

Resistance

Components of Resistance to Fusarium Ear Rot in Field Corn

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ABSTRACT

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Crosses involving various generations derived from two susceptible and two resistant inbred corn lines were inoculated with *Gibberella zeae* (isolate U5373) for three consecutive years. Analysis of variance of disease reactions in inbred and F₁ generations revealed differences among lines and blocks and a year × line interaction. Generation means analysis involving inbred, F₁, F₂, F₃, backcross, and selfed backcross generations implicated additivity (lack of dominance) as the predominant genetic effect. A

maternal influence was apparent in one set of reciprocal crosses. Seven inbred lines also were inoculated with seven *G. zeae* and four *Fusarium sporotrichioides* isolates in two blocks. *G. zeae* was generally more virulent than *F. sporotrichioides*. Inbred × isolate interactions were observed. Disease reactions of these inbred lines followed similar rankings regardless of the pathogen isolate tested.

Additional key words: *Fusarium graminearum*, Gibberella ear rot, quantitative inheritance, *Zea mays*.

Gibberella ear rot of corn caused by *Gibberella zeae* (Schwabe) Petch (anamorph: *Fusarium graminearum* Schwabe) is sometimes epidemic in the midwestern United States (20). The disease is a cause for concern, even when no significant yield loss occurs, because the causal fungus often produces deoxynivalanol, a cytotoxic trichothecene (21), and zearalenone, an estrogenic lactone (18). Other *Fusarium* species, such as *F. moniliforme*, *F. moniliforme* var. *subglutinans*, *F. culmorum* (14), and *F. sporotrichioides* Sherb. (= *F. tricinctum*) (8,14), cause similar ear rots with or without the presence of mycotoxins.

Some work has been done in determining the nature of resistance to ear rots caused by various *Fusarium* species, but variation in

experimental methods and pathogen species tested has limited the amount of useful information obtained. Early studies involved *F. moniliforme* isolates. Boling and Grogan (3) used generation means analysis of a susceptible × resistant cross and found year-to-year differences in genetic effects. There was also evidence of epistasis. Other workers found significant maternal effects when using diallel analysis of data on resistance to seedling blight caused by *F. moniliforme* (15). Variation in resistance to one or many *Fusarium* species among popular hybrids has also been found (2,6,16,17). Differences among isolates and isolate × hybrid interactions were apparent (2), but no large rank reversals among these interactions were evident. Host morphological components also have been implicated as factors influencing resistance (6,13).

Recent work has focused on *G. zeae*, primarily because of concern about mycotoxins. Inbred and F₁ analyses have been used to sort out significant genetic components. Cullen et al (5) found more resistance among F₁s than among the inbreds from which they were derived.

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In an earlier report, we found large differences in resistance among 58 inbreds (10). Diallel analysis among the 10 most resistant and susceptible inbreds revealed a significant amount of general combining ability but not specific combining ability. This work carries that analysis further, with a generation means analysis of some of those crosses. Environmental variation among inbred and F_1 generations over 3 yr also was analyzed. Last, isolates of *G. zeae* and *F. sporotrichioides* were compared for their relative ability to cause disease (virulence) on seven inbreds.

MATERIALS AND METHODS

Inoculations were made as previously described (10), using the toothpick method modified from Young (22). Only the uppermost ear of each plant was inoculated. Disease ratings were taken as previously described (10), during November of each year. Husks were removed, and disease was rated by amount of mycelium visible on the ear. The ratings were: 0 = no disease present, 0.1 = a few kernels around the inoculation point infected, 1 = 10% or less of the ear infected, 2 = 11–25%, 3 = 26–50%, 4 = 51–75%, and 5 = 76–100% of the ear infected.

