Host Resistance to Stewart's Disease in Maize

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ABSTRACT

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The effect of host genotype, cytoplasm, and rating date on development of Stewart's wilt (*Erwinia stewartii* [E. F. Smith] Dye) of corn (*Zea mays* L.) was studied. The 15 possible single crosses among six inbred line parents in both normal and Texas male-sterile cytoplasm were grown in a split-plot design, with the split on cytoplasms. In a diallel analysis, inbred parents B37, 33-16, and Mo17 had significant negative general combining ability (gca) effects for each of three rating dates. The parents B14A and N28 had significant positive gca effects, with B14A conditioning susceptibility in a dominant manner. Reaction to Stewart's disease was largely additive in this set of crosses, as the gca mean square was 20 times the magnitude of the specific combining ability (sca) mean square. Therefore, the conventional recurrent selection method should be effective in improving populations for resistance to Stewart's wilt disease. The most resistant plants could be identified before anthesis when artificially inoculated at about the 12th leaf stage. Highly significant differences were found among the 15 genotypes, three rating dates, and two cytoplasms, the differences between cytoplasms being the least important. Disease response interactions were significant for genotypes × rating date, cytoplasm × rating date, gca × rating date, and sca × rating date. The genes conditioning lesion development were determined to be inherited qualitatively by the Castle-Wright formula. Diallel mating schemes using multiple disease ratings over time should be useful in studying gene action affecting disease development or general resistance.

Additional key words: bacterial wilt.

Stewart's wilt and leaf blight incited by the bacterium *Erwinia stewartii* (E. F. Smith) Dye has long been known as an important disease of both sweet corn (*Zea mays* var. *saccharata*) and dent corn (*Z. mays* var. *indentata*). Intensive studies were initiated during the 1930s, when *E. stewartii* caused catastrophic losses, but research soon subsided with the rapid success of locating resistant germ plasm and incorporating it into adapted strains. In recent years, Stewart's disease has again become prominent on sweet corn in parts of the northeast (4) and on dent corn in the southern corn belt.

Ivanoff and Riker (5) suggested in 1936 that three factors were involved in resistance: vigor, maturity, and true genetic resistance. They found that host resistance was inherited in a partially dominant manner. Wellhausen (10) postulated in 1937 that genetic resistance was governed by two completely dominant complementary unlinked genes, designated Sw_1 and Sw_2. More recently, Smith (7) proposed that two major dominant genes (Sw_1 and Sw_2) and two modifier genes (Sw_3 and Sw_4) were associated with resistance to *E. stewartii*.

All previous studies dealt with the inheritance of Stewart's disease reaction in a single time period. We have observed that maize genotypes differ in both the extent and the rate at which they become diseased. The purpose of this study was to improve knowledge of Stewart's disease and of the type of gene action that influences disease increase during plant growth and development.

MATERIALS AND METHODS

The interrelationship of host genotype, cytoplasm, and Stewart's disease development was studied among six inbred lines and their diallel sets of nonreciprocal crosses. Three lines (B37, Mo17, and 33-16) were previously classified as resistant (R), one line (Va35) was classified as intermediate (I), and two lines (B14A and N28) were classified as susceptible (S). The six lines were in normal (N) and Texas male-sterile (cms-T) cytoplasm. In addition, F_2 generation seed for each F_1 cross was generated by selfing plants in the N cytoplasm and by crossing each fertile N hybrid to the male-sterile female counterpart in cms-T.

A single culture of *E. stewartii* was used as the source of inoculum. The isolate was obtained from an infected plant found in a field located near Golden City, MO, in 1975. The pathogen was identified as *E. stewartii* by the method of Turner et al (9) and maintained on nutrient agar. Several weeks after collection, the culture was lyophilized in test tubes and stored at room temperature. The original lyophilized culture was used as the source of inoculum in all inoculation trials. Inoculum was prepared by growing the lyophilized cultures in nutrient broth in Erlenmeyer flasks and shaking it for 24 hr at room temperature. The inoculum was diluted to five times its original volume before inoculation.

The inoculator device (1) consisted of two household sponges attached to the end of two 30-cm long cedar boards. Embedded in each sponge were 30 23-mm brads. Plants were inoculated by saturating the sponges in the bacterial suspension and clamping the inoculator through the whorl of each plant. Additional inoculum was applied directly into the whorl with a hand sprayer. This method introduced the pathogen into the innermost leaves of the whorl and simultaneously infected four to six leaves. Although the bacterial concentration was not calibrated, the same suspension was used throughout inoculation and every effort was made to standardize procedures among workers. All plants were inoculated on 30 June (plants were 7 wk old) and again on 8 July 1976.

