Characterization of the Chlorotic Lesion Response by Maize to *Cercospora zeae-maydis*

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ABSTRACT

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Previous research indicated that variation in symptom response to *Cercospora zeae-maydis* is associated with maize genotype. An association between lesion type at a specific time and overall symptom response has not been described. Inbreds and hybrids grown at several locations during 1989–1992 were artificially inoculated with *C. zeae-maydis*. Genotypes were evaluated for lesion type responses and percent area affected on ear leaves. These evaluations and determinations of secondary sporulation were performed in 1991 and 1992. Lesion size reduction, delay in disease progress, and inhibition of sporulation were associated with chlorotic lesions on resistant inbreds NC250A, NC288, and NC262A. Pa875 had fleck lesions that developed into necrotic, susceptible lesions by the end of the season, although lesions were few in number and restricted in size. Hybrids developed by crossing chlorotic lesion inbreds with nonchlorotic lesion inbred B73 displayed the chlorotic lesion response, indicating the response may be controlled by dominant allelic interaction.

Gray leaf spot (GLS), caused by *Cercospora zeae-maydis* Tehon & E.Y. Daniels (22), continues to be a problem in many maize (*Zea mays* L.) producing regions of the eastern United States (2,3,14). Disease incidence increased when maize was planted into the previous year's infested maize residue using minimum- or no-tillage residue management (2,3,7–9,13,14). Present control recommendations include crop rotation and use of hybrids with some degree of resistance.

Genetic resistance to *C. zeae-maydis* is a highly heritable, additive trait with a dominant allelic interaction (2,9,10, 13,23,24). Several quantitative trait loci with additive gene action are associated with resistance (4). Resistance is expressed as a reduction in the rate of disease increase compared to susceptible genotypes (2,10,13).

Lesion type influences disease progress and can be responsible for low disease severity ratings (2). Many susceptible inbreds display necrotic lesions (2,13,14, 19). Resistant inbreds display fleck-type lesions (2,14), and moderately resistant hybrids display chlorotic-type lesions (18) after infection by *C. zeae-maydis*.

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We observed that inbred Pa875, described as having a fleck reaction to *C. zeae-maydis* (2), also produces flecks constitutively. We also noted that typical GLS lesions developed from leaf flecks over time. Constitutive or genetic flecking also occurred on inbreds NC264, NC288, Va59, and Oh43; yet their lesion type responses to *C. zeae-maydis* infection differed.

We observed a chlorotic response to *C. zeae-maydis* infection distinct from the fleck and susceptible rectangular, necrotic lesion symptom responses. This chlorotic response developed on several inbreds. It has not been adequately described by previous researchers and may not be analogous to the chlorotic lesion response described by Hooker (12) for *Exserohilum turcicum*. A single dominant gene conditions chlorotic lesion response to *E. turcicum* in maize (12). Chlorotic lesion response to *Bipolaris maydis* in maize is inherited as two closely linked genes (6). Yet both chlorotic lesion responses exhibit a reduction in lesion size, a delay of necrosis, and inhibition of sporulation, in addition to chlorosis similar to the resistant response elicited by *C. zeae-maydis*.

The chlorotic lesion response to *C. zeae-maydis* is described in order to define its relationship with fleck and necrotic lesion responses, as well as to determine its relationship to degree of host resistance. Our objectives were to 1) characterize the chlorotic lesion response; 2) classify inbreds and hybrids for lesion type; 3) determine if dominant allelic interaction occurs for lesion type; and 4) assess the effect of chlorotic lesion response on percent leaf area affected (PLAA), lesion size, and sporulation within lesions.

MATERIALS AND METHODS

Genetic material. In 1989 and 1990, maize inbreds B73, NC250A, NC260, NC262A, NC264, NC270, NC288, NC290, Pa875, and Va59 were evaluated for reaction to *C. zeae-maydis*.

In 1991, inbreds B73, NC250A, NC262A, NC288, Oh43, Pa875, and Va59, and hybrids B37 × Oh43, NC250A × B73, NC250A × Oh43, NC262A × B73, NC262A × Oh43, NC288 × B73, NC288 × Oh43, Va59 × B73, Va59 × Pa875, Funks G4680, Pioneer Brand 3233, and Pioneer Brand 3569 were evaluated.

