Inheritance of Resistance to Anthracnose Stalk Rot of Corn

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ABSTRACT

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Inheritance of resistance to anthracnose stalk rot of corn (*Zea mays*), caused by *Colletotrichum graminicola*, was studied in progeny derived from a cross between the resistant inbred DW1035 [(MP305 × FRB73$^{[S]}$)$_{S8}$] and the susceptible inbred FRB73. In 1987, 1988, and 1989, the parental lines and the F_1, F_2, and both backcross generations were tested. Generation mean analysis indicated significant additive and dominance genetic effects were of primary importance in all 3 yr. Dominance estimates ranged from 8.3 to 33.9%. Estimates of the number of effective factors conditioning resistance ranged from 0.38 to 2.08. Significant dominance genetic effects from generation mean analysis, low estimates of the number of effective factors, and frequency distributions of individual plant reactions indicate genetic dominance controlled by one or a few genes. To further evaluate the inheritance, individual stalk rot reactions within a particular generation were classified as resistant or susceptible by discriminant analysis. Expected stalk rot reaction distributions were then determined by means of a partitioning method. The observed distributions were compared for goodness of fit to the expected distributions using a chi-square test. The data could best be explained by a single, dominant gene difference between DW1035 and FRB73 for anthracnose stalk rot resistance in some generations. The use of discriminant analysis to identify resistant and susceptible plants, followed by partitioning, is a method that can be extremely useful with data where genetically resistant and susceptible plants may not be correctly identified by the phenotypic reaction.

Anthracnose stalk rot, caused by the fungus *Colletotrichum graminicola* (Ces.) G.W. Wils., is a major economic stalk rot disease of corn (*Zea mays* L.) in the United States, (9,13,18,23, 27,33,34) as well as in other parts of the world (22,25). From the early 1970s, corn anthracnose has become more prevalent. It is especially serious in the southeastern United States and extending north and west into Illinois (33). In 1972, some fields of sweet corn in Indiana were lost totally because of damage caused by *C. graminicola* (31). Severe outbreaks of anthracnose devastated cornfields in North Carolina in 1972 and 1973 (18). In 1975, *C. graminicola* was found in 78% of Illinois fields with rotting stalks and prematurely dying plants (13). In a field survey done in 1982 and 1983 in Illinois, *C. graminicola* was found in 73 and 89% of the fields, respectively, and was the most prevalent stalk rot fungus found in rotted stalks (1). Also, the pathogen was found frequently in northern Illinois, where it had not been previously reported (1).

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The increase in incidence and severity of anthracnose has prompted research on all facets of the disease, including the evaluation of various inbreds and hybrids for resistance. Breeding for resistance to *C. graminicola* has been successful with anthracnose leaf blight (17) and anthracnose stalk rot of sorghum (6), suggesting that it may be successful in corn. Several studies have been done on the inheritance of resistance to anthracnose stalk rot of corn. Lim and White (20) found significant heterotic effects in 45 F_1 diallel crosses and their 10 parental inbreds for both leaf blight and stalk rot reactions, suggesting partial dominance for resistance. Both additive and nonadditive genetic effects were important, with more importance attributed to the additive genetic effects.

Carson and Hooker (4) studied the inheritance of resistance in progeny from five sets of crosses involving four resistant and two susceptible inbred lines. 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, with significant dominance genetic effects in some populations. Carson and Hooker (5) continued this work using

reciprocal translocation testcross analyses to locate genes in the resistant inbred A556. The long arms of chromosomes 1, 4, and 8 and both arms of chromosome 6 were found to carry resistance genes.

Carson (3) also studied the inheritance of resistance of the inbred MP305 in crosses with A632. The frequency distributions for the number of internodes discolored by anthracnose stalk rot were similar for MP305, the F_1, and the $F_1 \times$ MP305, with the majority of individual plants having only one internode discolored. However, some individual plants had two or three internodes discolored. The inbred line A632 exhibited more variation, with plants having two to five internodes discolored. Variation within the F_2 was continuous, with the distribution highly skewed to fewer internodes discolored. On the basis of the frequency distribution in the A632 $\times$ MP305 cross, a single-gene hypothesis was tested in the F_2 generation in 1 yr. A single-gene model with complete dominance could not be satisfactorily fitted to the F_2 data by a partitioning method, so a two-gene model was fitted to the data. Results could best be explained by the hypothesis that MP305 has one major and one minor dominant gene for resistance to anthracnose stalk rot not found in A632. However, the results of the study were considered preliminary, as larger population sizes and more advanced generations were needed for further genetic studies of MP305.

