Genetic Analysis of *Exserohilum turcicum* Lesion Expansion on Corn

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ABSTRACT

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All possible crosses between four corn inbred lines known to differ in reaction to *Exserohilum turcicum* were made in order to study the genetics of polygenic resistance to the fungus. Inbreds and single crosses were inoculated in the field, at two locations, with a conidial suspension of the pathogen. Lesion measurement commenced 3 wk after inoculation and continued every other day for a total of five measurements. Polynomial curve fitting of the lesion expansion data was used to estimate genetic effects. Lesion area, lesion expansion rate, and the shape of the lesion area expansion curve appeared to be under host genotype control. By means of regression techniques, the data were fitted to four genetic models. All the models had significant deviations from regression; the mean squares for deviations were smallest for the model that assumed additive and unequal genetic effects. Diallel analysis showed that general combining ability effects were much larger than specific combining ability effects. The results indicate that genetic analyses of lesion expansion curves would provide more information than analyses at a given time.

Additional key words: *Helminthosporium turcicum*, horizontal resistance, maize, northern leaf blight, quantitative inheritance, *Zea mays*.

Efforts to control northern leaf blight (NLB) of corn (*Zea mays* L.), caused by *Exserohilum turcicum* (Pass.) Leonard & Suggs (syn. *Helminthosporium turcicum* Pass.; perfect state, *Setosphaeria turcica* (Luttrell) Leonard & Suggs, syn. *Trichometasphaeria turcica* Luttrell), have been directed toward resistance since the studies of Jenkins and Robert (13–15) and Jenkins et al (16–18). Results of their studies indicated that resistance was controlled by several genes and could be transferred by phenotypic recurrent selection. Additive effects were most important, although dominance and epistatic effects were observed in another study (12).

Monogenic resistance to *E. turcicum* is available and is expressed as chlorotic lesions with negligible sporulation (7–10). In recent years, monogenic resistance, usually in lines with the Ht_1 gene, has been used as a primary means of control of NLB (11). The existence of physiological specialization in *E. turcicum* (4,21,24) and subsequent virulence to Ht_1 emphasizes the importance of polygenic resistance in the control of NLB. This resistance can be used alone or in combination with monogenic resistance to minimize yield loss if the monogenic resistance becomes ineffective.

Corn inbred lines CI28A, CI42A, and CI64 were developed by crossing susceptible inbreds B2, HY, and K64, respectively, with inbred Mo21A, which contains polygenic resistance to *E. turcicum* (17). CI28A, CI42A, and CI64 carry polygenic resistance factors on three, five, and six chromosome arms, respectively (15). Differences in the amount of resistance conferred by these three inbreds were demonstrated previously (19). Measurements of lesion size and number indicated that CI28A exhibited the least amount of resistance, CI42A was intermediate, and CI64 was the most resistant. Gregory et al (6) measured the size of NLB lesions of inbreds CI28A, CI42A, CI64, and R4 and their hybrids from a diallel crossing scheme. R4 has no resistance factors (13). Crosses having R4 as a parent, which would have resistance factors in a heterozygous condition, had lesions that were intermediate in size, compared to lesions on the respective parents, which carried the factors in a homozygous condition.

The research reported here is an expansion of earlier research with CI28A, CI42A, CI64, and R4 (6). The objectives of the research were twofold: first, to determine if simple genetic models, based on the presence of specific chromosome arms known to be present in the inbreds, could adequately explain the variation observed in the lesion expansion rate and the size of lesions caused by *E. turcicum* and, second, to determine the effect of host genotype on the rate of lesion expansion and the shape of the lesion expansion curve.

MATERIALS AND METHODS

Corn inbreds CI28A, CI42A, CI64, and R4 were crossed in all possible combinations, including reciprocals. The inbred lines and the 12 single crosses were planted at two locations, Rock Springs and University Park, near State College, Pennsylvania, on 4 and 15 June 1984, respectively. Because of the size difference between the inbreds and the single crosses, an experimental unit consisted of three rows. Data were collected from the center row only. Chromosome arms of the inbreds carrying resistance genes as identified by Jenkins and Robert (15) are shown in Table 1. Similarly, chromosome arms with resistance genes are identified for the 12 single crosses.