Disease ratings were made three times during the growing season: 19 July, 12 August, and 3 September, corresponding to about 50% tasseling, the soft-dough stage, and physiological maturity, respectively. A descriptive rating scale (Table 1) ranging from 1 = most resistant to 9 = plant dead was used for all three dates. The scale was based on the development of disease, ie, amount of necrotic tissue, from the inoculation site. Each plant was rated individually. Ratings of all plants in a plot were averaged, and the plot mean was used in the analysis.

The 15 single crosses in both cytoplasms were planted in a split-plot design in time and space as described by Steel and Torrie (8). Each single cross was randomized as a whole plot, with subplots of

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paired rows for each cytoplasm. Plots were planted on 13 May at the Rollins Bottom Nursery in Columbia, MO. Rows were 397 cm long and 91.4 cm apart, with three replications. Each row was thinned to a single plant per hill, with 13 plants per row. Griffing's Method IV Model I diallel (3), modified to include the effects of cytoplasms and rating dates, was used for analysis of variance. This analysis provided a test for the effects of general combining ability (gca) and specific combining ability (sca) and their interactions with rating dates and cytoplasms.

In addition, F_2 generations in both cytoplasms were planted in adjacent paired rows on 13 May, with two-row plots for each cytoplasm in four replications. Thus, twice as many F_2 as F_1 generation plants were observed. The parental inbred lines in both cytoplasms were also planted in paired rows in three replications. Inbred lines, single crosses, and F_2s were inoculated and rated on the same day or one day later.

Approximate gene numbers involved in the inheritance of *E. stewartii* reaction were estimated by Castle's formula, as modified by Wright (2):

$$n = \frac{D_2}{8\ (\sigma^2F_2 - \sigma^2F_1)}$$

where n is the estimated gene number, D is the difference between parental rating means, and σ is the variance of F_2 and F_1 generations. Environmental variance was calculated from individual ratings among the specific F_1 single cross, and genetic variance was computed as the difference between corresponding F_2 and F_1 variances. Variances were calculated for the pooled replications in the N cytoplasm for each susceptible X-resistant cross studied.

RESULTS

Natural infection with *E. stewartii* occurred in 1976, so ratings were based only on the extent of disease development from the site of inoculation. Because eliminating all bias of natural infection was impossible, more confidence was placed on the first two rating dates than on the final rating date. In addition to high levels of natural infection, plants rated at the final date were undergoing natural senescence and premature death due to drought.

The mean Stewart's disease rating for the six parental inbred lines at three rating dates is given in Table 2. No differences due to cytoplasms were detected, so the data were combined over cytoplasms for the six parental lines. On 19 July most of the plants of inbred B14A had necrotic lesions; this line showed symptoms first and had the highest disease rating on all three dates.

The rate of disease increase among the three resistant inbreds varied substantially between dates. Mo17 had the lowest initial rating (1.0, $P = 0.05$) and the slowest rate of disease development between 19 July and 12 August (0.6 units), but disease increased by 2.8 units between 12 August and 3 September. In contrast, disease in 33-16 developed faster initially (0.8 units) but increased by only 1.3 units between 12 August and 3 September. Disease tended to develop faster between the first and second rating dates in the two susceptible lines and between the second and third dates in the other lines, with the exception of 33-16. This probably was due in part to the confounding aspects of natural senescence with approaching plant maturity.

Highly significant differences were found among single crosses, rating dates, and cytoplasms (Table 3). The rating date mean square had the greatest magnitude, indicating a strong tendency for disease reaction to change with time. Although cytoplasms were highly significant, we interpreted the small mean square variance to indicate lesser importance than either of the other two main effects.