Research using F_1, F_2, and backcross generations from the cross of the resistant inbred LB31 and the susceptible inbred B37 indicated that anthracnose stalk rot resistance was conditioned by a single, dominant gene (2). Susceptible or resistant class limits were determined from the stalk rot reactions of the two parents. The F_1 generation and the backcross to the resistant parent, however, did not follow the Mendelian ratios expected for a single, dominant gene model at one of the two locations where the generations were evaluated in a single year.

The identification of resistance to anthracnose stalk rot that is simply inherited would be of immediate use to plant breeders, because the resistance could be backcrossed into established lines. The purpose of this study was to determine the inheritance of resistance to anthracnose stalk rot in the inbred DW1035, using quantitative and qualitative measures of anthracnose stalk rot reactions of progeny derived from crosses of DW1035 with the adapted inbred line FRB73. This study also proposes an analysis procedure to help account for the problem associated with predisposition that can lead to misclassification of individual plants.

MATERIALS AND METHODS

The inbred lines used were DW1035 [$(MP305 \times FRB73^{[5]})_{S8}$] (resistant) and FRB73 (susceptible). The inbred DW1035 was developed from a cross between FRB73 and MP305, which was backcrossed to FRB73 five times, with selection for anthracnose stalk rot resistance in each cycle of backcrossing. Following inoculation, the anthracnose stalk rot reaction of DW1035 was similar to that of MP305. FRB73 (P_1) was crossed with DW1035 (P_2). The cross and derived generations (F_2, BCP_1, and BCP_2) were evaluated in 1987, 1988, and 1989 at the University of Illinois Agronomy/Plant Pathology South Farm, Urbana.

The experiment was planted 5 May 1987, 26 April 1988, and 8 May 1989 in rows 76 cm apart and 8 m long, with 25 kernels per row. Plants were later thinned to spacings of approximately 38 cm. The experiment was arranged as a randomized complete block, with four replications. The single-row plots per replicate were: P_1, P_2, and F_1, four plots each; F_2, 12 plots; and BCP_1 and BCP_2, eight plots each. Actual numbers of plants per plot varied somewhat because of the loss of stand from poor germination and cultivation damage.

Plants were inoculated approximately 1–2 wk after midsilk by injecting 2 ml of a conidia suspension of *C. graminicola* into the first elongated internode above the brace roots with a 50-cm^3 Vaso Pistol Grip Rubber Plunger Syringe (Ideal Instruments Inc., Chicago) fitted with a stainless-steel needle, similar to that described by Koehler (16). Inoculum was prepared by washing conidia from the surface of cultures of *C. graminicola* grown on oatmeal agar, filtering the resulting suspension through a double thickness of cheesecloth, and adjusting the final concentration to 2.0×10^5 conidia per milliliter with deionized water. A single isolate, C1, obtained from sweet corn leaves collected at Hoopston, Illinois, was used. In previous studies it had been the most aggressive isolate over a number of corn lines (32).

Individual plants were evaluated 4 wk after inoculation by cutting off the top of the plant just below the ear and splitting the stalk longitudinally to ground level. The total number of internodes with discoloration and the number of internodes that were >75% discolored were recorded. Frequency distribution graphs of anthracnose stalk rot reactions (total number of internodes discolored and number of internodes >75% discolored) were visually compared for the parental lines and the derived generations (F_1, F_2, BCP_1, and BCP_2) summed over years.

Anthracnose stalk rot data (internodes discolored, internodes >75% discolored, and the sum of internodes discolored plus internodes >75% discolored) from parental inbred lines and F_1, F_2, BCP_1, and BCP_2 generations were analyzed by generation mean analysis, by fitting means to a regression model, $Y = m + a_1 d + a_2 h$ in which Y is the mean of the 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 (21). Narrow-sense heritability (h^2) of anthracnose stalk rot reactions of internodes discolored, internodes >75% discolored, and the sum of internodes discolored plus internodes >75% discolored in the F_2 populations were estimated by: $h^2 = (2V_{F2} - V_{BCP1} - V_{BCP2})/V_{F2}$ (30). The number of effective factors (K) conditioning anthracnose stalk rot reactions of internodes discolored, internodes >75% discolored, and the sum of internodes discolored plus internodes >75% discolored in the F_2 population were estimated according to Mather and Jinks (21) using additive variance estimates of Warner (30): $K = (P_1 - P_2)^2/8(2V_{F2} - V_{BCP1} - V_{BCP2})$.