A single isolate of *E. turcicum* race 2, collected in Lancaster County, Pennsylvania, in 1983, was selected for this study on the basis of the uniformity and size of lesions produced on R4. For long-term storage of the pathogen, several plants of R4 were inoculated in the greenhouse with a conidial suspension made from single spore isolation of field leaf material. After lesion development, diseased leaves were harvested, dried, and stored for later use.

Cultures for inoculum production were grown by single-sporing from leaf material onto potato-dextrose agar in petri dishes, transferring the resulting colony onto lactose-casein hydrolysate agar (23) in petri dishes, and incubating in the dark at room temperature for 1 mo. To prepare inoculum, the contents of three to five petri dishes were homogenized in a blender with 500 ml of water and two drops of polyoxyethylene sorbitan monolaurate (Tween 20, Atlas Chemical Industries, Inc.). This suspension was filtered through cheesecloth, which removed most of the mycelial fragments. Inoculum, adjusted to a concentration of 3,000 conidia per milliliter, was sprayed onto all plants to runoff in the center row of each experimental unit, with a compressed-air backpack sprayer. Inoculation at Rock Springs occurred on 20 July and at University Park on 20 August, when the plants were at the six- to

eight-leaf stage.

Measurements of lesion length and width began 3 wk after inoculation and continued every other day for a total of five measurements. One lesion each from five plants per experimental unit was selected from separate plants in the center row. Lesions selected for measurement were on one of the three leaves below the ear and were chosen on the basis of uniform shape and distribution on the leaf. The same lesions were used for all five measurements. Uninoculated plants were free of *E. turcicum* lesions when the measurements began, so it is assumed that all lesions resulted from controlled inoculation. Length and width measurements for each lesion were converted to area (length times width, in square centimeters), which was used for statistical analyses. Preliminary studies indicated this was an adequate approximation of lesion area.

The experimental design consisted of observations over time in a randomized complete block experiment with four replications and 16 treatments (four inbred lines plus the 12 single crosses) at each of two locations. Orthogonal contrasts (1) were used to determine polynomial regression responses of lesion size over time. The regression equation was $\hat{Y}_{ij} = \bar{Y}_i + b_{i1}C_{1j} + b_{i2}C_{2j} + b_{i3}C_{3j}$, where $\bar{Y}_i$ is the mean lesion area over the entire time period; b_{i1}, b_{i2}, and b_{i3} are linear, quadratic, and cubic regression coefficients, respectively; and the C_{ij}s are orthogonal contrast coefficients, obtained from reference 1. An important difference between the polynomial regression and the "usual" regression equations is that $\bar{Y}_i$ is the mean of all observations contributing to the curve rather than the intercept. Estimates of the mean ($\bar{Y}$) and each of the regression coefficients (b_1, b_2, and b_3) were computed for each lesion. Estimates of the mean and regression coefficients are linear functions of the original observations of lesion size. Linear functions of normally distributed variables are also normally distributed (3). Thus, our estimates of the means and polynomial regression coefficients were normally distributed, assuming the original observations were from a normal distribution. These estimates were subjected to an unweighted means analysis of variance (22). Variation due to observations within plots was always smaller than the replicate by entry mean square; therefore, only the analysis of plot means is presented in this paper.

The response curve for each genotype was predicted from the average values for the mean area and the regression coefficients obtained from the unweighted means analysis of variance. Regression coefficients for a genotype that did not exceed twice their respective standard errors were assumed to be zero and were not included in the estimated response curve. Thus, response curves for the different genotypes could differ because of variations in estimates of the mean curve parameters or because one or more parameter estimates were judged to be not significantly different from zero.

The knowledge of chromosome areas containing resistance factors in the inbred lines permitted testing of successively more complex genetic models for the response of lesion size. Estimates of the mean and the regression coefficients as described above were the dependent variables. Four genetic models in which chromosome areas were treated as units were tested before application of the diallel analysis.

In Model I, $\hat{Y}_i = a + bX_i$, where $\hat{Y}_i$ is the observed response for genotype i, a is the intercept, b is the regression coefficient, and X_i is the total number of unique chromosome segments with resistance factors for genotype i (Tables 1 and 2). Model I was constructed with the assumption that all resistance factors were additive and had equal effects.

In Model II, $\hat{Y}_i = a + bX_i$, where $\hat{Y}_i$, a, and b are the same as for Model I, and X_i is the total number of chromosome segments with resistance factors for genotype i. Model II assumes that all resistance factors had equal effects and were completely dominant.