The gca mean square was highly significant and approximately 22 times the sca mean square. The additive genetic effects were more important than dominance or epistatic effects in the inheritance of Stewart's disease reaction in this set of 15 diallel crosses. The gca × rating date interaction was significant (not all crosses

TABLE 1. Rating scale for reaction of corn (*Zea mays*) to *Erwinia stewartii* in the field

Scale	Symptom
1	No visible chlorosis surrounding inoculator perforations
2	Localized chlorotic lesions
3	Buff-white lesions surrounding inoculator perforations; some necrosis
4	Necrotic lesions beginning to coalesce
5	Extensive coalescing and necrosis from site of inoculation to leaf tip
6	Inoculated leaves below ear dead
7	Two leaves above ear dead
8	Three or more leaves above ear dead
9	Plant dead

TABLE 2. Mean Stewart's disease ratings on three dates for the six inbred lines combined over cytoplasms

	Rating date		
Inbred line	19 July	12 August	3 September
B14A	3.4	5.5	7.0
N28	2.1	4.6	5.8
Va35	2.0	3.1	5.7
33-16	2.0	2.8	4.1
B37	1.4	2.6	5.0
Mo17	1.0	1.6	4.4
LSD[a] (0.05)	0.30	0.37	0.90
Coefficient of variation (%)	12.6	9.3	14.2

[a]LSD is least significant difference at $P = 0.05$.

TABLE 3. Analysis of a diallel of six inbred corn lines for 15 single crosses in two cytoplasms for reaction to *Erwinia stewartii* at three rating dates

Source of variation	Degrees of freedom	Mean square
Replications	2	1.01
Single crosses	14	6.59**[a]
General combining ability	5	17.11**
Specific combining ability	9	0.75
Error a	28	0.46
Rating dates	2	388.15**
Rating dates × single crosses	28	0.81**
General combining ability × rating dates	10	1.61**
Specific combining ability × rating dates	18	0.36*
Error b	56	0.16
Cytoplasms	1	1.40**
Cytoplasms × single crosses	14	0.17
General combining ability × cytoplasms	5	0.16
Specific combining ability × cytoplasms	9	0.19
Error c	30	0.18
Cytoplasms × rating dates	2	0.85**
Cytoplasms × rating dates × single crosses	28	0.23*
General combining ability × cytoplasms × rating dates	10	0.37**
Specific combining ability × cytoplasms × rating dates	18	0.15
Error d	60	0.13

[a]* and ** indicate statistical significance $P = 0.05$ and $P = 0.01$, respectively.

were responding the same for disease reaction at different rating dates). No significant interaction with cytoplasm was detected for either gca or sca. To a lesser extent, sca also depended on the time of rating and, like gca, did not depend on the cytoplasmic background of the single cross.

Mean disease ratings, gca effects, and sca effects combined over cytoplasms are given for the first rating (19 July) in Table 4. Line means were calculated by averaging the five crosses with a common inbred as parent. The parental line B14A had the highest line mean (2.2) and positive gca effect (0.51), whereas Mo17 had the lowest line mean (1.5) and negative gca effect (−0.42). Inbred lines B37 and 33-16 had almost equal negative gca effects of −0.22 and −0.27, respectively.

Disease reaction ratings increased significantly between 19 July and 12 August for all 15 single crosses. Inbred line B14A was again the most susceptible and had the highest positive gca effect (1.36) (Table 5). Mo17 remained the most resistant parental line and had the lowest negative gca effect (−0.61). Parental lines B37 and 33-16 still ranked closely in gca effects, with −0.47 and −0.48, respectively.

The rate of disease development among single crosses depended on the relative susceptibility of the parental lines used. Whereas resistant × resistant (R×R) crosses maintained high levels of resistance, the rate of disease development among all R×S and S×S crosses was rapid, especially when B14A was the parent. The single cross Mo17 × 33-16 (R×R) had the lowest disease rating on both 19 July and 12 August, with means of 1.2 and 2.2, respectively (Tables 4 and 5). The crosses B14A × N28 (S×S) and B14A × Va35 (S×I) were most susceptible on 19 July and 12 August, with disease ratings changing from 2.7 to 4.6 and from 2.6 to 5.0, respectively. The changes in single cross means for the susceptible × resistant parental lines B14A × B37 and B14A × Mo17 between the two rating dates were large. The former cross increased from 1.9 to 4.4 and the latter from 1.6 to 4.3, representing the greatest increase in disease development per unit of time among any of the 15 single crosses. Susceptibility may therefore be a genetically dominant trait in B14A single crosses. An alternative possibility is that the original inoculum concentration was very high and may have overcome low thresholds of host resistance. Further inoculation testing using various inoculum densities is needed to determine which hypothesis is correct.