For the genetic studies, various crosses of the inbreds B79, B73HT, A509, and Pa347 were inoculated in each of 3 yr (1982–1984). Inbreds B79 and B73HT were consistently susceptible and inbreds A509 and Pa347 were consistently resistant in previous work (10). A randomized complete block design with three replicates was used. Each plot was a single row of one cross, and 30–70 plants in each row were inoculated with *G. zeae*, isolate W8 (Penn State Univ. No. U5373, *F. graminearum* R6576). Inbreds and all F_1 combinations were inoculated in all 3 yr. The only reciprocals included were those of A509 × B73HT. In 1983 and 1984, F_1 backcrosses (F_1P_1 and F_1P_2) and F_2 s were also included. In 1984, selfed F_1 backcrosses ($F_1P_1S_1$ and $F_1P_2S_1$) and F_3 s were included when available. Significance of additive (a), dominance (d), and the three digenic epistatic (aa, ad, and dd) effects were determined on the weighted means by generation means analysis, using the method Hayman (11,12). A brief explanation of that method follows. Using the terminology of Gamble (7), the expectations of the means of two inbred lines and their descendants can be listed as follows (1,11,12):

$$\begin{aligned} P_1 &= m + a - 1/2d + aa - ad + 1/4dd \\ P_2 &= m - a - 1/2d + aa + ad + 1/4dd \\ F_1 &= m + 1/2d + 1/4dd \\ F_2 (=SF_1) &= m \\ P_1F_1 (=BC_1) &= m + 1/2a + 1/4aa \\ P_2F_1 (=BC_2) &= m - 1/2a + 1/4aa \\ F_3 (=SF_2) &= m - 1/4d + 1/16dd \\ S(P_1F_1) &= m + 1/2a - 1/4d + 1/4aa - 1/4ad + 1/16dd \\ S(P_2F_1) &= m - 1/2a - 1/4d + 1/4aa + 1/4ad + 1/16dd. \end{aligned}$$

Note that the mean (m) is a statistical midpoint, defined as the F_2 generation mean. The other generations are therefore defined in terms of the F_2 generation. Estimates of these genetic effects were derived from the means of the generations tested by solving the equations listed above for each effect. For example, if means are obtained for the first six generations listed, the various effects are estimated as follows (7):

$$\begin{aligned} m &= F_2 \\ a &= P_1F_1 - P_2F_1 \\ d &= -1/2P_1 - 1/2P_2 + F_1 - 4F_2 + 2P_1F_1 + 2P_2F_2 \\ aa &= -4F_2 + 2P_1F_1 + 2P_2F_2 \\ ad &= -1/2P_1 + 1/2P_2 + P_1F_1 - P_2F_1 \\ dd &= P_1 + P_2 + 2F_1 + 4F_2 - 4P_1F_1 - 4P_2F_1. \end{aligned}$$

The significance of each effect was tested by a two-tailed *t* test, where the variance for each effect is the variance of each generation summed as above. For example, the variance of aa would be 16 (var. F_2) + 4 (var. P_1F_1) + 4 (var. P_2F_1). Significance of interactions between each of the six genetic effects and the 2 yr (ym, ya, yd, yaa, yad, and ydd) also was determined by *t* test.

Analysis of variance (19) of the inbred and F_1 generations was used to determine environmental effects. The analysis included the reciprocal generation described above. Orthogonal comparisons (19) had been designed.

Seven inbreds were used to determine variability of resistance to several isolates of *G. zeae* and *F. sporotrichioides*. Three inbreds (B79, B73HT, and Mo17HT) were susceptible and four inbreds (A509, Ms74, Pa347, and NDI00) were resistant to *G. zeae* isolate U5373 in previous work (10). Each inbred was inoculated by the toothpick method with each of 11 pathogen isolates. A split-plot design (19) was used, and each plot contained one inbred inoculated with each isolate. Two blocks were used, and each block contained one row of 20–40 plants per inbred-isolate combination. Seven isolates (U5373, U5372, U5371, M3, S1, SA2, and VWA1) were *G. zeae*. The first four were from the culture collection of L. P. Hart; S1 was isolated from infected wheat obtained from R. Stuckey, University of Kentucky; and SA2 and VWA1 were from D. Cullen, University of Wisconsin. The remaining four isolates (T-340, F27, NRRL3299, and F38) were *F. sporotrichioides* (= *F. tritici* [8]). T-340 was obtained from E. B. Smalley, University of Wisconsin; NRRL3299 and F38 were from C. J. Mirocha, University of Minnesota; and F27 was from the culture collection of L. P. Hart.

RESULTS

Variation in *G. zeae* resistance. Analysis of variance of disease ratings of the inbred and F_1 generations for individual years and for the 3 yr combined are shown in Table 1. There were significant differences among these lines, implying genetic differences. The significant block effects in 1983 and 1984 suggested that variation in environmental conditions within a field also affected disease severity. Differences in overall disease severity from year to year were not significant (Table 1), although there were significant year × line interactions (Table 1).

