Inheritance of Resistance to Anthracnose Leaf Blight in Five Inbred Lines of Corn

M. L. Carson and A. L. Hooker

Former graduate research assistant and professor, respectively, Department of Plant Pathology, University of Illinois, Urbana 61801. Present title and address of senior author: assistant professor, Department of Plant Science, South Dakota State University, Brookings 57007.

Portion of an M.S. thesis submitted by the senior author to the Graduate College, University of Illinois, Urbana.
Accepted for publication 23 September 1980.

ABSTRACT

Carson, M. L., and Hooker, A. L. 1981. Inheritance of resistance to anthracnose leaf blight in five inbred lines of corn. Phytopathology 71:488-491.

Parent inbred lines, F_1, F_2, and backcross generations from five corn crosses were evaluated in the field for reaction to anthracnose leaf blight (ALB) which is caused by *Colletotrichum graminicola*. The crosses involved the susceptible inbred Oh07B crossed with resistant inbred lines H55, H84, Pa91, Pa762, and Syn B High-3. Individual plants were rated for reaction on the basis of percent leaf tissue infected. Generation mean analysis indicated that additive genetic effects were of primary importance in four of the five families. Resistance appeared to be controlled by a few (two to four) dominant genes. Mass selection, pedigree, and backcross breeding methods should all be effective means of breeding for resistance to ALB.

Additional key words: Zea mays, maize.

Anthracnose leaf blight (ALB) of corn is caused by the fungus *Colletotrichum graminicola* (Ces.) Wils. and it occurs in warm, humid corn-growing areas throughout the world. In the United States, ALB is prevalent in the Southeast and in the more humid warmer areas of the Corn Belt (7,14). Formerly ALB was of minor importance to corn production in the USA, but increasing prevalence and evidence of yield losses has drawn the attention of corn breeders and seed producers. Yield losses up to 19 and 28% in corn hybrids and inbreds, respectively, have been demonstrated in comparisons of inoculated and uninoculated field plots (12).

Since genetic resistance currently is the only economical method for controlling fungal leaf blights of corn, genetic information on ALB resistance should be valuable in the development of breeding procedures to increase resistance in inbred lines and hybrids.

The purpose of this study was to examine the mode of inheritance of resistance to ALB in five inbred lines of corn and to determine the types and magnitudes of gene action involved.

MATERIALS AND METHODS

Six corn inbred lines were chosen on the basis of preliminary evaluations of ALB reaction. Five inbred lines (H55, H84, Pa91, Pa762, and Syn B High-3) were chosen as resistant parents, and the sixth inbred, Oh07B, was chosen as the susceptible parent. The five resistant parents were crossed with Oh07B, and subsequent generations were produced. Genetic analyses were based on P_1 (the resistant parent), P_2 (Oh07B); F_1, F_2; BCP_1 and BCP_2 generations. The F_2 generation was produced by selfing F_1 plants. The BCP_1 and BCP_2 generations (backcrosses) were produced by crossing F_1 plants with the resistant parent and Oh07B, respectively. Since all five families had the inbred Oh07B in common, they are designated by the name of the resistant parent.

The six generations (two parents and four sets of progenies) of the H55, H84, Pa91, and Syn B High-3 families were grown on the Agronomy South Farm at Urbana in the summers of 1977 and 1978. The Pa762 population was grown in 1977 only. Each experiment consisted of a randomized complete block with three replications. There were 12 plants in each plot and each replication included one plot each for the P_1, P_2, and F_1 generations, three plots for the BCP_1 and BCP_2 generations, and six plots for the F_2 generation.

Individual plants were inoculated and rated, and the data were analyzed statistically. Plants were inoculated by placing oat grain cultures of *C. graminicola* into the leaf whorls of young plants on three dates in 1977 and on two dates in 1978. Disease ratings were taken on individual plants approximately 2 wk after midsilk. Plants were placed in disease severity classes based upon visual estimates of percent leaf tissue infected. The data were arc sine-transformed before analysis, which is suggested for variables expressed as percentages (13).