Single cross B37 × Va35 (R×I) showed a highly significant negative sca effect (−0.47) for 12 August. Its disease rating mean (2.5) at this date was lower than would have been predicted from the gca estimates of the two parents involved. This single cross also had the lowest disease rating increase among the 15 single crosses between 19 July and 12 August.

Mean disease ratings and combining ability effects on 3 September are shown in Table 6. Most of the single crosses had high disease ratings, but premature senescence was certainly a factor. The parental inbred B14A retained a high positive gca effect (0.79), and the line mean of 6.5 was significantly higher than that of any of the other parental line means.

No significant differences among cytoplasms were detected at the first rating on 19 July. Although B14A combinations tended to be more susceptible in normal cytoplasm, line means between cytoplasms were not significant. At the 12 August rating, only one single cross (B37 × Mo17) in cms-T had a significantly greater mean disease rating than its normal counterpart, 2.8 vs. 2.1. Significant cytoplasmic differences were detected for three single crosses (B37 × N28, B37 × Va35, and Mo17 × 33-16) on 3 September. In all cases, the single crosses in normal cytoplasm had significantly higher disease ratings than their cms-T counterparts (Table 7).

Mean ratings of the 15 F_2 generation crosses were slightly higher but agreed closely with the means of their respective F_1 single crosses (results not tabulated). Rank correlation coefficients for the

TABLE 4. Mean Stewart's disease ratings of 15 single crosses on 19 July (above diagonal) and specific combining ability and general combining ability effects (below diagonal) combined over cytoplasms

Parental line	B14A	B37	Mo17	N28	Va35	33-16	Line means
B14A	...	1.9[a]	1.6	2.7	2.6	2.2	2.2
B37	-0.23*[b]	...	1.3	1.8	1.8	1.3	1.6
Mo17	-0.26*	0.10*	...	1.5	1.7	1.2	1.5
N28	0.22*	0.06	0.05	...	2.1	1.6	1.9
Va35	0.09	0.05	0.12*	-0.10*	...	1.6	2.0
33-16	0.18*	0.02	0.09	-0.13*	-0.16*	...	1.6
$\hat{g}_i$[c]	0.51*	-0.22*	-0.42*	0.20*	0.21*	-0.27*	...

[a]Least significant difference (LSD), ($P = 0.05$) = 0.59, denotes statistically significant differences among single cross means within or among rating dates.
[b]LSD ($P = 0.05$), ($\hat{s}_{ij}$) = 0.10 denotes statistical significance from 0 for any specific combining ability effect; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{ik}$) = 0.52 denotes statistical significance between specific combining ability effects among crosses with one parent in common; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{kl}$) = 0.42 denotes statistical significance between combining ability effects among crosses with no parent in common; LSD ($P = 0.05$), ($\hat{g}_i$) = 0.14 denotes statistical significance from 0 for any general combining ability effect; LSD ($P = 0.05$), ($\hat{g}_i - \hat{g}_j$) = 0.29 denotes statistical significance between general combining ability effects.
[c]$\hat{g}_i$ = general combining ability effects.

TABLE 5. Mean Stewart's disease ratings of 15 single crosses on 12 August (above diagonal) and specific combining ability and general combining ability effects (below diagonal) combined over cytoplasms

Parental line	B14A	B37	Mo17	N28	Va35	33-16	Line means
B14A	...	4.4[a]	4.3	4.6	5.0	4.4	4.5
B37	0.03	...	2.4	3.4	2.5	2.7	3.1
Mo17	0.08	0.08	...	2.9	2.9	2.2	2.9
N28	-0.40*[b]	0.18*	0.17*	...	4.0	3.2	3.6
Va35	0.20*	0.47*	0.11*	0.36*	...	2.7	3.4
33-16	0.10*	0.17*	-0.10*	0.04	0.20*	...	3.0
$\hat{g}_i$[c]	1.36*	-0.47*	-0.61*	0.25*	-0.04	-0.48*	...