In 1992, inbreds B73, NC250A, NC288, and Pa875, and hybrids B73 × Pa875, NC250A × B73, and NC288 × B73 were evaluated.

Field plots. On 1 June 1989, seeds of inbreds were planted in a randomized complete-block design with three replicates near Warsaw, Ohio. Seeds were planted with a John Deere 71 flex planter fitted with ALMACO cone seeder units. Fluted coulters were mounted to the tool bar to cut the seed furrow. Experimental units were three-row plots, 3.5 m long with a between-row spacing of 0.76 m. Fifteen kernels were planted in each row, and plants were thinned to 12 per row to eliminate differences in disease spread within plots due to variable plant populations (8). Fertilizers and herbicides were applied according to standard recommendations (1).

In 1990, seeds of inbreds were planted in a randomized complete-block design with three replicates near Wooster, Ohio, on 24 April, as described for the 1989 plot.

In 1991, seeds of inbreds and hybrids were hand-planted in a randomized complete-block design with three replicates near Wooster on 18 May. All other planting procedures were as described for the 1989 plot.

In 1992, seeds of four inbreds and three hybrids were hand-planted in a randomized complete-block design with three replicates near Wooster on 3 June. The

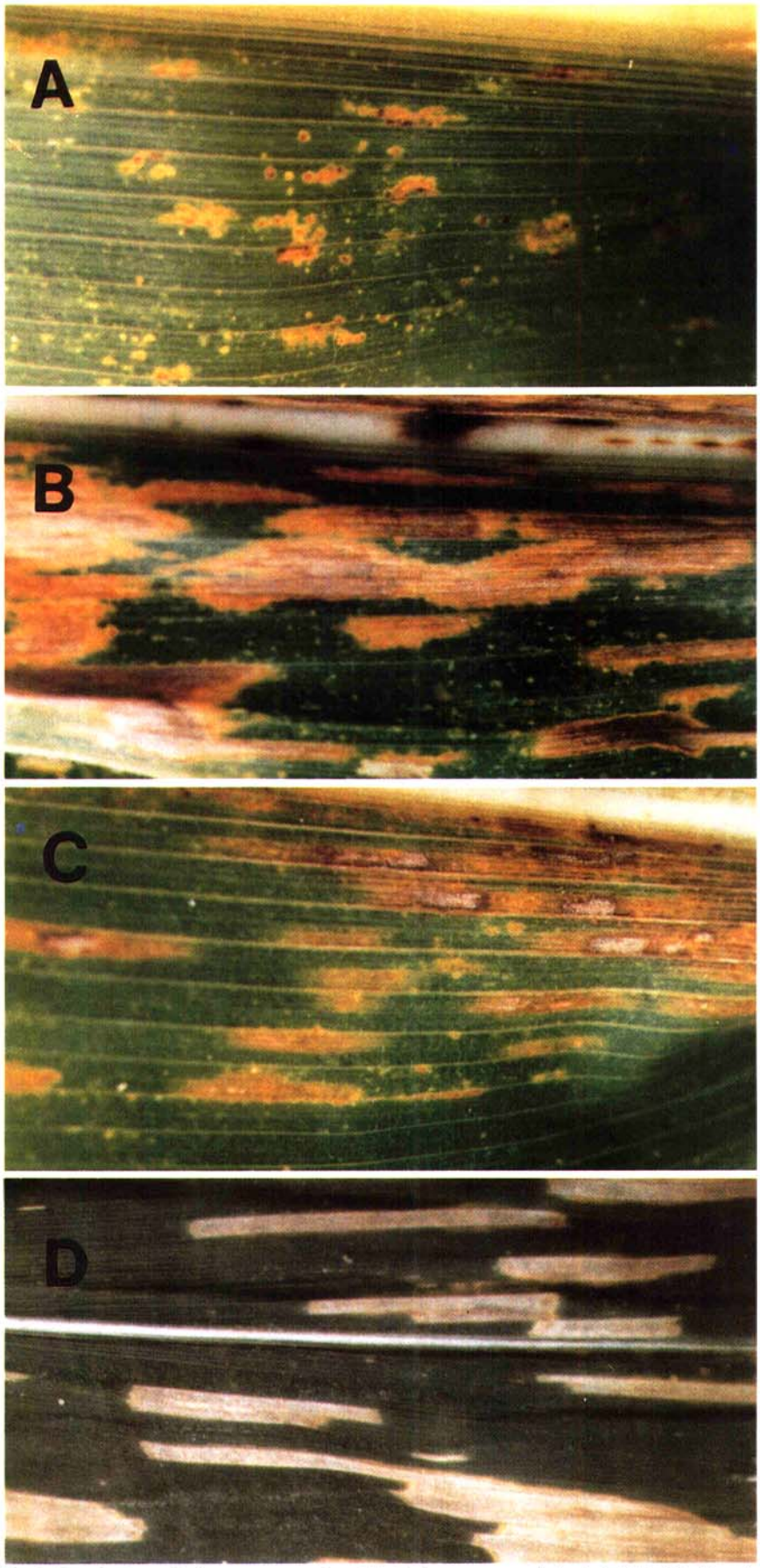

Fig. 1. Gray leaf spot lesion types on maize: **(A)** fleck response on inbred Pa875, **(B)** chlorotic lesions with orange borders on inbred NC262A, **(C)** chlorotic lesions with yellow borders on inbred NC250A, and **(D)** necrotic, rectangular, susceptible-type lesions on inbred B73.

same experiment was hand-planted at Columbus, Ohio, on 23 May. All other planting procedures for both experiments were as described for the 1989 plot.

Inoculum preparation. Portions of infested air-dried leaf tissue, collected the previous season, were placed under high relative humidity (≥90%) for 3 days to stimulate sporulation. Single conidia were picked from conidiophores with a sterile glass needle and placed on V8 juice agar in petri plates (14). Cultures were grown for 5 days at 28 C with 12 hr of darkness and 12 hr of fluorescent light (320 $\mu E/m^2/sec$). Conidia were spread over the agar surface by placing 1 ml of sterile distilled water on the colony and streaking the plate with a sterile glass rod. Cultures were maintained an additional 5 days as before. Agar cultures were transferred onto moistened autoclaved oat (*Avena sativa* L.) kernels in 2-L flasks and maintained as before for 10–12 days. Flasks were shaken every other day. Kernels colonized by the fungus were air-dried for 3–4 days at 22–24 C prior to use.

