA formed no detectable pattern with respect to the diallel trait studied. The GCA mean square was three or more times larger than the SCA mean square for each of the analyses.

Field studies. The data from one replication of diallel set A was not used because a preliminary analysis indicated that the values obtained in that replication were much different than the others. That replication was located in a corner of the field where soil compaction and other environmental factors appeared to be different from the rest of the experiment. Pa33A in set B was identified as a parent with uncharacteristic (Wr-Vr) values and was eliminated from subsequent analyses. Variation in (Wr-Vr) values over arrays for each diallel set was not significant after these changes to sets A and B. Furthermore, regression of Wr on Vr resulted in regression coefficients not significantly different from 1.0, indicating that the nonadditive variation present was in the form of dominance.

The largest source of variation was due to the additive effect of genes (A) in each of the diallel crosses (Table 2). Average heterosis (B_1) was smaller than the additive effects but was also significant when tested against the error mean square. Line heterosis (B_2) was significant in sets B and C but not in set A. Specific heterosis effects were significant only in set C. Maternal (C) and reciprocal (D) effects were not significant.

The only genetic component that exceeded twice its standard error, and was considered signficant, was that due to the additive effect of genes (D) (Table 3). Other genetic components were not significant.

The Griffing (5) analysis supported the Mather and Jinks (14) analysis in that both indicated that additive effects of genes (general combining ability (GCA) in the Griffing analysis) was the most important source of genetic variation (Table 4). The specific

TABLE 2. Genetic component mean squares for reaction to *Cochliobolus carbonum* race 3 measured as disease efficiency (DE), lesion length (LL), and sporulation capacity (SC) in three sets of diallel crosses of corn inbreds from a growth chamber study and LL (only) from the field

	Genetic component mean squares					
	Set A[b]		Set B[b]		Set C[b]	
Components[a]	df	MS	df	MS	df	MS
Growth chamber						
DE						
A	4	54.2**	4	238.9**	4	125.2**
B_1	1	0.1	1	0.4	1	19.7**
B_2	4	0.6	4	1.3	4	1.1
B_3	5	0.7	5	4.3**	5	4.3**
C	4	0.5	4	0.7	4	1.2
D	6	0.4	6	0.6	6	1.6
E	48	0.3	48	0.8	48	0.7
LL						
A	6	24.8**	4	16.0**	5	45.0**
B_1	1	9.0**	1	12.9**	1	6.4**
B_2	6	3.2**	4	5.5**	5	0.8**
B_3	14	1.5**	5	0.3	9	7.5**
C	6	0.6**	4	0.1	5	0.2
D	15	1.1**	6	0.4**	10	0.2
E	96	0.2	48	0.1	70	0.1
SC						
A	6	113,380**	5	32,698**	4	69,616**
B_1	1	26,388**	1	492	1	4,651**
B_2	6	4,270**	5	1,078	4	10,280**
B_3	14	6,281**	9	709	5	5,680**
C	6	1,268	5	833	4	2,415**
D	15	2,794	10	1,176	6	739
E	96	1,495	70	911	48	1,045
Field						
LL						
A	6	46.7**	5	151.2**	5	196.3**
B_1	1	3.9**	1	92.8**	1	26.4**
B_2	4	1.7	5	69.4**	5	24.8**
B_3	14	2.5	9	4.1	9	7.8
C	6	1.7	5	4.9	5	0.9
D	15	1.0	10	5.8	10	1.7
E	48	0.8	70	2.5	70	2.6

[a] A = additive effects, B_1 = average heterosis effects, B_2 = line heterosis effects, B_3 = specific heterosis effects, C = maternal effects, D = reciprocal effects, and E = environmental effects (14).
[b] ** Denotes statistical significance at $P = 0.01$.