[a]Least significant difference (LSD), ($P = 0.05$) = 0.59, denotes statistically significant differences among single cross means within or among rating dates.
[b]LSD ($P = 0.05$), ($\hat{s}_{ij}$) = 0.10 denotes statistical significance from 0 for any specific combining ability effect; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{ik}$) = 0.52 denotes statistical significance between specific combining ability effects among crosses with one parent in common; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{kl}$) = 0.42 denotes statistical significance between combining ability effects among crosses with no parent in common; LSD ($P = 0.05$), ($\hat{g}_i$) = 0.14 denotes statistical significance from 0 for any general combining ability effect; LSD ($P = 0.05$), ($\hat{g}_i - \hat{g}_j$) = 0.29 denotes statistical significance between general combining ability effects.
[c]$\hat{g}_i$ = general combining ability effects.

comparative ratings of the F_1s vs. corresponding F_2s combined over cytoplasms were highly significant—0.85, 0.93, and 0.78 on 19 July, 12 August, and 3 September, respectively. These results supported the diallel analysis findings that the inheritance of *E. stewartii* reaction was largely additive.

Environmental variance, genetic variance, and estimated number of genes involved in *E. stewartii* reaction are given in Table 8 for the six crosses involving resistant × susceptible inbreds. Generally, environmental variance was substantially higher than genetic variance and gene estimates were low at the first rating. At the second rating, environmental variance was generally reduced and genetic variance increased. Several genes were estimated to be segregating in crosses B14A × B37, B37 × N28, and Mo17 × N28 on 12 August, whereas nine, five, and six genes were estimated in the crosses B14A × Mo17, B14A × 33-16, and N28 × 33-16.

DISCUSSION

Although interpretation must be restricted to the series of crosses used, results of these experiments are encouraging and may be applicable to population improvement. Disease reaction was a highly heritable trait caused by predominantly additive effects. Conventional recurrent selection breeding programs should therefore be a useful means for developing maize populations with improved disease resistance.

The use of artificial inoculation before flowering enables the identification of some genotypes that are highly resistant to *E. stewartii*. Plants in this study were 7 wk old when inoculated; inoculation of 4–6-wk-old plants is more successful in identifying resistant types (M. H. Blanco, *unpublished*). With artificial inoculation, the most resistant plants could be identified before flowering and intermated, thereby eliminating a generation for each cycle of improvement. Because some genotypes did not become extensively damaged until the first rating date, a final rating after pollination is recommended for selected plants with high levels of resistance based on their prepollination rating date.

The experimental design used in this study proved a means to identify a line's genetic contribution to disease reaction in a series of diallel crosses at three periods during plant growth and development. Inbreds B37, 33-16, and Mo17 contributed genes for slow disease development and appeared to be excellent sources of resistance to *E. stewartii*. Mo17 was most effective in reducing the rate of disease increase; B37 and 33-16 were slightly less effective.

TABLE 6. Mean Stewart's disease ratings of 15 single crosses on 3 September (above diagonal) and specific combining ability and general combining ability effects (below diagonal) combined over cytoplasms

Parental line	B14A	B37	Mo17	N28	Va35	33-16	Line means
B14A	...	6.5[a]	6.5	6.6	6.3	6.8	6.5
B37	0.04	...	5.3	6.2	4.8	5.9	5.7
Mo17	0.03	-0.13*	...	5.7	5.9	5.2	5.7
N28	-0.19*[b]	0.34*	-0.09	...	6.0	5.7	6.0
Va35	-0.10*	-0.60*	0.50*	0.20*	...	5.5	5.7
33-16	0.22*	0.35*	-0.31*	-0.25*	0.01	...	5.8
$\hat{g}_i$[c]	0.79*	-0.22*	-0.26*	-0.11	-0.28*	-0.14*	...

[a]Least significant difference (LSD), ($P = 0.05$) = 0.59, denotes statistically significant differences among single cross means within or among rating dates.
[b]LSD ($P = 0.05$), ($\hat{s}_{ij}$) = 0.10 denotes statistical significance from 0 for any specific combining ability effect; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{ik}$) = 0.52 denotes statistical significance between specific combining ability effects among crosses with one parent in common; LSD ($P = 0.05$), ($\hat{s}_{ij} - \hat{s}_{kl}$) = 0.42 denotes statistical significance between combining ability effects among crosses with no parent in common; LSD ($P = 0.05$), ($\hat{g}_i$) = 0.14 denotes statistical significance from 0 for any general combining ability effect; LSD ($P = 0.05$), ($\hat{g}_i - \hat{g}_j$) = 0.29 denotes statistical significance between general combining ability effects.
[c]$\hat{g}_i$ = general combining ability effects.