Analyses of the transformed data were performed by fitting the following genetic model of Mather and Jinks (10) to the generation means: $\bar{Y} = m + a_1d + a_2h$, in which $\bar{Y}$ 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, and a_1 and a_2 are the relative contributions of these effects to each generation mean. Tests of significance of the genetic effects and deviations from the model were made by using F tests in the analysis of variance of generation means. A pooled error term (generation × blocks within years) was used to test the significance of the generation × year mean square in the analyses of populations studied during both years. The generations × year mean square, when significant, was used to test genetic effects over years. Broad-sense heritability

TABLE 1. Mean anthracnose leaf blight disease severities (percent leaf tissue infected) of six generations and deviations of the F_1 mean from the midparent value[a] in each of five corn families studied in 1977 and 1978

		Family				
Generation	Year	H55	H84	Pa91	Pa762[b]	Syn B High-3
P_1	1977	1.0	1.0	2.4	9.7	1.7
	1978	3.0	1.0	2.3	···	5.2
Oh07B	1977	35.5	31.1	37.4	42.7	35.3
	1978	40.0	40.0	31.7	···	37.0
F_1	1977	35.1	1.0	1.0	45.1	10.2
	1978	3.0	1.0	1.7	···	3.0
F_2	1977	40.5	11.0	20.4	64.2	34.6
	1978	29.9	7.8	9.9	···	11.0
BCP_1	1977	3.3	1.2	1.6	20.4	1.0
	1978	4.6	1.3	3.6	···	2.8
BCP_2	1977	17.6	6.0	6.8	12.2	5.9
	1978	22.3	4.9	8.3	···	9.3
$\bar{F}_1$-MP[a]	1977	16.8**[c]	−15.0**	−18.9**	18.9**	− 8.3*
	1978	−18.5**	−19.5**	−15.3**	···	−18.1**

[a] Midparent value = $\overline{MP} = (P_1 + Oh07B)/2$.
[b] This family was studied in 1977 only.
[c] Asterisks (*,**) indicate difference is statistically significant $P = 0.05$ and $P = 0.01$, respectively.

0031-949X/81/05048804/$03.00/0

estimates in F_2 generations were calculated by the formula $(\sigma^2_p - \sigma^2_e)/\sigma^2_p$, in which σ^2_p is the phenotypic variance of individuals in the F_2 generation and σ^2_e is the environmental variance between individuals of the same genotype, estimated by pooling the within-plot sums of squares and degrees of freedom of the inbred parents and the F_1 generation. Estimates of the minimum number of effective factors (K_1) were calculated according to the Castle-Wright formula (1,15) as modified by Mather and Jinks (10) in which K_1 = (phenotypic range in the $F_2)^2/8(\sigma^2_p - \sigma^2_e)$.

RESULTS

Inspection of generation means for the six families (Table 1) reveals that the susceptible inbred had from 29.0 to 39.0% more infected tissue than did the resistant parents. The F_1 mean percentage of tissue infected was significantly less than the mid-parent value in the H83, Pa91, and Syn B High-3 families in 1977, and in the H55, H84, Pa91, and Syn B High-3 families in 1978. Midparent values are the average disease severity ratings of the two parents in a cross and significant deviations of F_1 means from them are indicative of genic dominance. In 1977, however, the mean percentage of infected tissue for the F_1 generations in the H55 and Pa762 families was significantly higher than the midparent values. In all families in both years, the F_2 mean percent tissue infected exceeded that of the F_1. The F_2 mean in the H55, Pa762, and Syn B High-3 families in 1977 approached or exceeded the mean disease severity of Oh07B. In all families in both years, the average percentage of infected tissue for backcross generations was lower than that of the F_2 generations, and in all but one family, the mean percentage of tissue infected in the BCP_1 was lower than that of the BCP_2. In the Pa762 family, the mean percentage of infected tissue for the BCP_1 was greater than that for the BCP_2.

The F_2 frequency distributions of plant ratings of all families indicate transgressive segregation for susceptibility (Table 2). In the H84, Pa91, and Syn B High-3 families, the BCP_2 contained no

TABLE 2. Frequency distributions of individual plant anthracnose leaf blight disease reactions (percent leaf tissue infected scale) in each of five corn families evaluated after artificial inoculation in 1977 and 1978