The mean of the disease ratings used in the previous analysis (Table 1) is shown in Table 2. Although some year-to-year differences occurred within some lines, the disease severity of every F_1 fell between its two parents. There were no differences between the reciprocals of the cross B73HT × A509 (Table 2); therefore, no maternal effect was evident among the F_1 generation of this cross.

Generation means analysis. Table 3 shows the mean and variance of the disease rating for each generation and cross used in the

TABLE 1. Analyses of variance for disease ratings of *Gibberella zeae* ear rot on various corn lines^a

Source	df	MS	<i>F</i> ^b
1982			
Blocks	2	0.568	1.3 NS
Lines	10	5.133	11.9**
Error	20	0.432	...
Within plots	1,550	0.035	...
1983			
Blocks	2	4.804	12.9**
Lines	10	4.916	13.2**
Error	19	0.373	...
Within plots	673	0.104	...
1984			
Blocks	2	4.337	7.8**
Lines	10	3.094	5.5**
Error	20	0.559	...
Within plots	1,961	0.038	...
Three years combined			
Years	2	6.839	2.3 NS
Blocks in years	6	2.982	6.4**
Lines	10	8.944	4.1**
Year × line	20	2.199	4.7**
Error	59	0.467	...

^aOnly inbred and F_1 generations are included.

^bNS = not significant at $P \leq 0.05$; ** = significant at $P \leq 0.01$.

generation means analysis. There were large variances for some inbred and F_1 generations, which are genetically homogeneous determinations. Table 4 summarizes the results of the generation means analysis. The major genetic effect in the susceptible \times resistant crosses was the additive effect. No genetic effects were indicated in the two crosses of inbreds with the same disease reaction (B79 \times B73HT [both susceptible] and A509 \times Pa347 [both resistant]). There were interactions between years and genetic effects (Table 4) in only one of the seven crosses: the cross involving the two susceptible inbreds. Genetic differences were found between the two reciprocal crosses of B73HT \times A509. This indicates a possible maternal effect in disease reaction to *G. zeae* in generations later than the F_1 , because there were no differences between the F_1 reciprocals of this cross (Table 2).

An attempt was made to fit a relatively simple Mendelian gene model using the ratings obtained for the various crosses involving the most susceptible (B79) and most resistant (Pa347) inbreds. The ratings of the F_1 generation of that cross, as a percentage of the total, were 0 and 0.1 = 39.8%, 1 = 16.9%, 2 = 8.7%, 3 = 13.9%, 4 =

11.5%, and 5 = 7.9%. Because all plants in the F_1 generation were genetically identical, this variability must have been due to environmental factors. We concluded, therefore, that this large environmental influence made it impossible to fit these data to a simple gene model.

Inbred \times fungal isolate interactions. Partitioning of the variation among inbred lines showed that the significant differences were between the resistant and susceptible groups and among the susceptible lines (Table 5). B79 was more susceptible than Mo17HT and B73HT when ratings were averaged over all fungal isolates (Table 6), but there were no differences among the resistant inbreds (Table 5). Similarly, there were significant differences between isolates of *G. zeae* and *F. sporotrichioides* (Table 5), with the former being more virulent (Table 6). There also were differences in virulence among *G. zeae* isolates but not among *F. sporotrichioides* isolates. Although there were fungal isolate \times inbred interactions (Table 5), these did not involve major rank reversals, i.e., an inbred that was resistant to one isolate was never susceptible to another (Table 6). Therefore, the inbred rankings determined with isolate *G. zeae* U5373 were similar to other isolates.

The inbred \times isolate interaction involved differences among inbreds in their reactions to different isolates. For example, a significant interaction was evident when comparing the reactions of resistant lines with those of susceptible lines inoculated with isolates of *G. zeae*, but resistant and susceptible lines responded similarly when inoculated with isolates of *F. sporotrichioides* (Table 5). The only other significant inbred \times isolate interaction occurred when the virulence of the two species among the resistant inbreds was compared (Table 5). Among the resistant inbreds, therefore, the *G. zeae* isolates caused more disease than the *F. sporotrichioides* isolates (Table 6).