TABLE 7. Mean Stewart's disease ratings and line means for the 15 single crosses in normal (above diagonal) and cms-T (below diagonal) cytoplasm on 3 September

Parental line	B14A	B37	Mo17	N28	Va35	33-16	Line means
B14A	...	6.4	6.7	6.7	6.4	6.6	6.6
B37	6.7	...	5.6	6.6	5.1	6.0	5.9
Mo17	6.4	5.1	...	5.8	6.6	5.8	6.1
N28	6.6	5.7	5.5	...	6.0	5.5	6.1
Va35	6.3	4.6	5.2	5.9	...	5.8	6.0
33-16	7.0	5.8	4.6	5.8	5.2	...	6.0
Line means[a]	6.6	5.6	5.4	5.9	5.4	5.7	...

[a]Least significant difference (LSD), ($P = 0.05$) = 0.70, denotes statistically significant differences for any single cross mean in normal cytoplasm and its counterpart in cms-T cytoplasm.

TABLE 8. Environmental and genetic variances and estimated gene numbers segregating for *Erwinia stewartii* reaction during the F_2 generation at two rating dates

	19 July			12 August		
F_2 cross	Environmental variance	Genetic variance	Estimated gene number[a]	Environmental variance	Genetic variance	Estimated gene number[a]
B14A × B37	0.50	0.37	0.16	0.24	0.69	1.74
B14A × Mo17	0.44	0.23	0.66	0.34	0.22	9.55
B14A × 33-16	0.61	0.30	2.04	0.24	0.22	5.82
B37 × N28	0.24	0.17	0.07	0.34	0.54	1.22
Mo17 × N28	0.25	0.11	0.56	0.64	0.66	2.06
N28 × 33-16	0.45	[b]	[b]	0.69	0.11	6.55

[a]Estimated gene numbers were calculated from the Castle-Wright formula (2).
[b]F_1 variance exceeded F_2 variance in this population.

Significantly negative gca effects for each of these inbreds at three rating dates indicated that inheritance of disease (lesion) development is predominantly due to additive gene action. One inbred (Va35) contributed outstanding resistance to several crosses late in the growing season, as determined by slow disease development between the second and third ratings. This resistance was due to significantly negative sca effects and, therefore, to predominantly nonadditive gene action. Whereas this type of resistance is needed in a breeding program, population improvement with a nonadditive type of inheritance may be difficult.

Although differences between cytoplasms were significant, the low magnitude of cytoplasmic effects indicated they were not an important source of variation. Most of the differences observed between cytoplasms among single crosses were not apparent until the last rating and were largely caused by natural senescence. Host reaction to *E. stewartii* was not substantially influenced by type of cytoplasm.

A major stumbling block in studying the inheritance of disease reaction is the indeterminate colonization of host tissue by *E. stewartii*. Since discrete Mendelian classes were not discernible, the 1–9 rating scale was adopted for convenience and reflects the extent of lesion development at a given time. This scale, being continuous in nature, could bias our interpretation if inheritance was indeed qualitative. The Castle-Wright formula (2), being based on genetic variance (of F_2 generations), supported the contention that reaction to *E. stewartii* is qualitative. The calculated gene estimates may be on the conservative side, however, since the number of genes would be underestimated if the formula's five assumptions are not fulfilled. These assumptions are: (i) independent genes each produced the same additive effect, (ii) there was no dominance, (iii) parents were homozygous, (iv) environmental differences combined additively with genetic ones, and (v) the number of individuals was large enough to make sampling errors negligible. Whereas these assumptions are never fulfilled entirely in genetic experiments, errors in the number of genes predicted depend on the magnitude of deviation from the assumptions.

An observation in the breeding nursery also supports the theory that few genes govern lesion development. This has been the rapid success in developing highly resistant genotypes in cyclic selection programs (M. S. Zuber, *unpublished*). We believe that several major genes reduce the rate of lesion development, with possible modifying genes also involved. Should a major gene(s) be identified for disease reaction, backcross breeding programs could be effective in incorporating resistance into elite inbred lines such as B14A and N28.

Diallel crosses provide a useful means for studying disease reaction and have recently received increased attention in corn disease research. Whereas diallel analysis has been used extensively to determine gene action at a single rating date, disease ratings made at several different times are useful for assessing the changing magnitude of genetic variance as disease develops. Information gained from such research may be helpful to corn breeders in the selection and development of lines with inherent capacity to reduce disease development—a concept often referred to as general resistance (6).

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