In addition to examining the data quantitatively by generation mean analysis, a specific genetic hypothesis was tested using qualitative classes (resistant or susceptible) of individual plant anthracnose stalk rot reactions. A prior study (2) examined quantitative data of individual plant anthracnose stalk rot reactions using qualitative classes, resistant or susceptible, based on reactions of the parental lines. We used parametric procedures with SAS discriminant analysis (26) to classify individual plants as having resistant or susceptible reactions to *C. graminicola*. The DISCRIM procedure computes linear or quadratic discriminant functions for classifying observations into two or more groups on the basis of one or more numerical variables. The numerical variables used to classify observations were the total number of internodes with discoloration and the number of internodes >75% discolored. The establishment of the susceptible and resistant classes was based on the discriminant functions for the parental lines. The discriminant functions were determined by a measure of generalized squared distances. The classification criteria of the two parents were then applied to the parental lines and derived generations to establish observed anthracnose stalk rot reaction distribution (resistant:susceptible). An individual plant was placed in the class with which it had the smallest generalized squared distance.

Expected distributions were determined by the partitioning method (19,24), assuming segregation of a single, dominant gene for resistance. On the basis of this hypothesis, the genotype of the resistant parent is AA, that of the susceptible parent is aa, and that of the F_1 is Aa. Expected distributions of the F_2, BCP_1, and BCP_2 generations can be predicted from the frequency distributions of the F_1 (Aa), P_1 (aa), or P_2 (AA) when combined in the correct proportions. This procedure formulates expected distributions that are not pure Mendelian. Using Mendelian expected ratios to test a genetic hypothesis does not allow for misclassified plants. In evaluating anthracnose stalk rot reactions, environmental effects and experimental methodology, rather than plant genetics, can account for misclassification of individual plants. The partitioning method can help account for genetically susceptible plants misclassified as resistant and for genetically

resistant plants misclassified as susceptible.

Observed distributions of F_1 plants were compared with the observed distributions of P_2 plants using contingency tables and the chi-square test for statistical independence (28). In the F_2 generation, 25% of the plants would be expected to be of the AA genotype (predicted from the frequency distribution of the resistant parent, P_2), 50% to be of the Aa genotype (predicted from the frequency distribution of the F_1), and 25% to be of the aa genotype (predicted from the frequency distribution of the susceptible parent, P_1). In the BCP_1 generation, 50% of the plants would be expected to be of the Aa genotype (predicted from the frequency distribution of the F_1) and 50% to be of the aa genotype (predicted from the frequency distribution of the susceptible parent, P_1). In the BCP_2 generation, 50% of the plants would be expected to be of the AA genotype (predicted from the frequency distribution of the resistant parent, P_2) and 50% to be of the Aa genotype (predicted from the frequency distribution of the F_1). These predicted distributions for each postulated genotype were then combined to obtain an overall expected distribution for the F_2, BCP_1, and BCP_2 generations. The observed F_2, BCP_1, and BCP_2 distributions were tested for goodness of fit to the expected distributions using a chi-square test for homogeneity. A correction for continuity, applicable when the criterion has a single degree of freedom, was used to improve the approximation of the chi-square distribution (28).

RESULTS

Anthracnose stalk rot in DW1035 (P_2) was generally restricted to the inoculated internode, with that internode not >75% discolored. Some plants, however, had more than one internode discolored, with one or more internodes >75% discolored (Figs. 1 and 2). The inbred line FRB73 (P_1) varied in its susceptibility to anthracnose stalk rot, with most plants having at least two internodes discolored and with one or more internodes >75% discolored. Some plants, however, had only one internode discolored, with no internodes >75% discolored (Figs. 1 and 2).

The F_1 and BCP_2 frequency distributions for anthracnose stalk rot reactions were generally similar to those of DW1035 (Fig. 1), with each line or population having a high percentage of plants with only one internode discolored and a low percentage of plants with more than one internode discolored. The F_1 and BCP_2 generations also had a high percentage of plants with no or one internode >75% discolored and only a low percentage of plants with more than one internode >75% discolored (Fig. 2). The F_2 generation was continuous, with the distribution highly skewed to fewer internodes discolored (Fig. 1) and to fewer internodes >75% discolored (Fig. 2). These frequency distributions are indicative of genetic dominance.