DISCUSSION

These data generally support the contention that environmental influences have an important effect on the reaction of corn to Fusarium ear rot. Temperature, moisture, and other factors have been previously implicated (6,13,14,17). The differences between blocks in 1983 and again in 1984 as well as the year \times line interaction among F_1 and inbred generations (Table 1) clearly implicate an environmental effect. The large error variances in Table 1 also indicate a substantial variation within rows. Other indications of an environmental influence are the large variances among inbred and F_1 generations (Table 3) and the inability to fit the cross B79 \times Pa347 to a Mendelian model.

TABLE 2. Mean disease ratings for inbred and F_1 generations of corn inoculated in the developing ear with *Gibberella zeae* isolate U5373^w

Line ^x	Mean disease rating ^{y,z}			
	All three years	1982	1983	1984
Pa347	0.3 a	0.5 ab	0.2 a	0.3 a
A509 \times Pa347	0.7 ab	0.1 a	0.4 a	1.5 ab
A509	1.5 abc	0.5 ab	1.2 ab	2.7 bc
Pa347 \times B73HT	1.6 abc	0.9 ab	1.8 bcd	2.0 bc
B79 \times Pa347	1.7 abcd	0.8 ab	2.4 cd	1.9 bc
B73HT(F) \times A509	1.9 bcd	1.7 bc	1.8 bcd	2.2 bc
A509(F) \times B73HT	2.1 bcde	1.3 abc	2.1 bcd	3.0 cd
A509 \times B79	2.8 cde	1.6 bc	3.7 ef	3.0 cd
B73HT	2.9 cde	2.2 c	2.8 de	3.2 cd
B79 \times B73HT	3.2 de	2.3 c	4.5 f	2.7 bc
B79	3.6 e	4.9 d	1.5 bc	4.2 d

^wThe lines are ranked from most resistant to most susceptible over 3 yr.

^xInbreds B79 and B73HT are susceptible and inbreds A509 and Pa347 are resistant to ear rot.

^yDisease ratings: 0 = no disease present; 0.1 = a few infected kernels around the inoculation point; 1 = 10% or less of the ear infected; and 2 = 11–25%, 3 = 26–50%, 4 = 51–75%, and 5 = 76–100% of the ear infected.

^zMeans followed by the same letter within a column are not significantly different at $P \leq 0.05$ according to Duncan's multiple range test (17).

TABLE 3. Means and variances of disease ratings for various generations of corn crosses^a

Generation ^b	Year	Disease ratings of crosses ^c													
		B79 \times Pa347		A509 \times B79		B73(F) \times A509 ^d		B73(M) \times A509		B73 \times Pa347		B79 \times B73		A509 \times Pa347	
		Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance	Mean	Variance
P ₁	1983	1.6	3.1	1.0	2.6	3.5	2.7	3.5	2.7	3.5	2.7	1.6	3.1	1.0	2.6
	1984	4.1	2.7	2.7	3.2	3.2	4.0	3.2	4.0	4.1	2.7	2.8	3.2
P ₂	1983	0.2	0.2	1.6	3.1	1.0	2.6	1.0	2.6	0.2	0.2	3.5	2.7	0.2	0.2
	1984	0.3	0.3	4.1	2.7	2.8	3.2	0.3	0.3	3.2	4.0	0.3	0.3
F ₁	1983	2.3	3.3	3.6	2.1	2.2	3.5	1.2	3.4	1.8	2.5	4.4	1.1	0.4	1.2
	1984	1.9	3.5	3.0	2.7	2.6	2.4	2.1	4.0	2.7	3.9	1.5	1.9
F ₂	1983	1.6	2.7	2.1	3.6	2.2	3.5	2.2	3.5	1.6	3.2	4.3	1.4	0.5	0.8
	1984	1.5	2.4	1.8	3.3	1.2	2.8	1.2	2.4	3.2	3.9	1.8	2.8
P ₁ F ₁	1983	3.4	2.9	2.1	3.2	2.4	3.9	2.8	2.5	3.0	2.5	4.5	1.4	0.5	1.3
	1984	3.2	2.9	2.2	3.7	2.8	1.8	3.7	3.5	1.7	3.2
P ₂ F ₁	1983	0.5	1.0	3.9	2.3	1.0	2.4	1.0	2.5	0.5	0.7	4.4	1.5	0.4	0.7
	1984	1.6	3.2	3.2	2.5	2.8	3.5	1.4	2.5	3.0	3.3	1.5	2.6
F ₃	1984	1.8	2.7	3.1	3.6	2.0	3.0	1.0	1.8	3.2	4.8	2.2	2.6
P ₁ F ₁ S ₁	1984	3.0	3.2	2.7	3.3	2.5	2.9	2.2	4.9	3.6	3.6	1.5	2.5
P ₂ F ₁ S ₁	1984	0.8	2.2	3.1	3.5	2.9	3.5	0.8	0.9	3.1	4.2	2.1	2.5