Inoculation. In 1989 and 1990, plants were inoculated by placing approximately 2 g of infested oat kernels into the whorl at the V10 growth stage (17).

In 1991 and 1992, plants were inoculated by placing approximately 2 g of infested oat kernels into the whorl at the V6, V8, and V10 growth stages (17).

In 1990, plots were irrigated overnight following inoculation using an overhead sprinkler system. In 1991 and 1992, a 1-hr irrigation delivering 0.3 cm of water was used at dusk for each of 14 days after inoculation when a rainfall event was not predicted.

Disease assessment. In 1989 and 1990, 10 plants from the middle row of each plot were selected and tagged for assessment of disease severity and lesion type reaction. The PLAA was estimated on the ear leaf approximately 60 days after flowering by using the GLS assessment scales developed by Smith (21).

In 1991, ear leaves of five plants in the center of the middle row in each plot were rated for PLAA as described for 1989–1990. Disease assessments were made weekly during a 5-wk period beginning approximately 45 days after inoculation. Three randomly selected lesions were marked on each of five plants per replicate with a nonphytotoxic marking pen, and lengths and widths were measured weekly for 4 wk beginning approximately 50 days after inoculation.

Sporulation was determined by excising and measuring four randomly selected intact lesions from the leaf above the ear on two plants from two replicates of each genotype on two sampling dates approximately 10 days apart. The excised lesions were placed on a wire mesh screen in a glass petri dish with a water-saturated filter paper in the bottom and maintained on a lab bench at 28 C with 12 hr of

darkness and 12 hr of fluorescent light (320 $\mu E/m^2/sec$). After 48 hr, the four lesions were placed in 10-ml test tubes containing 2.5 ml of distilled water and agitated for 15 sec using a bench-top vortex-type mixer to dislodge conidia. The conidial suspension was pipetted onto a hemacytometer, counted, and reported as the number of conidia $\times 10^4/mm^2$ of lesion area.

In 1992, five plants near the center of the middle row in each plot were assessed. Five randomly selected lesions were marked per plant, as before, 45 days after inoculation; and lengths and widths were measured 45, 57, 69, and 81 days after inoculation. Lesion types on the inbreds were also recorded on the same four dates. Percent ear leaf area affected was determined as described for the 1989–1990 experiments at 45, 55, 62, 72, and 80 days after inoculation. Sporulation within lesions was determined as described for the 1991 experiment.