The analysis of variance of generation means for the number of internodes discolored, the number of internodes >75% discolored, and the sum of the number of internodes discolored plus the number of internodes >75% discolored (Table 1) showed that significant additive and dominance effects were important in each year and in all years combined. Additive genetic effects accounted for 63.7–90.3% of the total variation, while dominance genetic effects accounted for 8.3–33.9% of the total variation (Table 2). Estimates of narrow-sense heritabilities ranged from 9.2 to 52.4% over the 3 yr, and estimates of the minimum number of effective factors involved ranged from 0.38 to 2.08 (Table 3). A single gene model with complete dominance was hypothesized based on the frequency distributions, generation mean analysis, estimates of the number of effective factors (genes), and previous work of Carson (3) with the inbred MP305.

Because a significant replication within year effect was found, the discriminant analysis procedure to determine the observed susceptible and resistant anthracnose stalk rot reactions was done on each replication each year. Expected distributions determined by the partitioning method were also done for each replication. Individual observed and expected distributions for each generation were summed over replications within a year, and a corresponding chi-square value was calculated. Chi-square values evaluating the significance of deviations between observed and expected distributions for a single gene model in the F_1, F_2, and BCP_1 in 1987; the F_2, BCP_1, and BCP_2 in 1988; and the F_1 and F_2 in 1989 support the hypothesis of a single, dominant gene in the FRB73 × DW1035 cross (Table 4).

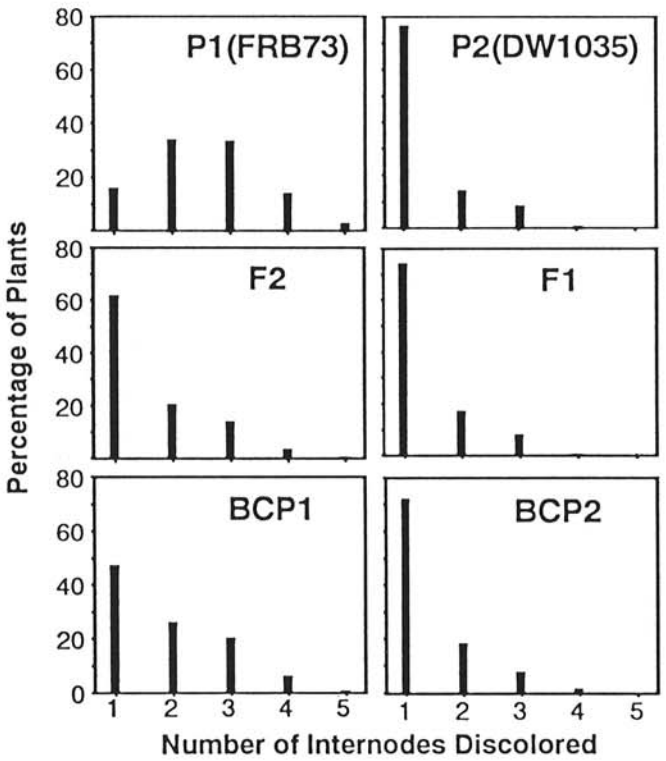

Fig. 1. Frequency distributions of plant stalk rot reactions, summed over 1987, 1988, and 1989 for number of internodes discolored, by *Colletotrichum graminicola* in the inbred lines FRB73 and DW1035 and the F_1, F_2, BCP_1, and BCP_2 generations.

