Effect of Corn Genotypes on Ear Rot Infection by *Gibberella zeae*

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ABSTRACT

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In a disease screening of 58 inbred corn lines, 10 lines with varying degrees of susceptibility to *Gibberella zeae* ear rot were selected for diallel analysis. The parental inbreds were rated resistant (PA347, MS74, and A509), susceptible (A669, ND100, and A637), and highly susceptible (B73HT, Mo17HT, B79, and A670). The F_1 progeny of all possible crosses between the 10 inbred lines were inoculated through the husks with toothpicks infested with *G. zeae* isolate W-8 and rated for percentage of rotted kernels on the ears. Differences among genotypes in their reaction to inoculation by *G. zeae* were significant ($P = 0.05$). General combining ability (GCA) effects were significant ($P = 0.01$) but specific combining ability (SCA) effects were not. Therefore, the reaction of single-cross hybrids to infection by *G. zeae* could be predicted on the basis of the parental inbred reaction to *G. zeae*. Analysis of infected grain from the inbred lines indicated a positive but low correlation ($r = 0.47$) between the concentration of the mycotoxin deoxynivalenol and disease ratings. The concentration of deoxynivalenol was low at lower disease ratings but varied considerably at higher disease ratings. Zearalenone concentrations varied and no trends were observed.

Additional key words: *Fusarium graminearum*, vomitoxin

Gibberella ear rot of corn is caused by *Gibberella zeae* (Schwabe) Petch, the perfect stage of *Fusarium graminearum* (Schw.). Gibberella ear rot occurs each year in most geographic areas, but it was epidemic in the midwest in 1965 and 1972 (10). Two mycotoxins, zearalenone and deoxynivalenol (vomitoxin), are commonly associated with Gibberella ear rot. Zearalenone is responsible for estrogenic mycotoxicoses in animals; symptoms include enlargement of the uteri and mammary glands, vulvar swelling, testical atrophy, and vaginal prolapse (6,7). Deoxynivalenol is a cytotoxic trichothecene responsible for emesis and feed refusal in swine (11,12).

Shannon et al (8) determined the effect of corn genotypes on zearalenone production in fermentation studies. There were significant differences among the inbred lines tested, but interactions between cultivars and pathogen isolates were not significant. Cullen et al (2) reported that inbred lines were more susceptible than hybrids and that isolates of *G. zeae* differed in virulence and toxin production. They tested four inbreds and two single-cross hybrids and found that production of zearalenone was positively correlated with increasing disease severity. Hart et al (5) reported that deoxynivalenol production increased as disease severity increased and that deoxynivalenol and zearalenone levels varied with isolates of *G. zeae*. These reports suggest that mold development in inoculated ears could be used to screen for resistance and that deoxynivalenol and/or zearalenone production would presumably be lower in the more resistant lines. This paper reports the results of testing inbred parents and the F_1 generation of single-cross hybrids for resistance to *G. zeae*.

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MATERIALS AND METHODS

A preliminary disease screening was made of 58 inbred lines planted in single rows, 20 plants per row. Ten ears of each inbred were inoculated by inserting toothpicks infested with *G. zeae* through the husks into the center of the ear. Preparation of the toothpick inoculum has been described previously (5). Inoculations were made within 2 days after the silks turned brown. All inoculations were made with *G. zeae* isolate W-8, which was selected after comparing three isolates of *G. zeae* for pathogenicity and toxin production in sweet corn (5). Isolate W-8 was intermediate in virulence but was the only isolate of the three to produce zearalenone. Control ears were inoculated with uninfested toothpicks.

Disease ratings based on the percentage of the ear rotted were made after the first killing frost. The ratings were: 0 = no disease, 0.1 = only a few infected kernels around the inoculation point, 1 = 10% or less of the ear infected, 2 = 11–25%, 3 = 26–50%, 4 = 51–75%, and 5 = 76–100% of the kernels rotted. Deoxynivalenol and zearalenone were extracted from infected grain and quantified by procedures reported previously (5). Concentrations of both toxins were determined on thin-layer chromatography (TLC) plates by comparison with standards of known concentration. Infected grain from within each inbred line was pooled and a single 50-g subsample analyzed for zearalenone and deoxynivalenol.

Based on the results of the initial screening, inbred lines with disease ratings of 0.1 or less, and 5, were selected for diallel analysis. These lines were PA347, MS74, A509, A669, ND100, A637, B73HT, Mo17HT, B79, and A670. The diallel analysis consisted of inbreds crossed in all possible combinations, reciprocals not included. Parents and crosses were tested at a single location in a completely randomized block design with three replicates. Each plot consisted of a single row of 100 plants. Ears of 50–60 plants in each row were inoculated by inserting toothpicks infested with isolate W-8 as described. Control ears were inoculated with uninfested toothpicks. Disease ratings of each inoculated ear were made as described previously.