In Model III, $\hat{Y}_i = a + bX_{1i} + bX_{2i} + bX_{3i}$, where $\hat{Y}_i$ is the observed response; X_{1i} is the number of 3L, 5L, and 7S chromosome arms with resistance factors in genotype i (L and S refer to long and short arms, respectively); X_{2i} is the number of 2L and 8L arms with resistance factors in genotype i; X_{3i} is the number of 4L, 4S, and 9L arms with resistance factors in genotype i; a is the intercept; and b is the regression coefficient. The X values reflect the allotment of chromosome arms with resistance factors among the different inbreds. Model III was constructed with the assumption that all resistance factors were additive and had different or unequal effects.

Model IV was similar to Model III except the X values were computed with the assumption that all resistance factors were completely dominant and had unequal effects.

The diallel model was tested by partitioning the sum of squares for genotypes into variation due to parents, parents versus hybrids,

TABLE 1. Identification of chromosome arms containing resistance factors to *Exserohilum turcicum* for four corn inbred lines and genotypes resulting from diallel crossing of the four inbreds

	Chromosome arms[a]							
	3L	5L	7S	2L	8L	4L	4S	9L
R4	0/0[b]	0/0	0/0	0/0	0/0	0/0	0/0	0/0
CI28A	+/+	+/+	+/+	0/0	0/0	0/0	0/0	0/0
CI42A	+/+	+/+	+/+	+/+	+/+	0/0	0/0	0/0
CI64	+/+	+/+	+/+	0/0	0/0	+/+	+/+	+/+
R4 × CI28A	0/+	0/+	0/+	0/0	0/0	0/0	0/0	0/0
CI28A × R4	+/0	+/0	+/0	0/0	0/0	0/0	0/0	0/0
R4 × CI42A	0/+	0/+	0/+	0/+	0/+	0/0	0/0	0/0
CI42A × R4	+/0	+/0	+/0	+/0	+/0	0/0	0/0	0/0
R4 × CI64	0/+	0/+	0/+	0/0	0/0	0/+	0/+	0/+
CI64 × R4	+/0	+/0	+/0	0/0	0/0	+/0	+/0	+/0
CI28A × CI42A	+/+	+/+	+/+	0/+	0/+	0/0	0/0	0/0
CI42A × CI28A	+/+	+/+	+/+	+/0	+/0	0/0	0/0	0/0
CI28A × CI64	+/+	+/+	+/+	0/0	0/0	0/+	0/+	0/+
CI64 × CI28A	+/+	+/+	+/+	0/0	0/0	+/0	+/0	+/0
CI42A × CI64	+/+	+/+	+/+	+/0	+/0	0/+	0/+	0/+
CI64 × CI42A	+/+	+/+	+/+	0/+	0/+	+/0	+/0	+/0

[a] L and S refer to long and short arms, respectively, of each chromosome.

[b] + = resistance factor present, and 0 = no resistance factor present, from the female (to the left of the slash) and the male (to the right of the slash) used in cross. Information on inbred lines is from Jenkins and Robert (13).

and hybrids. The hybrid sum of squares was partitioned further into general combining ability (GCA), specific combining ability (SCA), maternal effects, and reciprocal effects, following the procedures of Pederson et al (20), which are a minor modification of Analysis III of Gardner and Eberhart (5). Location and genotypes were considered fixed effects in all of the analyses. An analysis combined over locations was conducted in order to determine if there were significant location interaction effects.

RESULTS

The curves in Figure 1 graphically display the progress of lesion expansion over the five sampling dates. The data agree with that of Gregory et al (6) in that the lesion area of crosses involving R4 was intermediate to that of the parents involved. Increasing numbers of resistance genes generally resulted in smaller lesion areas, although the difference between CI28A, with three chromosome arms containing resistance genes (Table 1), and CI42A, with five chromosome arms containing resistance genes, was minimal. CI64 exhibited the most resistance in terms of lesion size.