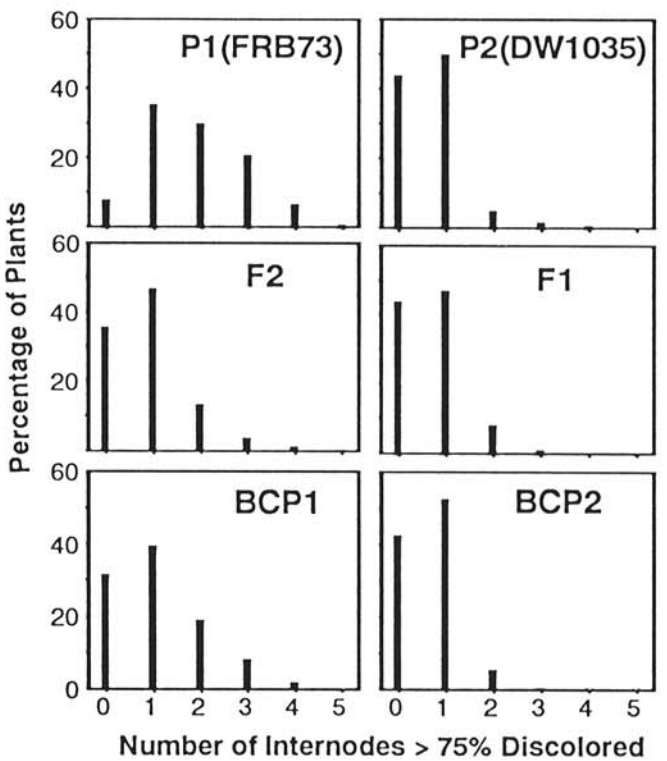

Fig. 2. Frequency distributions of plant stalk rot reactions, summed over 1987, 1988, and 1989 for number of internodes >75% discolored, by *Colletotrichum graminicola* in the inbred lines FRB73 and DW1035 and the F_1, F_2, BCP_1, and BCP_2 generations.

DISCUSSION

By generation mean analysis, we determined that additive and dominance genetic effects were of primary importance in all 3 yr and for each variable studied (number of internodes discolored, number of internodes >75% discolored, and the sum of internodes discolored plus internodes >75% discolored). In several quantitatively inherited traits, including disease resistance, generation mean analysis has been used to detect type of gene action involved (14,15). Major drawbacks associated with generation mean analyses lie in the assumptions made. 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 (21). 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 (21). In certain inheritances studies, it is useful not only to identify dominance genetic effects but also to try to quantify the number of genes involved.

TABLE 1. Analyses of variance of mean anthracnose stalk rot disease reactions of six generations derived from the cross FRB73 × DW1035 studied in 1987, 1988, and 1989 and all years combined

Variable[a]			Generations[b]			
Year	Year	Replicates	Additive	Dominance	Deviations	Error
NID						
1987						
df	...	3	1	1	1	15
MS[c]	...	0.394**	2.657**	0.821**	0.103	0.028
1988						
df	...	3	1	1	1	15
MS	...	0.044	7.779**	0.719**	0.067	0.034
1989						
df	...	3	1	1	1	15
MS	...	0.035	1.648**	0.824**	0.036	0.025
All years						
df	2	3	1	1	1	61
MS	4.434**	0.215*	10.842**	2.362**	0.173	0.055
NID >75%						
1987						
df	...	3	1	1	1	15
MS	...	0.147	3.318**	1.743**	0.124	0.084
1988						
df	...	3	1	1	1	15
MS	...	0.019	4.369**	0.779**	0.185	0.024
1989						
df	...	3	1	1	1	15
MS	...	0.130*	2.039**	0.977**	0.098	0.033
All years						
df	2	3	1	1	1	61
MS	3.179**	0.103	9.503**	3.396**	0.359	0.050
SUM						
1987						
df	...	3	1	1	1	15
MS	...	0.874**	11.925**	4.970**	0.415	0.179
1988						
df	...	3	1	1	1	15
MS	...	0.056	23.793**	3.004**	0.452	0.103
1989						
df	...	3	1	1	1	15
MS	...	0.285**	7.336**	3.635**	0.265	0.116
All years						
df	2	3	1	1	1	61
MS	9.835**	0.452	40.624**	11.482**	0.998	0.180

[a] NID = number of internodes discolored, NID >75% = number of internodes >75% discolored, and SUM = sum of number of internodes discolored plus number of internodes >75% discolored.
[b] Degrees of freedom = 5.
[c] Mean squares; * = significance at $P = 0.05$, ** = significance at $P = 0.01$.

Although estimates of the number of effective factors segregating in a population are imprecise and may be based on incorrect assumptions, they are reasonable estimates of the relative magnitude of gene number (21). Our estimates of the relatively low numbers of genes conditioning resistance to anthracnose stalk rot in the cross FRB73 × DW1035 is consistent with the observation that so-call polygenic disease resistance is usually controlled by a few genes (29). Based on the significant dominance from generation mean analysis, low estimates of effective factors, and frequency distributions of individual plant reactions, it was appropriate to begin by examining a single, dominant gene model.