Analysis of the data was based on method 2, model I, as proposed by Griffing (4). The analysis is based on a fixed model and assumes that genotypic effects are constants. The analysis was of the parents and one set of F_1s.

RESULTS AND DISCUSSION

Results of the preliminary screening in 1981 are shown in Table 1. Most inbred lines were moderately to highly susceptible. There was a high degree of variation in the disease ratings of individual ears within an inbred line, except for the resistant and most susceptible lines. An inbred with a rating greater than or equal to 1 probably is not acceptable as a potential source of resistance because of the wide variation in susceptibility to ear rot among individual plants. Infected grain from the inbred lines in the preliminary screening was extracted and zearalenone and deoxynivalenol quantified by TLC (Table 1). The correlation between deoxynivalenol concentration and disease rating was significant (F = 15.98 with df = 1,56) but low (r = 0.47). Inbreds with disease ratings of less than 1 consistently had low concentrations of deoxynivalenol, but at higher levels of infection, the inbreds varied in the amount of toxin present. Zearalenone concentrations varied considerably and no trends were observed. Results of toxin production in the F_1 progeny are not presented, but as indicated by Cullen et al (2), the overall toxin level should decrease as the amount of ear rot decreases.

Results of the inoculations of the F_1 generation, and the inbred parents in 1982, are presented in Table 2. Disease ratings of the parents differed from the preliminary screen tests except for three lines, PA347, MS74, and A509, which were rated less than 1 or resistant. Rated susceptible were A669, ND100, and A637, which had disease ratings between 1 and 2. Highly susceptible lines, B73HT, Mo17HT, B79, and A670, were all rated greater than 2. Differences in disease ratings of the parental lines between 1981 and 1982 probably reflect the greater number of ears inoculated in 1982.

The analysis of variance for differences among genotypes was significant (P = 0.05, F = 3.45, df = 54,103), and differences among replicates were not significant. The general combining ability (GCA) is a function of additive gene action and average dominance and is used to indicate the average performance of a line in hybrid combinations (9). Variation among GCA effects was highly significant (P = 0.01, F = 192.8, df = 9,405). The GCA of each inbred line is included in Table 2. The specific combining ability (SCA) is used to designate cases in which certain combinations do better or worse than would be expected on the basis of the average performance of the lines involved (9). Although SCA effects were not significant, when the resistant inbred PA347 was one parent all F_1 crosses were resistant, regardless of the disease reaction of the other parent. This indicates that PA347 possesses more dominant alleles than the other resistant inbreds.

Generally, the disease ratings of all F_1s were intermediate between those of the parents. The disease ratings of all F_1s with PA347, MS74, and A509 as one parent and a resistant or susceptible inbred as the other parent were less than 1. However, only PA347 appeared to possess the genetic background to overcome the susceptibility in B79, Mo17HT, and B73HT. The GCA of −0.8 for PA347 also indicates a higher combining ability for resistance. In several years of testing, B79 has consistently been the most susceptible inbred, with average ratings approaching

Table 1. Results of preliminary inoculation tests with 58 inbred lines of corn in 1981[a]

Inbred	Disease rating[b]	RDM[c]	Deoxynivalenol conc.[d] (μg/g)	*Zearalenone conc.*[d] (μg/g)
GLH-552 hybrid	0.7	···	18	0
GLH-5922 hybrid	3.4	···	147	0
ND100	0.04[e]	80	8	0
ND246	3.8	80	78	8
ND301	0.8	80	39	0
A509	0.04[e]	85	26	5
A554HT	1.5	85	7	0
A668	0.6	85	0	0
A641	1.3	85	4	0
CM105	0.6	85	64	6
MS74	0.0[e]	85	0	0
PA347	0.0[e]	85	3	0
PA351	0.3	85	13	6
SD10	1.3	85	73	4
MS93	1.6	90	0	0
MS116	2.8	90	111	0
MS153	3.4	90	303	19
MS1334	1.0	90	298	37
W182B	0.8	90	0	3
W182BN	0.9	90	71	0
W117	3.0	93	4	2
W153R	2.7	95	5	0
A637	0.0[e]	97	0	0
A638HT	2.9	100	181	9
A666	0.6	100	18	4
MS75	1.9	100	74	0
A554	1.3	105	0	0
A635HT	3.4	105	362	0
A639	3.0	105	86	5
W64A	0.7	105	4	0
A654	3.7	105	247	0
A661	2.5	105	43	0
A667	2.4	105	107	0
W454	1.1	105	259	74
FR634	3.4	108	252	0
A619HT	1.0	110	237	0
A632HT	2.4	110	261	0
A658	5.0	110	303	0
A660	0.8	110	0	0
A665	1.0	110	49	0
A669	0.08[e]	110	2	1
A670	0.1[e]	110	58	0
MS76	2.1	110	147	2
W452	1.9	110	223	22
A634HT	1.9	111	58	0
R-OH43 HTB	4.2	112	182	0
OH561	1.8	113	37	0
VA26	1.6	113	18	55
B85	2.5	115	74	18
OH545	2.7	115	8	19
OH562	1.4	115	73	7
OH563	1.0	115	76	10
B73HT	5.0[e]	116	367	0
B77	4.2	118	9	0
B78	3.9	118	400	8
Mo17HT	5.0[e]	118	34	0
H93	1.8	121	335	0
B79	5.0[e]	120	1,922	0