Characterization of lesion response. In 1989–1990, inbreds were classified as restricted lesions with chlorosis (Cl), rectangular necrotic lesions (S), both types (Cl/S), or irregular, chlorotic flecks 1–3 mm in diameter (F).

In 1991–1992, inbreds and hybrids were classified as Cl, S, or F.

Data analysis. Apparent infection rates (*r*) were determined by calculating the slopes of regression lines representing increase in disease severity over time, using the exponential model (5). Area under the disease progress curve (AUDPC) (20) was calculated for PLAA and lesion size using the midpoint rule standardized by dividing the AUDPC by the number of days from the first to the last assessment for each observation (11). Differences in *r*, PLAA on the ear leaf, and AUDPC values were determined by analysis of variance (ANOVA). Mean separation was based on Fisher's least significant difference procedure (LSD) at the 5% level of probability.

RESULTS AND DISCUSSION

Lesion types. Inbred Pa875, which has a high level of resistance (2,9,10), produced the fleck-type (F) response (Fig. 1A) described previously (2,14). We also noted that irregular flecks developed on leaves of Pa875 irrespective of *C. zeae-maydis* inoculation; thus Pa875 presumably has constitutive chlorotic spots.

Lesions on inbreds that produced the chlorotic lesion response (Cl) began as chlorotic flecks that enlarged and eventually coalesced. Lesions on inbred NC262A were surrounded by distinctive bright orange borders (Fig. 1B), although leaves quickly produced gray, necrotic lesions. Orange borders surrounded lesions on NC288, while lesions on NC250A were encircled by yellow borders (Fig. 1C). The onset of necrosis within chlorotic lesions was either delayed or absent on inbreds NC250A and NC288.

Susceptible (S) lesions were characterized by the absence of chlorosis from infection to sporulation (Fig. 1D). Necrotic, rectangular susceptible lesions developed quickly on inbred B73.

Lesion types on some inbreds studied tended to change over time (Table 1). Lesions on Pa875 and NC288 initially were detected as flecks that later developed into chlorotic lesions. Eventually, some of the chlorotic lesions on Pa875 and NC250A appeared similar to susceptible lesions. This indicated that lesion types on certain genotypes may vary over time.

1989-1990 Inbred tests. Our initial evaluation of putative resistant inbreds indicated different resistance levels (Table 2). Inbred Pa875, which has been used as a resistant check in other studies (2,9,10,19,24), was also relatively disease free in our 1989–1990 study. It possesses quantitative resistance (2,13). This also has been termed rate-reducing resistance, where few lesions develop over the course of the epidemic (2). Pa875 typically produces zero or few lesions under conditions that favor moderated disease intensity (2,9,10,19,24). This type of resistance is mainly additive (13,23), although dominance may also be important (10). Typical necrotic lesion development is delayed, but it eventually occurs on Pa875, as shown by a 10.7% ear leaf area affected during 1990 (Table 2). Inbred Va59 produced the least amount of disease during the 2-yr study. Restricted, orange lesions developed in response to *C. zeae-maydis* infection.

Among other inbreds reported to be resistant to GLS, NC288 produced distinctive leaf flecking similar to Pa875, and lesions were restricted and chlorotic. The 3.2 mean percent ear leaf area affected for 1989–1990 was second lowest for the study. The inbreds NC262A, NC250A, and NC290 also delayed disease development and produced restricted chlorotic lesions. More lesions developed on inbreds NC260, NC264, and NC270 than on other inbreds in the test, although none were as susceptible as B73. These three inbreds exhibited susceptible lesion types as the epidemic proceeded.

1991 Inbred and hybrid test. NC250A, NC262A, and NC288 were selected for additional study because each exhibited chlorotic lesions. These inbreds were crossed to the susceptible inbred B73, which displayed necrotic lesions lacking chlorosis. Inbreds Pa875, Va59, and the hybrid Pioneer Brand 3233 were included because previous experience indicated that these genotypes had some degree of resistance to *C. zeae-maydis*. Inbreds B73 and Oh43, and hybrids B37 × Oh43, Funks G4680, and Pioneer Brand 3569 were included as susceptible checks.