Family	Generation	Year	Percent of plants in disease class (% tissue infected)										
			1	3	5	10	20	40	60	80	90	95	100
H55	F_2	1977	29.0	4.5	5.5	6.5	3.7	9.2	12.0	9.7	3.2	2.8	14.3
		1978	11.9	7.8	10.5	18.3	19.2	10.5	5.0	3.7	0.5	···	12.8
	BCP_1	1977	87.6	2.9	5.7	···[a]	···	1.0	2.9	···	···	···	···
		1978	37.5	27.9	17.3	13.5	1.9	1.9	···	···	···	···	···
	BCP_2	1977	31.9	10.3	17.5	11.3	6.2	8.2	8.2	3.1	1.0	···	2.1
		1978	8.3	12.0	18.5	22.2	14.8	7.4	9.3	0.9	0.9	···	5.6
H84	F_2	1977	59.8	14.1	6.4	5.0	2.3	3.6	1.8	1.8	2.7	0.5	1.8
		1978	34.2	26.0	15.1	12.3	7.3	1.8	1.4	0.9	···	0.9	···
	BCP_1	1977	90.4	9.6									
		1978	83.8	16.2									
	BCP_2	1977	40.4	31.2	12.8	9.2	1.8	1.8	1.8	···	···	···	0.9
		1978	26.9	35.2	18.5	15.7	1.9	1.9	···	···	···	···	···
Pa91	F_2	1977	46.8	5.8	6.3	8.8	7.3	9.8	1.9	3.4	3.4	1.5	4.9
		1978	20.9	22.3	16.4	15.5	15.0	2.7	0.9	0.9	···	···	···
	BCP_1	1977	76.3	18.4	4.4	0.9	···	···	···	···	···	···	···
		1978	36.7	33.9	13.8	15.6	···	···	···	···	···	···	···
	BCP_2	1977	54.6	15.7	12.0	4.6	10.2	0.9	1.8	···	···	···	···
		1978	14.8	17.6	26.8	25.9	11.1	3.7	···	···	···	···	···
Pa762	F_2	1977	7.1	4.9	5.8	3.1	5.8	5.8	7.1	15.2	12.5	7.1	25.4
	BCP_1	1977	13.1	12.1	20.2	19.2	10.1	12.1	5.1	3.0	4.0	···	1.0
	BCP_2	1977	36.1	16.5	15.5	12.4	6.2	5.1	2.1	1.0	2.1	···	2.1
Syn B High-3	F_2	1977	40.3	3.7	2.8	1.8	6.9	6.9	11.6	13.0	4.6	4.2	4.2
		1978	38.4	25.5	14.8	13.9	3.2	1.9	1.4	···	···	···	0.5
	BCP_1	1977	100.0	···	···	···	···	···	···	···	···	···	···
		1978	48.2	33.6	10.9	7.3	···	···	···	···	···	···	···
	BCP_2	1977	52.3	16.2	0.9	19.8	7.2	3.6	···	···	···	···	···
		1978	10.8	28.8	25.2	19.8	9.9	1.8	2.7	···	1.0	···	···

[a]The symbol ··· indicates that no plants fell into that disease severity class.

TABLE 3. Analyses of variance of mean anthracnose leaf blight disease severities (arc sine-transformed percent leaf tissue infected) of six generations in each of five corn families studied in 1977 and 1978

Source	Family									
	H55		H84		Pa91		Pa762[a]		Syn B High-3	
	d.f.[b]	m.s.[c]	d.f.	m.s.	d.f.	m.s.	d.f.	m.s.	d.f.	m.s.
Block (years)	4	43.7	4	0.6	4	11.5	2	0.0	4	10.1
Years	1	57.4	1	5.1	1	1.1	···	···	1	51.8
Generations	5		5		5		5		5	
Additive	1	3,190.3**[d]	1	2,269.1**	1	2,097.3**	1	471.7**	1	2,050.7**
Dominance	1	0.6	1	1,130.3**	1	1,000.7**	1	286.4**	1	671.7
Deviations	3	463.1	3	136.3**	3	160.6*	3	1,072.7**	3	224.1
Generations × years.	5	206.9**	5	9.0**	5	22.6*	···	···	5	138.0**
Error	20	28.4	20	1.11	20	7.0	10	15.3	19[e]	12.6

[a]This family was studied in 1977 only.
[b]d.f. = degrees of freedom.
[c]m.s. = mean squares.
[d]Asterisks (*,**) indicate significance at $P = 0.05$ and $P = 0.01$, respectively.
[e]Estimated with 19 d.f. since one plot was lost in 1977.

segregates that exceeded Oh07B in percent tissue infected. The distributions of the BCP_1 generations in the H55, H84, Pa91, and Syn B High-3 families were highly skewed with the majority of progeny showing a lower percentage of infection (Table 2).

The analysis of variance of generation means (Table 3) shows that significant additive genetic effects were detected in all families. Additive genetic effects accounted for 58.6–69.7% of the total variation among generation means in the four families studied for 2 yr (Table 4). Dominance genetic effects were significant in families H84, Pa91, and Pa762. Significant deviations from the additive dominance genetic model were detected in the H84, Pa91, and Pa762 families. In all families evaluated for 2 yr, there was a significant generation × year interaction.

It was possible to estimate heritabilities and numbers of effective factors (genes) involved in the inheritance of ALB resistance in the families. Estimates of broad-sense heritability in all families were high and ranged from 85.4 to 96.2% (Table 5). Estimates of the minimum nmber of effective factors involved ranged from 1.19 to 3.80 (Table 5).