Results suggest that DW1035 could differ from FRB73 for resistance to anthracnose stalk rot by a single, dominant gene. The lack of fit of the F_1 in 1988 and the BCP_1 and BCP_2 in 1989 was due to more individuals being classified as susceptible than expected for a single, dominant gene. The susceptible reactions could be real or could be attributed to environmental effects.

Stalk rot reactions of individual plants as resistant or susceptible may be masked by predisposing factors that make interpretation of results difficult. Dodd (10) proposed a photosynthetic stress-translocation balance concept of predisposition (sensu Colhoun [7]) of corn stalk rot in 1977, which relates stalk rot susceptibility to sugar movement in the plant. In support of the concept Dodd (10,11) observed that when interplant spacing and genotypes were constant, plants with more kernels had stalk rot and those with fewer kernels did not. Such environmental stresses as cloudiness, drought, fertilizer imbalance, poor water-holding capacity, and leaf disease epidemics are important predisposing factors that influence the rate of photosynthesis (11,12). The Midwest suffered a severe drought in the summer of 1988, and rainfall amounts in 1989 were well below average after pollination. Heterosis also relates to the photosynthetic stress-translocation balance concept and makes interpretation of stalk rot reactions difficult in the F_1 generation and to a lesser extent in other generations. Hybrid vigor results in large ears, which have a greater grain sink for sugars, causing earlier senescence of stalk tissue and therefore increasing susceptibility of the F_1 hybrid. The development of DW1035 using FRB73 as the recurrent parent was an attempt to minimize heterosis in the derived crosses with FRB73. The photosynthetic stress-translocation concept is a significant factor in the stalk rot complex; however, genetic differences do exist within the host for stalk rot resistance.

In our study, the generations that did not support the hypothesis of a single, dominant gene model also failed when tested for a two dominant gene model. Research identifying anthracnose stalk rot resistance with the inbred LB31 in crosses with B37 also failed to support a single, dominant gene model in some generations (2). Higher numbers than expected of individuals classified as susceptible can be explained by predisposition as it relates to the photosynthetic stress-translocation balance concept of Dodd (10). The greater grain sink for sugars on genetically

TABLE 2. Percentage of variation in anthracnose stalk rot reactions among generation means accounted for by additive and dominance genetic effects and deviations from additive dominance model[a]

	1987			1988			1989			All years		
Effect	NID	NID >75%	SUM	NID	NID >75%	SUM	NID	NID >75%	SUM	NID	NID >75%	SUM
Additive	72.1	64.4	69.1	90.3	81.9	87.1	65.2	63.7	64.1	80.8	71.6	76.6
Dominance	22.3	33.9	28.8	8.3	14.6	11.0	32.6	30.5	31.8	17.6	25.6	21.7
Deviations	5.6	1.7	2.1	1.4	3.5	1.9	2.2	5.8	4.1	1.6	2.8	1.7

[a] Data are from analysis of variance in 1987, 1988, and 1989 and combined analyses over all 3 yr. Reaction variables are: NID = number of internodes discolored, NID >75% = number of internodes >75% discolored, and SUM = sum of number of internodes discolored plus number of internodes >75% discolored.

resistant individual plants can cause earlier senescence of stalk tissue and therefore increase susceptibility. Higher numbers than expected of individuals classified as resistant can be explained by this same concept, where genetically susceptible plants may have a resistant phenotypic reaction due to a small grain sink for sugars. Other contributing factors, such as poor inoculation technique, failure to detect discoloration during the evaluation process, invasion by other stalk rot pathogens, and damage by the European corn borer, can result in misclassification. The large number of plants that are inoculated and evaluated during a stalk rot inheritance study inevitably results in misclassification of some individual plants.

The discriminant analysis procedure eliminates bias that may occur in establishing resistant and susceptible classes. Once the individual plant is classified as resistant or susceptible, genetic models can be tested using expected distributions determined by the partitioning method. The partitioning method can help account for the misclassification of individual plants that may occur between classes. This method will provide a useful tool in other inheritance studies where qualitative classes must be defined and the possibility of misclassification of individual plants exists. This method would be especially useful in studies where environmental conditions, other pathogens, or nonuniform inoculation procedures may lead to escapes. Although this procedure gives a more accurate depiction of the amount of genetic dominance than does generation mean analysis, it also assumes the absence of gene linkage between interacting loci.