Analysis of variance of the mean and the regression coefficients (Table 3) suggests that the mean lesion size over time was significantly greater at University Park than at Rock Springs. The linear, quadratic, and cubic effects were similar at both locations, which was expected, since the shapes of the curves were similar (Fig. 1). Genotypes were a significant source of variation for all regression response parameters. The location × genotypes interaction was significant for the mean, linear, and quadratic responses. Further analyses of variance for separate locations showed significant genotype variation for all effects at each location except for the cubic effect at Rock Springs. Examination of Figure 1 indicates that most genotypes responded similarly at each location. There are some exceptions, notably R4, CI28A × R4, and CI28A × CI64. Locations differed more in the magnitude of differences between genotypes than in the ranking of genotypes between locations. In all cases, levels of significance were similar. Since the significant location × genotypes interaction appeared to be due to differences in the magnitude of variation at each location, and since the lesion growth curves were similar at each location, the data are presented with locations combined.

On the basis of the relative size of the mean squares for deviation from regression, Model III provided the best fit of the first four genetic models tested (Table 4). Deviation from regression was smallest for Model III in all cases, but it was significant for all parameters other than the cubic effect. Significant deviation from regression suggests that the model tested does not adequately explain the variation observed. Since these deviations were significant for all of the first four models tested, Models I through IV were rejected, and the data were fitted to the diallel model.

Significant variation between parents was observed for mean lesion area and for two of the three regression coefficients (Table 4). The mean lesion area was greater and increased at a greater rate with time for R4 than for any of the other parents (Fig. 1). The response curve for R4 was nonlinear, in that the lesion area expansion rate increased during the time of measurements. Curves for CI28A and CI42A were nearly linear, and a slight increase in lesion area occurred with time. Lesions on CI64 were small during the measurement period.

The mean of the parents was significantly different from the mean of the hybrids for mean lesion area and the linear regression coefficient (Table 4). Most of this significant difference was attributed to the response of R4, which had a greater mean lesion area and a greater increase in area with time than any other entry in the experiment.

GCA effects were significant for mean lesion area and for each of the regression coefficients (Table 4). The greatest lesion area and the greatest rate of increase in lesion area were observed in crosses with R4 (Fig. 1). Lesion area increased more rapidly between day 7 and day 9 in crosses with R4 than between earlier days of the measurement period. GCA means were greater than zero, and lesion area increased with time in crosses with the resistant parents. A large portion of the GCA effect for the resistant parents was the result of the response of crosses between the resistant parents and R4.

Significant SCA variation was observed for mean lesion size and for the linear and quadratic regression coefficients (Table 4). The significant SCA effects were attributed to the observation that hybrids with R4 were more susceptible and hybrids with CI64 and

TABLE 2. X values used for genetic models assuming genes on chromosome arms had equal and additive effects, equal and completely dominant effects, unequal and additive effects, and unequal and completely dominant effects

	Equal effects		Unequal effects					
	Additive (Model I)	Dominant (Model II)	Additive (Model III)			Dominant (Model IV)		
Entry	X_i[a]	X_i[b]	X_{1i}[c]	X_{2i}[d]	X_{3i}[e]	X_{1i}[f]	X_{2i}[g]	X_{3i}[h]
R4	0	0	0	0	0	0	0	0
CI28A	6	3	2	0	0	1	0	0
CI42A	10	5	2	2	0	1	1	0
CI64	12	6	2	0	2	1	0	1
R4 × CI28A[i]	3	3	1	0	0	1	0	0
R4 × CI42A	5	5	1	1	0	1	1	0
R4 × CI64	6	6	1	0	1	1	0	1
CI28A × CI42A	8	5	2	1	0	1	1	0
CI28A × CI64	9	6	2	0	1	1	0	1
CI42A × CI64	11	8	2	1	1	1	1	1

[a] X_i = number of unique chromosome arms with resistance factors in entry i.
[b] X_i = number of chromosome arms with resistance factors in entry i.
[c] X_{1i} = number of 3L, 5L, and 7S unique chromosome arms with resistance factors in entry i.
[d] X_{2i} = number of 2L and 8L unique chromosome arms with resistance factors in entry i.
[e] X_{3i} = number of 4L, 4S, and 9L unique chromosome arms with resistance factors in entry i.
[f] X_{1i} = number of 3L, 5L, and 7S chromosome arms with resistance factors in entry i.
[g] X_{2i} = number of 2L and 8L chromosome arms with resistance factors in entry i.
[h] X_{3i} = number of 4L, 4S, and 9L chromosome arms with resistance factors in entry i.
[i] Reciprocal crosses have the same X values.