DISCUSSION

The detection of significant additive, dominance, and deviation effects indicate that all may be important in resistance to ALB. Additive genetic effects were of primary importance in four of the five families studied. Genic dominance may be of greater importance than indicated by generations mean analysis. Lim and White (9) found highly significant negative heterosis effects for adult plant ALB severity (%) in all resistant × susceptible crosses studied in a diallel analysis, indicating a predominance of dominant genes for resistance. Significant deviations of the F_1 means from midparent values toward lower percentages of tissue infected were found in all four families studied in 1978 and in three of five families in 1977 (Table 1). Further evidence lies in the distributions and variances of the backcross generations (Table 2). The marked skewness of the BCP_1 distributions and the small variances of the BCP_1s compared to those of the BCP_2s are indicative of genic dominance (10). Although significant deviations from the additive dominance genetic model were detected in three of the families studied, they were of major importance in only the Pa762 family in which they accounted for 80.9% of the total variation among generation means (Table 4). Deviations could have been due to the lack of an adequate scale to measure ALB resistance, nonallelic interactions of genes, or the failure to meet all the assumptions of generation mean analysis (10). Analyses based upon actual data for percent tissue infected and upon logarithmic and square root-transformed data all showed these significant deviations. Conceivably, many of the deviation effects can be accounted for by the failure of genetic materials used to meet an important assumption of generation mean analysis.

Environment can have an important role in ALB development (2,8). Significant generation × year effects in the analysis of variance (Table 3) can be interpreted as genotype × environment interactions. These effects were of a much higher magnitude in the H55 and Syn B High-3 populations than in the remaining two populations. Much of the interaction was probably due to the failure of the F_1 means to be the same in both 1977 and 1978 (Table 1). In 1977, the F_1 mean of the H55 population approached that of Oh07B, while in 1978 it showed a complete reversal of dominance, equalling H55 in percent tissue infected. Similarly, the F_1 mean of the Syn B High-3 population in 1977 was intermediate in percent tissue infected, while in 1978 it had a lower mean percent tissue infected than did Syn B High-3. In 1977, growing conditions early in the season favored the development of anthracnose stalk rot (ASR) in these families, both of which appeared susceptible. The apparent susceptible ALB reactions of the F_1s in 1977 could have been due to ASR causing a "firing" or wilting of the lower leaves, resulting in their unusually high ALB disease severity ratings. Lim and White (9) encountered similar difficulties in assessing ALB severity on adult plants of certain genotypes in the field due to early "senescence" of lower leaves. Due to the high incidence of ASR in all of the populations, ALB ratings taken at later dates in the growing season in both 1977 and 1978 were considered unreliable and were disregarded.

Generation mean analysis has been used to detect types of gene action involved in several quantitatively inherited traits including disease resistance (3,5,11). Major drawbacks associated with generation mean analyses lie in the assumptions made in using the technique. Apart from the usual assumptions concerning the analysis of variance, additional assumptions include the isodirectional distribution of genes between the two parental lines, the absence of linkage between interacting loci, and the absence of selection favoring certain gametes (10). Transgressive segregation in the F_2 generation of all families indicates that the first assumption probably was not met and is evidence that the inbred line Oh07B has at least one factor for resistance to ALB (Table 2). Further, the lack of segregates more susceptible than Oh07B in the BCP_2 generations in three of the families indicates that this factor is dominant or partially dominant. The lack of genes being isodirectionally distributed among the parents would result in significant deviations from the simple additive dominance genetic model and furthermore would seriously bias any attempt to partition the genetic variances of the segregating generations into additive or dominance variance components. Because of this difficulty, only broad-sense heritabilities were estimated and the Castle-Wright formula was modified to take this lack of isodirectional distribution of genes into account (10). A further limitation is the lack of sensitivity of generation mean analysis to detect types of gene action when individual gene effects of differing direction cancel each other and are not detected by means (3,5,10).

Unweighted least squares regression techniques were used to fit the data to the genetic model. Mather (10) suggested that weighted least squares would be more appropriate if it is evident that assumptions about a common variance of generation means are not met. Moll et al (11) found that no information was gained by using weighted regression and suggested that weighting might actually add extraneous variability to the data set. For this reason, plus the extra effort involved in weighted regression analysis, unweighted least squares were considered adequate.

Selection for resistance to ALB should be effective since heritabilities were high, additive gene effects were of major

TABLE 4. Percent of variation in anthracnose leaf blight reaction among generation means (arc sine-transformed percent leaf tissue infected) accounted for by additive and dominance genetic effects and deviations from additive dominance model. Data are from the combined analyses of variance over 2 yr

	Family				
Effect	H55	H84	Pa91	Pa762[a]	Syn B High-3
Additive	69.7	63.4	58.6	11.9	60.4
Dominance	0.0	26.9	27.9	7.2	19.8
Deviations	30.3	9.7	13.5	80.9	19.8

[a]This family was studied in 1977 only.