^aIn each year, three replicates (rows) of 30–70 plants per row were ear-inoculated with a toothpick infested with *Gibberella zeae* U5373. Plants were rated after the first frost.

^bP₁ = first parent listed and P₂ = second parent listed; P₁F₁ = F₁ backcrossed to P₁, P₂F₁ = F₁ backcrossed to P₂, P₁F₁S₁ = P₁F₁ selfed, and P₂F₁S₁ = P₂F₁ selfed.

^cDisease ratings: 0 = no disease present; 0.1 = a few kernels around the inoculation point infected; 1 = 10% or less of the ear infected; 2 = 11–25%, 3 = 26–50%, 4 = 51–75%, and 5 = 76–100% of the ear infected; ... = no data.

^dIn 1984, this included both reciprocals B73(F) \times A509 and B73(M) \times A509.

TABLE 4. Significant genetic effects determined by generation means analysis^y

Cross	Significant effects ^z
B79 × Pa347	m a
A509 × B79	m a
B73Ht(M) × A509 (1983)	m a d
B73Ht(F) × A509 (1983)	m a aa ad
B73Ht × A509 (1984)	m aa ad
B73Ht × Pa347	m a
B79 × B73Ht	m y ya
A509 × Pa347	m

^y Means and variances listed in Table 3 were analyzed for six genetic effects by the method of Hayman (11,12). These effects were mean (m), additive (a), dominant (d), and the three epistatic effects (aa, ad, dd). Differences between the two years (y) and the interaction between years and genetic effects (ya, yd, etc.) were also analyzed. The reciprocal crosses of B73Ht × A509 were analyzed separately in 1983 but together in 1984; therefore, no determination of year effects were made for these crosses.

^z Significant at $P \leq 0.05$ according to Student's *t* test.

In spite of the environmental influence, significant genetic differences were consistent from year to year among the inbred and F₁ generations (Table 2). As previously noted, all F₁s analyzed were rated intermediate to the inbred parents in disease reaction. This lack of overdominance (because the hybrid disease ratings were not outside the range defined by the inbreds) is contrary to the findings of Cullen et al (5), although they used different lines.

The generation means analysis indicated additivity was the predominant genetic effect, occurring in five of the eight analyses conducted (Table 4). Other effects (aside from the mean effect) were sometimes significant but not as often as additivity. This implies that most segregating genes for resistance exhibited little dominance or digenic epistasis (interaction between two loci). One of the deficiencies of generation means analysis is an inability to identify when opposing effects cancel each other (9,11,12). Therefore, dominant genes for resistance and susceptibility could occur within a particular cross and would not be evident in the analysis. This possibility is minimized, however, when each inbred is analyzed in more than one cross (9), as was done here. The mean, which is the statistical midpoint defined as the mean of the F₂ generation, was significant in all cases. This implies that the mean F₁ rating is different from zero. There were no significant genetic effects between the two crosses of inbreds with the same disease reaction (B79 × B73HT and A509 × Pa347). This suggests few genetic differences existed between these comparably rated inbred lines. However, because the environmental differences were greater than the segregating genetic differences, the ability to detect such differences is poor. The predominance of additivity in resistance to *G. zeae* should make incorporation of resistance into agronomically useful corn lines practical, because genes for a susceptible or resistant disease reaction would not be masked by other dominant or epistatic alleles (9).

Although there were significant differences in generation means effects between the two reciprocals of the cross B73Ht × A509 (Table 4), there were no differences between the two F₁ reciprocals (Table 2). Therefore the genetic differences must have been evident in later generations, even though the differences between various crosses involving these reciprocals were generally not great (Tables 2 and 3).