CI42A were more resistant than expected on the basis of the respective parental averages. The SCA mean squares were smaller than their respective GCA mean squares in each case, which indicates that GCA is the more important source of variation.

Neither maternal nor reciprocal effects were significant for any of the traits measured (Table 4). Reciprocal curves were similar for each of the parental combinations (Fig. 1).

DISCUSSION

A decision was made to emphasize the analysis of lesion expansion rate in this experiment, which required the collection of a large amount of data on a relatively small number of genotypes. Available resources, including the availability of suitable inbred lines for parents, required the use of a small diallel. Assumptions required for the estimation of genetic variances in diallel crosses (2) were not met in our experiment, and extrapolation to broader corn populations should be made with caution.

Nonetheless, our results indicated that the average level of resistance (mean lesion area), the rate of increase in lesion size (linear regression with time), and the shape of the lesion size increase curve (quadratic and cubic regression coefficients) are strongly influenced by the host genotype. The genetic control is a type that is manifested largely through an average expression of

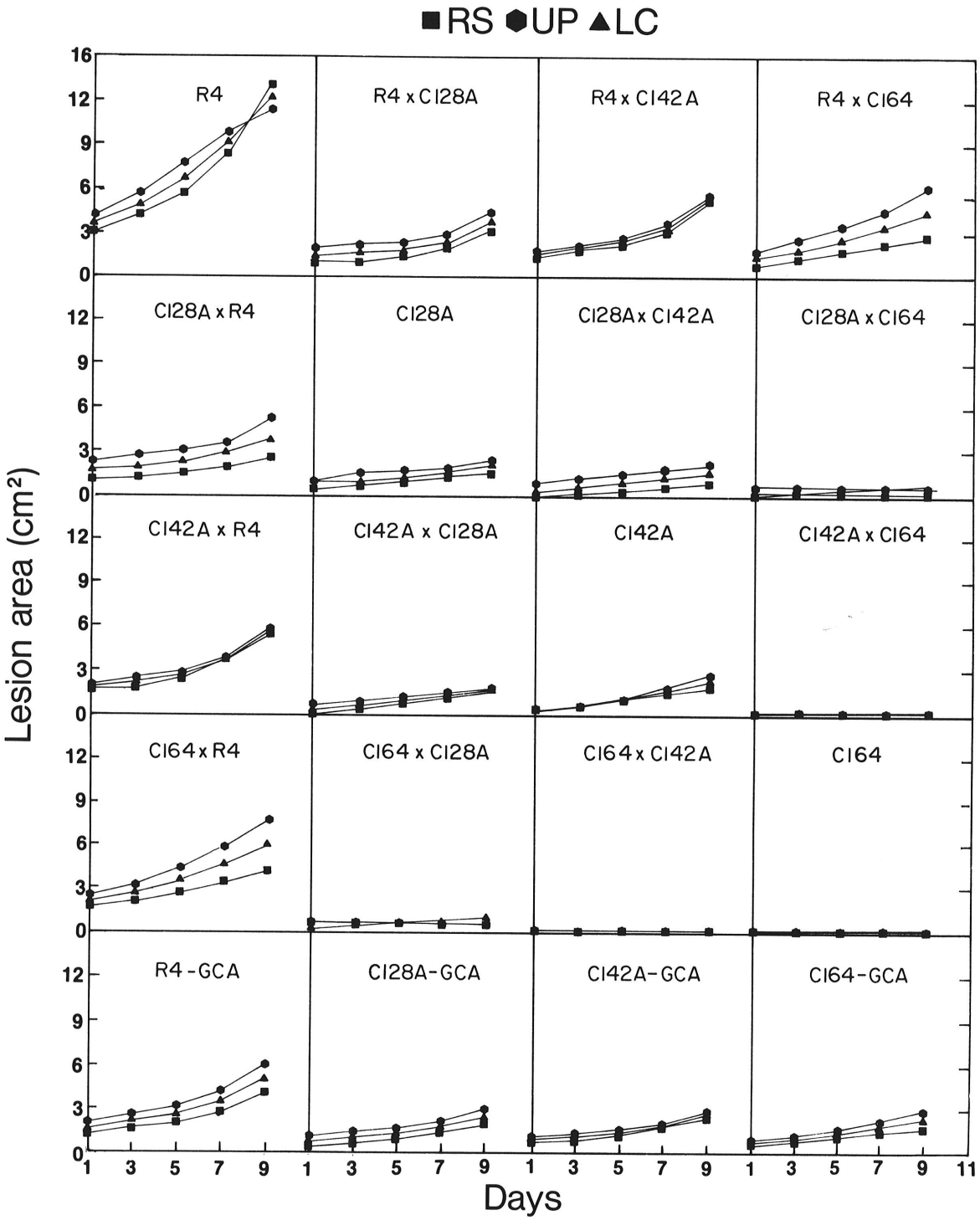

Fig. 1. Curves plotting lesion area versus time for corn inbreds R4, CI28A, CI42A, and CI64 and single crosses in a diallel mating scheme after inoculation with *Exserohilum turcicum* at Rock Springs (RS), University Park (UP), and these locations combined (LC). The bottom row represents the average curves of all crosses involving the four inbreds (GCA = general combining ability).

TABLE 3. Analysis of variance of mean area and linear, quadratic, and cubic responses associated with lesion expansion resulting from inoculation of 16 corn genotypes with *Exserohilum turcicum* at two locations

		Mean squares[a]			
			Regression coefficients		
Source	df	Mean area	Linear	Quadratic (10^{-1})	Cubic (10^{-1})
Location	1	16.37**	0.19	0.00	0.00
Replications in location	6	0.46	0.10	0.09	0.13
Genotypes	15	27.99**	2.33**	0.82**	0.12
Location × genotypes	15	1.03**	0.16**	0.47**	0.13
Error	90	0.45	0.07	0.07	0.07

[a] Asterisks (**) denote significance at $p = 0.01$.

TABLE 4. Further breakdown of genotypic variation associated with mean area and linear, quadratic, and cubic responses of lesion expansion resulting from inoculation of 16 corn genotypes with *Exserohilum turcicum turcicum* at two locations