In another study, 80 chromosomal positions, covering all chromosomes, were examined for restriction fragment length polymorphism (RFLP) between DW1035 and FRB73. Since DW1035 is a backcross version of FRB73, the only chromosomal regions that may contribute to differences in anthracnose stalk rot resistance between them are those retained in DW1035 from the donor parent, MP305. All probes detecting polymorphism between DW1035 and FRB73 map to the long arm of chromosome 4. These probes were used in further genetic analysis of a limited number of F_3 and backcross progeny (BCP_1 and BCP_2). The results of this study suggest that there are two genes closely linked for anthracnose stalk rot resistance (8).

The identification of two closely linked genes could not have been obtained by the traditional methods of generation mean analysis, determination of effective factors, or the testing of a single or a two dominant gene model. All of these analyses require the assumption that the genes affecting the trait segregate independently. Results from RFLP analysis help explain why neither single gene models nor models assuming two independent genes could account for the observed results in all years and generations. The presence of two closely linked genes for resistance in MP305 also accounts for the conclusion of Carson (3). The hypothesis of modifiers of a major gene is often invoked after the data do not support the conclusion of a single gene or of two independent genes. Identification of genetically resistant and susceptible individual plants is more precise with RFLP data because the variation influenced by predisposition, environmental conditions, nonuniform technique, and other pathogens is eliminated.

TABLE 3. Estimates of narrow-sense heritabilities (h^2) and number of effective factors (K_1) involved in resistance to anthracnose stalk rot in the cross of FRB73 × DW1035 in 1987, 1988, and 1989 and all years combined

Year	Variable[a]	Narrow-sense heritability[b]	Effective factors[c] (no.)
1987	NID	9.2	1.48
	NID >75%	40.0	0.38
	SUM	37.9	0.48
1988	NID	48.7	0.94
	NID >75%	47.5	0.94
	SUM	52.4	0.95
1989	NID	15.4	2.05
	NID >75%	39.3	0.65
	SUM	17.3	2.08
All 3 yr	NID	21.7	1.28
	NID >75%	42.1	0.60
	SUM	36.6	0.88

[a] NID = number of internodes discolored, NID >75% = number of internodes >75% discolored, and SUM = sum of number of internodes discolored plus number of internodes >75% discolored.
[b] Estimated by: $h^2 = [(2V_{F2} - V_{BCP1} - V_{BCP2})/V_{F2}] \times 100$.
[c] Estimated by: $K_1 = (P_1 - P_2)^2/8(2V_{F2} - V_{BCP1} - V_{BCP2})$.

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TABLE 4. Segregation for resistance to *Colletotrichum graminicola* in corn parental lines and F_1, F_2, and backcross generations involving crosses of the susceptible (S) inbred FRB73 with the resistant (R) inbred DW1035

	1987			1988			1989		
Generation	Obs. ratio[a] (R:S)	Exp. ratio[b] (R:S)	χ^2[c]	Obs. ratio (R:S)	Exp. ratio (R:S)	χ^2	Obs. ratio (R:S)	Exp. ratio (R:S)	χ^2
P_1 (FRB73)	52:136	...	...	15:199	...	...	65:242	...	...
P_2 (DW1035)	137:37	...	...	184:26	...	...	268:48	...	...
F_1 ($P_1 \times P_2$)	66:16	...	0.109[d]	112:80	...	43.171**[e]	255:43	...	0.052
F_2 ($P_1 \times P_2$)	335:183	344:174	0.625	342:279	322:299	2.453	633:252	610:275	2.671
BCP_1 ($P_1 \times P_2$) P_1	192:163	190:165	0.025	151:264	135:280	2.638	400:221	326:295	34.884**
BCP_2 ($P_1 \times P_2$)P_2	314:57	295:76	5.663*	267:97	255:109	1.732	404:122	437:89	14.285**

[a] Observed ratios obtained with SAS discriminant analysis procedure (26) based on discriminant function for parental lines.
[b] Expected ratios obtained with partitioning method of genetic analysis to test single gene model.
[c] Chi-square test for homogeneity in F_2, BCP_1, and BCP_2 generations calculated with Yates correction for continuity.
[d] Chi-square test for independence between P_2 and F_1 generations calculated with contingency tables.
[e] ** = $P < 0.01$, * = $P < 0.05$ but > 0.01.

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