The chlorotic lesion response was most prominent on NC250A, NC262A, NC288, Va59, NC250A × B73, NC250A × Oh43, NC288 × B73, and NC288 × Oh43 (Table 3). Since NC250A and NC288 inbreds and hybrids consistently

Table 1. Change in gray leaf spot lesion types[a] on inbreds tested at Columbus during 1992

	Days after inoculation[b]			
Entry	45	57	69	81
NC250A	Cl	Cl	Cl	Cl/S
NC288	F	F/Cl	Cl	Cl
Pa875	F	F	F/Cl	Cl/S
B73	S	S	S	S

[a] F = fleck, Cl = chlorotic, S = susceptible.
[b] Number represents days after plants were inoculated on 16 July 1992.

Table 2. Gray leaf spot severity and lesion type responses of maize inbreds to *Cercospora zeae-maydis* in 1989 and 1990 at Wooster, Ohio

	Percent leaf area affected[a]			
Entry	1989	1990	1989–1990 mean	Lesion type[b]
NC250A	1.0	19.0	10.0	Cl
NC290	0.1	16.7	8.4	Cl
NC288	0.0	6.3	3.2	Cl
NC262A	0.7	15.0	7.8	Cl
NC260	2.0	36.7	19.3	S
Pa875	0.0	10.7	5.3	F
Va59	0.0	4.4	2.2	Cl
NC270	0.7	40.0	20.3	Cl/S
NC264	0.8	33.3	17.1	Cl/S
B73	36.7	50.0	43.3	S
LSD ($\propto = 0.05$)	10.2	19.3	7.3	

[a] Mean percent ear leaf area affected based on assessment of 10 individual plants per replicate 60 days after flowering.
[b] Cl = chlorotic, F = fleck, S = susceptible.

displayed chlorotic lesion responses, this trait is most likely controlled by dominant allelic interaction. Additionally, the distinctive constitutive leaf flecking appeared on inbreds Pa875, NC288, Va59, and Oh43.

Inbred lines NC250A, NC288, and Pa875, and the hybrids NC250A × Oh43, NC288 × Oh43, and Va59 × Pa875 were most effective in inhibiting GLS development. Few, slowly enlarging, isolated lesions were produced on these genotypes. These genotypes also suppressed secondary sporulation compared to B73 (Table 3). Thus, the chlorotic response displayed by NC250A and NC288 was associated with restricted lesion enlargement and subsequent sporulation.

Chlorotic lesions were also present on inbred NC262A; yet lesion enlargement was not inhibited per se. Reduced lesion elongation did occur on hybrids NC262A × B73 and NC262A × Oh43. These results may be due to the influence of heterosis, which tends to reduce disease progress (16). NC262A × Oh43 also displayed low disease severity. This is probably due to Oh43's relative resistance to GLS (Table 3). At the other extreme, numerous, rapidly enlarging lesions producing abundant conidia appeared on the susceptible inbred B73. Also susceptible, but to a lesser extent, were hybrids Pioneer Brand 3569, Funks G4680, and Va59 × B73.

Hybrid Va59 × Pa875 and inbreds Pa875 and Va59 had very low *r* values. Few lesions were produced, and those that developed remained small (Table 3). Other researchers (2) indicated that inbred Pa875 appeared to have quantitative resistance to GLS. Thus, it is likely that inbred Pa875 and possibly inbred Va59 possess horizontal or rate-reducing resistance to GLS (2,15,25).

Table 3. Gray leaf spot severity and sporulation of *Cercospora zeae-maydis* on maize inbreds and hybrids in Ohio during 1991