		Mean squares[a]			
			Regression coefficients		
Source	df	Mean area	Linear	Quadratic (10^{-1})	Cubic (10^{-1})
Genotypes[b]	15	27.99**	2.33**	0.82**	0.12
Model I					
Regression	1	316.92**	23.52**	8.78**	0.75**
Deviation	14	7.35**	0.81**	0.26**	0.07
Model II					
Regression	1	218.37**	16.79**	5.00**	0.22
Deviation	14	14.39**	1.29**	0.52**	0.11
Model III					
Regression	3	125.96**	9.99**	3.59**	0.31**
Deviation	12	3.50**	0.41**	0.13*	0.07
Model IV					
Regression	3	93.26**	7.93**	1.92**	0.16
Deviation	12	11.67**	0.93**	0.55**	0.10
Diallel analysis					
Parents	3	84.76**	7.48**	1.35**	0.02
Parents vs. hybrids	1	13.92**	1.44**	0.01	0.09
Hybrids	11	13.78**	1.00**	0.76**	0.15
GCA[c] effects	3	45.25**	3.07**	2.38**	0.46**
SCA[c] effects	2	6.79**	0.85**	0.44**	0.04
Maternal effects	3	0.52	0.03	0.02	0.02
Reciprocal effects	3	0.24	0.01	0.08	0.03
Error[b]	90	0.45	0.07	0.07	0.07

[a] Asterisk (*) denotes significance at $p = 0.05$. Double asterisk (**) denotes significance at $p = 0.01$.
[b] From Table 3.
[c] GCA = general combining ability; SCA = specific combining ability.

genes contributed by each parent (GCA), with a small amount of variability due to specific interactions between parents (SCA) present. Regression analysis with the four simple genetic models indicated that the genotypic response could not be completely explained by simple additive or complete dominance genetic models, although the model constructed under the assumption that genes for resistance were additive, with different effects for the different chromosomes, had the smallest residual sum of squares. Although a genetic study with a larger and different set of inbred lines would probably exhibit differences in the relative magnitudes of SCA and GCA, we doubt that our conclusion that host genotype influences the mean lesion size, the rate of lesion size increase, and the shape of the lesion size increase curve would change.

The fact that lesion growth rates differ and the increase is not always linear implies that multiple measurements are needed for accurate assessment of reaction to *E. turcicum*. A genetic interaction with time exists, because response curves differ with genotype. Two entries with similar means early in the season could be markedly different at a later date. The most desirable genotypes would be those in which disease development was delayed and growth rate was slow—an evaluation that can only be made with more than one assessment.

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