TABLE 3. Estimates of genetic variance components and the standard error (SE) for reaction to *Cochliobolus carbonum* race 3 measured as disease efficiency (DE), lesion length (LL), and sporulation capacity (SC) in three sets of diallel crosses of corn inbreds from a growth chamber study and LL (only) from the field

	Genetic variance estimates ± SE		
Components[a]	Set A	Set B	Set C
Growth chamber			
DE			
D	6.0 ± 2.6	31.3 ± 8.5	18.5 ± 7.1
H_1	0.2 ± 9.9	1.2 ± 47.3	1.8 ± 24.7
H_2	1.7 ± 6.4	4.7 ± 27.4	5.4 ± 14.3
F	−1.3 ± 10.8	−0.8 ± 52.4	1.0 ± 29.5
E	0.3 ± 0.1	0.8 ± 0.1	0.7 ± 0.1
LL			
D	3.0 ± 1.1	6.2 ± 3.6	5.7 ± 2.1
H_1	2.1 ± 3.6	3.4 ± 6.1	3.5 ± 7.9
H_2	1.6 ± 2.4	2.8 ± 3.0	4.3 ± 5.3
F	1.3 ± 4.0	5.1 ± 8.4	0.8 ± 0.4
E	0.2 ± 0.1	0.1 ± 0.1	0.1 ± 0.1
SC			
D	13,070 ± 6,086	4,604 ± 2,264	14,832 ± 9,174
H_1	2,615 ± 17,103	968 ± 28,363	5,626 ± 19,600
H_2	9,721 ± 19,967	2,896 ± 28,601	9,684 ± 22,522
F	1,538 ± 27,801	1,466 ± 29,242	6,123 ± 15,228
E	1,495 ± 214	1,495 ± 108	1,054 ± 211
Field			
LL			
D	5.5 ± 2.0	59.7 ± 33.2	42.6 ± 20.2
H_1	−2.5 ± 3.8	32.1 ± 52.9	11.9 ± 97.1
H_2	5.9 ± 6.0	29.5 ± 23.9	20.5 ± 99.8
F	−5.2 ± 6.0	56.0 ± 74.9	23.0 ± 115.9
E	0.8 ± 0.2	2.5 ± 0.2	2.6 ± 0.4

[a] D = additive effects, H_1 = dominance effects, H_2 = asymmetry of positive and negative effects of genes, F = covariance of additive and dominance effects, and E = environmental effects (14).

combining ability mean square (SCA) was significant in set B, but small compared to the GCA mean square.

Narrow-sense heritability estimates as described by Mather and Jinks (14) for the three diallel crosses were 0.39, 0.24, and 0.48, respectively. These heritability estimates were calculated from the means of each entry without regard to the variation present within each entry. However, when within plot variances were considered, narrow-sense heritability estimates for sets A, B, and C were 0.29, 0.23, and 0.32, respectively.

Correlations between the reactions of all inbreds and single crosses in the growth chamber and the same genotypes in the field were 0.22, 0.66, and 0.68 for diallel sets A, B, and C, respectively. An examination of a graph of field data versus growth chamber data revealed that the correlation values for sets B and C were high primarily due to the effects of inbreds Pa33 and Pa33A, which had large lesion lengths in both the field and greenhouse.

DISCUSSION

Analyses of the three resistance parameters revealed highly significant differences among the entries, which indicated the presence of genetic variability. The diallel analysis, as outlined by Mather and Jinks (14), suggested the use of Wr and Vr relationships to test the basic assumptions of the diallel analysis. The initial analyses of variance of (Wr-Vr) values revealed significant array mean squares for one or more parameters in all three diallel crosses in both the growth chamber and field experiments. This suggested at least a partial failure of the assumptions for the diallel analyses. Our approach to achieve homogenous parental arrays was to eliminate from subsequent analyses crosses involving parent line(s) with uncharacteristic (Wr-Vr) values.

The estimates of genetic variance components were considered significant only when their values were greater than twice the standard error of the estimates. On this basis, the additive component of variation was the most important component involved in the inheritance of DE, LL, and SC in these studies.