TABLE 5. Estimates of broad-sense heritabilities (h^2) and numbers of effective factors (K_1) involved in resistance to anthracnose leaf blight (ALB) in five corn families

Family	Broad-sense heritability (h^2)[a]	Effective factors (K_1)[b]
H55	85.4	1.25
H84	96.2	3.80
Pa91	91.7	2.58
Pa762[c]	85.4	1.19
Syn B High-3	86.0	1.90

[a]Broad-sense heritabilities (h^2) estimated by the formula:

$$h^2 = \left[(\sigma^2_{F_2} - \sigma^2_e)/\sigma^2_{F_2} \right] \times 100$$

Estimates were based on ALB percent leaf tissue infected data (arc sine-transformed and pooled over 2 yr.

[b]Numbers of effective factors (K_1) estimated by:

$$K_1 = \frac{(\text{phenotypic range in } F_2)^2}{8(\sigma^2_{F_2} - \sigma^2_e)}$$

[c]This family was studied in 1977 only.

importance in most populations, and relatively few genes appear to be involved. The data indicate that probably only two to four dominant genes were involved in the populations studied and that interallelic interactions (epistasis) were probably of minor importance when effects of nonisodirectional distribution of genes are taken into account. Phenotypic mass selection for ALB resistance should give rapid progress in the improvement of corn populations. Further, pedigree selection and backcross breeding should be efficient ways to develop ALB-resistant inbred lines. Evidence of transgressive segregation in F_2 populations would further indicate that certain intermediate $\times$ intermediate crosses could produce progenies with higher levels of resistance than either parent. Resistance to ALB is common in a wide array of corn inbreds and germ plasm (4), and this study does not preclude the possibility that major genes for ALB resistance might be found, which has been the case with resistance to ALB in sorghum (6). Inferences made here apply to the plant populations studied and not necessarily to other corn populations.

LITERATURE CITED

1. Castle, W. E. 1921. An improved method of estimating the number of genetic factors concerned in cases of blending inheritance. Science 54:223.
2. Hammerschmidt, R., and Nicholson, R. L. 1977. Resistance of maize to anthracnose: Effect of light intensity of lesion development. Phytopathology 67:247-250.
3. Hughes, G. R., and Hooker, A. L. 1971. Gene action conditioning resistance to northern corn leaf blight in corn. Crop Sci. 11:288-290.
4. Humy, C. 1976. Reactions of *Zea mays* to *Colletotrichum graminicola.* M.Sc. thesis, University of Illinois, Urbana. 71 pp.
5. Kappelman, A. J., Jr., and Thompson, D. L. 1966. Inheritance of resistance to Diplodia stalk rot in corn. Crop. Sci. 6:288-290.
6. LeBeau, F. J., and Coleman, O. H. 1950. The inheritance of resistance in sorghum to leaf anthracnose. Agron. J. 42:33-34.
7. Leonard, K. J. 1974. Foliar pathogens of corn in North Carolina. Plant Dis. Rep. 8:718-820.
8. Leonard, K. J., and Thompson, D. L. 1976. Effects of temperature and host maturity on lesion development of *Colletotrichum graminicola* on corn. Phytopathology 66:635-639.
9. Lim, S. M., and White, D. G. 1978. Estimates of heterosis and combining ability for resistance of maize to *Colletotrichum graminicola.* Phytopatholgy 68:1336-1342.
10. Mather, K., and Jinks, J. L. 1971. Biometrical Genetics. Cornell University Press, Ithaca, NY. 382 pp.
11. Moll, R. H., Thompson, D. L., and Harvey, P. H. 1963. A quantitative genetic study of the inheritance of resistance to brown spot (*Physoderma maydis*) of corn. Crop. Sci. 3:389-391.
12. Smith, D. R. 1976. Yield reduction in dent corn caused by *Colletotrichum graminicola.* Plant Dis. Rep. 60:967-970.
13. Steel, R. G. D., and Torrie, J. H. 1960. Principles and procedures of statistics. McGraw-Hill, New York. 481 pp.
14. Wheeler, H., Politis, D. J., and Poneleit, C. G. 1974. Pathogenicity, host range, and distribution of *Colletotrichum graminicola* on corn. Phytopathology 64:293-296.
15. Wright, S. 1921. Systems of mating. I. The biometric relations between parent and offspring. Genetics 6:111-178.