Entry	*r*[a]	AUDPC				
		Percent leaf area affected[b]	Percent leaf area affected[c]	Lesion size (mm)[d]	No. conidia × 10^4/mm^2[e]	Lesion type[f]
NC250A	0.070	12.1	3.1	7.4	18.2	Cl
NC250A × B73	0.068	11.9	2.7	11.3	36.5	Cl
NC250A × Oh43	0.051	4.9	0.8	5.3	4.8	Cl
NC288	0.056	5.5	1.2	7.4	23.8	Cl
NC288 × B73	0.061	7.3	1.5	15.6	105.9	Cl
NC288 × Oh43	0.057	5.7	1.0	9.1	19.1	Cl
Pa875	0.042	2.5	0.5	3.2	11.7	F
Va59	0.046	6.9	1.3	17.2	56.2	Cl
Va59 × B73	0.049	5.9	1.1	15.0	193.5	S
Va59 × Pa875	0.041	2.7	0.3	5.0	4.6	Cl
NC262A	0.061	22.8	6.0	18.1	23.6	Cl
NC262A × B73	0.077	17.2	4.1	11.5	60.6	Cl
NC262A × Oh43	0.061	7.5	1.5	11.5	21.0	Cl
B73	0.105	51.4	12.9	19.1	216.3	S
Oh43	0.055	12.5	3.4	15.1	201.4	S
B37 × Oh43	0.050	6.3	1.33	12.9	23.3	S
Funks G4680	0.060	15.7	3.70	15.5	150.7	S
Pioneer Brand 3233	0.070	12.2	2.99	14.9	33.9	Cl
Pioneer Brand 3569	0.061	13.2	2.72	16.6	165.3	S
LSD ($\propto = 0.05$)	0.023	10.5	2.71	5.8	145.8	

[a] Apparent infection rate or slopes of regression lines representing increase in disease severity from 45 to 80 days after inoculation using the exponential model.
[b] Mean percent ear leaf area affected based on single late-season assessments 70 days after inoculation on five individual plants per replicate.
[c] Area under the disease progress curve based on percent ear leaf area affected from five assessments from 45 to 80 days after inoculation.
[d] Area under the disease progress curve based on lesion enlargement from 50 to 78 days after inoculation.
[e] Mean of two sample dates with two replicate samples of four lesions per date.
[f] Cl = chlorotic, F = fleck, S = susceptible.

Table 4. Gray leaf spot severity and sporulation of *Cercospora zeae-maydis* on maize inbreds and hybrids combined across Columbus and Wooster locations during 1992

Entry	AUDPC			
	Percent leaf area affected[a]	Lesion size (mm)[b]	No. conidia × 10^4/mm^2[c]	Lesion type[d]
NC250A	0.4	17.5	43.7	Cl
NC250A × B73	1.9	19.7	64.4	Cl
NC288	3.3	24.2	29.0	Cl
NC288 × B73	2.1	24.8	61.4	Cl
Pa875	2.4	18.6	40.2	F
B73 × Pa875	2.2	18.4	71.4	Cl
B73	15.4	38.7	157.4	S
LSD ($\propto = 0.05$)	2.7	6.6	49.9	

[a] Area under the disease progress curve based on percent ear leaf area affected assessed from 45 to 80 days after inoculation.
[b] Area under the disease progress curve based on lesion enlargement from 45 to 81 days after inoculation.
[c] Mean of two sample dates with two replicate samples of four lesions per date.
[d] F = fleck, Cl = chlorotic, S = susceptible.

1992 Inbred and hybrid test. Hybrids produced by crossing resistant and susceptible inbreds exhibited resistant lesions as well as low disease ratings (Table 4). Thus, as in 1991, the chlorotic lesion response appeared to be controlled by dominant allelic interaction.

Relative AUDPC values based on percent ear leaf area affected and increased lesion size were similar for all inbreds and hybrids except for susceptible inbred B73, which had significantly higher AUDPC values for both traits. B73 also produced a significantly higher number of conidia per square millimeter of lesion area than did the other genotypes.

The chlorotic lesion response displayed by NC250A and NC288 may reduce GLS progress by suppressing lesion number, restricting lesion size, and inhibiting secondary inoculum production similar to Pa875. Previous research indicated that GLS resistance exhibited by NC250A may be controlled by five or more genes (4). In this case, one or more specific alleles of this multigene complex may cause the chlorotic lesion response. The response may in turn influence one or more of the traits measured. We conclude that the chlorotic lesion response is not exclusively associated with the fleck response, nor is it exclusively associated with high levels of resistance, since in some inbred combinations (i.e., NC288 × B73) sporulation within chlorotic lesions can be relatively high (Table 3). Thus, germ plasm should be evaluated for percent ear leaf area affected, in addition to lesion types, to assess the degree of resistance expressed by the inbred or hybrid. Information on sporulation within lesions can be used to evaluate resistant germ plasm, but intrinsic variability associated with sporulation data is a problem.

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