The correlations and plots between growth chamber data and field data suggest that except for a few inbred lines, the overall relationship between LL estimates in both situations is not strong. However, this does not imply that the technique may not be useful. Screening breeding lines in the growth chamber by using LL could eliminate the most susceptible lines. Lines intermediate in response to *C. carbonum* race 3 would be difficult to identify. The danger is that some of the eliminated lines would have been acceptable under field conditions. However, if breeding populations are large enough, resistant lines should be identifiable in the field.

The estimates of genetic variance components obtained from these studies involved a group of selected parents and their offsprings. Therefore, interpretations must be limited to the particular materials tested and should not be considered representative of all corn inbreds. The fact that only additive genetic effects were found in a selected set of parents suggests that the occurrence of corn lines that show strong nonadditive resistance effects would probably be rare.

Fulfilling all assumptions required for diallel analysis is difficult (1) and we cannot claim that all assumptions were met in this study. Biases that result from failure to meet assumptions usually inflate the importance of nonadditive genetic parameters; however, we found no important nonadditive genetic variation. Failure to fulfill the required assumptions may have caused biases in our estimates, but the most important conclusion—inheritance of resistance to *C. carbonum* race 3 is determined by genes with additive effects—would not change. This conclusion was the same in the Mather and Jinks (14) analysis, in which parents with noncharacteristic (Wr-Vr) values were eliminated from the analyses, and with Griffing's (5) analysis in which all parents were included.

The DE, LL, and SC estimates in these studies were obtained by inoculating spore suspension of only one isolate of *C. carbonum* race 3 on corn seedlings. Since differences in virulence have been shown to exist among *C. carbonum* race 3 isolates (7) and host genotype by pathogen genotype interactions also exist (6), corn breeders should consider reaction to several isolates when breeding for resistance.

The use of three resistance parameters in this study has some limitations. While DE and SC measurements could be feasible in growth chamber studies, their applications in practical plant breeding may be difficult, if not impossible. Although the measurement of LL was time consuming, this measurement should provide a more accurate evaluation of quantitative differences than could be obtained by arbitrary classification of genotypes into subjective categories or by using a numerical rating scale (2). The predominance of additive gene action for resistance, indicates that mass, family, or progeny test selection would be effective in a breeding program for resistance to this disease.

TABLE 4. Analysis of variance using the Griffing method 1, model I (5) of reaction to *Cochliobolus carbonum* race 3 measured as disease efficiency (DE), lesion length (LL), and sporulation capacity (SC) in three sets of diallel crosses of corn inbreds from a growth chamber study and LL (only) from the field

	Set A		Set B		Set C	
Source[a]	df	MS[b]	df	MS[b]	df	MS[b]
Growth chamber						
DE						
GCA	6	48.0**	6	117.8**	5	57.5**
SCA	21	17.1	21	24.2	15	4.0
Reciprocal	21	1.2	21	0.8	15	1.1
Error	96	14.6	96	14.2	70	6.2
GCA:SCA ratios		3:1		5:1		14:1
LL						
GCA	6	14.6**	6	18.8**	5	64.9**
SCA	21	2.9**	21	2.6	15	5.4**
Reciprocal	21	0.6	21	0.2	15	0.9
Error	96	0.3	96	3.6	70	1.3
GCA:SCA ratios		5:1		7:1		12:1
SC						
GCA	6	73,469**	6	7,038**	5	41,432**
SCA	21	3,077	21	984	15	13,866**
Reciprocal	21	1,469	21	632	15	1,045
Error	96	1,521	96	1,611	70	2,794
GCA:SCA ratios		24:1		7:1		3:1
Field						
LL						
GCA	6	27.8**	6	60.3**	5	64.9**
SCA	21	2.6	21	20.5**	15	4.9
Reciprocal	21	1.3	21	5.5	15	0.9
Error	96	7.2	96	6.5	70	3.2
Plants in plots	882	0.6	972	0.7	972	0.6
Leaves in plots	1,764	0.4	2,916	0.4	2,916	0.3
GCA:SCA ratios		11:1		3:1		13:1

[a] GCA = general combining ability, SCA = specific combining ability.
[b] ** Denotes statistical significance at $P = 0.01$.

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