The study of inbred × fungal isolate effects (Tables 5 and 6) clearly show that *F. sporotrichioides* (= *F. tricinctum*) isolates were less virulent than those of *G. zeae*. This has been reported previously (14). However, *F. sporotrichioides* was capable of causing significant disease on the highly susceptible inbred B79 (Table 6).

There were no major rank reversals in susceptibility of inbreds among different *Fusarium* isolates tested (Table 6). Because each inbred was ranked similarly with each isolate, resistance incorporated into an inbred is likely to be stable (4).

In spite of the environmental influence, the large differences in resistance among inbreds and their crosses are readily evident. The

TABLE 5. Analysis of variance for corn inbred × fungal pathogen isolate effects in 1984^a

Source	df	MS	F ^b
Block	1	0.03	0.0 NS
Inbred	6	19.80	38.6**
R vs. S	1	82.77	162.3**
Among R	3	1.11	2.2 NS
Among S	2	15.52	30.4**
Error a	6	0.51	...
Isolate	10	12.77	39.2**
Z vs. F	1	93.66	283.8**
Among Z	6	6.05	18.3**
Among F	3	0.03	0.1 NS
Inbred × isolate	60	0.79	2.4**
(R vs. S) × (Z vs. F)	1	6.67	20.2**
(R vs. S) × (among Z)	6	3.25	9.8**
(R vs. S) × (among F)	3	0.17	0.5 NS
(Z vs. F) × (among S)	2	0.95	2.9 NS
(among Z) × (among S)	12	0.55	1.7 NS
(among F) × (among S)	6	0.10	0.3 NS
(Z vs. F) × (among R)	3	1.22	3.7*
(among Z) × (among R)	18	0.47	1.4 NS
(among F) × (among R)	9	0.02	0.1 NS
Error b	70	0.34	
Within plot	3,804	0.16	

^a Seven inbreds were inoculated with each of 11 pathogen isolates. Three inbreds were susceptible (S) and four were resistant (R) to pathogen isolate U5373. Seven isolates were *Gibberella zeae* (Z) and four were *Fusarium sporotrichioides* (F). Each inbred-isolate combination was represented as one row in each of two blocks.

^b NS = not significant at $P \leq 0.05$, * = significant at $P \leq 0.05$, and ** = significant at $P \leq 0.01$.

TABLE 6. Average disease ratings of corn inbred × fungal pathogen isolate inoculations ranked by disease rating

Fungus	Susceptible inbreds			Resistant inbreds				Mean ^z
	B79	Mol17Ht	B73Ht	Ms74	ND100	A509	Pa347	
<i>Gibberella zeae</i>								
U5372	4.40	3.55	3.45	2.95	2.10	1.90	1.40	3.06 a
S1	4.20	3.75	3.65	2.45	2.30	1.75	1.00	2.79 a
U5373	4.75	4.05	4.55	1.25	0.60	0.55	0.25	2.42 a
SA2	4.50	2.60	3.05	1.65	1.45	1.75	1.45	2.41 a
VWA1	4.05	2.00	2.40	2.60	2.50	1.95	0.60	2.39 a
U5371	3.65	3.55	2.70	2.45	0.75	2.40	1.70	2.18 a
M3	3.00	0.30	0.80	0.40	0.45	0.40	0.55	1.05 b
<i>Fusarium sporotrichioides</i>								
F38	2.30	0.25	0.55	0.00	0.40	0.25	0.25	1.08 b
T-340	2.85	0.80	0.45	0.00	0.20	0.30	0.30	0.79 b
NRRL3299	2.50	0.85	0.35	0.10	0.35	0.20	0.30	0.78 b
F27	2.10	0.45	0.50	0.20	0.50	0.20	0.25	0.62 b
Mean	3.52 a	2.07 b	2.02 b	1.33 bc	1.07 bc	1.04 bc	0.71 c	1.67

^z Means of inbreds or isolates followed by the same letter are not different at $P \leq 0.05$ according to Duncan's multiple range test.

large additivity component as well as the apparent "stable" reaction among the lines tested indicate that long lasting resistance to *G. zeae* can be readily incorporated into agronomically useful lines.

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