[a] Toothpicks infested with *Gibberella zeae* isolate W-8 were inserted into the center of the ear through the husks within 2 days after silk browning and evaluated after the first killing frost.
[b] Ratings are on a scale of 0–5, where 0 = no disease and 5 = >76% of the ear moldy. Each value is the average of 10 ears.
[c] Relative days to maturity.
[d] Toxins were extracted from infected grain. Concentrations were determined by thin-layer chromatography by comparison with standards of known concentrations.
[e] Lines selected for diallel analysis.

Table 2. Disease ratings of parental inbred lines and F_1 single-cross hybrids inoculated in 1982 with *Gibberella zeae*[a]

Parent	PA347	ND100	A670	MS74	A637	A509	A669	B79	Mo17HT	B73HT	GCA[b]	Mean
PA347	*0.5*	0.2	1.0	0.1	0.9	0.2	0.4	0.8	0.7	0.9	−0.80	0.6
ND100	0.2	*1.4*	0.9	0.6	1.5[c]	0.6	2.2	2.5	2.6	1.7	−0.03	1.4
A670	1.0	0.9	*2.3*	0.9	1.5	0.5	0.8	2.0	1.8[c]	1.5[c]	−0.03	1.3
MS74	0.1	0.6	0.9	*0.8*	0.9	0.3	1.6	2.2	1.7	2.2	−0.32	1.1
A637	0.9	1.5[c]	1.4	0.9	*1.3*	0.3	1.6	2.4	2.0	1.7[c]	−0.04	1.4
A509	0.2	0.6	0.5	0.3	0.3	*0.5*	1.8	1.6	2.0	1.5	−0.53	0.9
A669	0.4	2.2	0.8	1.6	1.6	1.8	*1.1*	2.2[c]	2.1	1.1	−0.01	1.4
B79	0.8	2.5	2.0	2.2	2.4	1.6	2.2[c]	*4.8*	1.6	2.3	+0.94	2.2
Mo17HT	0.7	2.6	1.8[c]	1.7	2.0	2.0	2.1	1.6	*3.1*	1.7	+0.54	1.9
B73HT	0.9	1.7	1.5[c]	2.2	1.7[c]	1.5	1.1	2.3	1.7	*2.2*	+0.37	1.8
										LSD 0.05	0.07	0.4
										LSD 0.01	0.10	0.6

[a] Averages of 50–60 ears for each of three replicates. Disease ratings are based on a scale of 0–5, where 0 = no disease and 5 = >76% of the ear moldy.
[b] GCA = general combining ability for each parent line.
[c] These averages were not available and were estimated as described by Eckhardt (3).

5 (*unpublished*).

Parental lines with disease ratings of less than 1 all had 85 days to relative maturity, whereas all other parents except ND100 matured in 97 or more days. The three lines with the highest GCA for susceptibility, B79, B73HT, and Mo17HT, had relative days to maturity of 120, 118, and 116, respectively. A regression analysis of disease ratings vs. days to maturity gave an F ratio of 27.13 (df = 1,26) with an $r^2 = 0.51$. Although it appears that resistance is associated with early maturity, only about 50% of the variation in disease ratings can be accounted for by days to maturity. Assuming that days to maturity of the hybrids can be approximated by averaging the days to maturity of the parents, PA347 × B79, MS74 × B79, and A509 × B79 would each have about 102 days to maturity. The average disease ratings were 0.8, 2.2, and 1.6, respectively, indicating that ear rot resistance and early maturity probably are not linked.

The diallel crosses were limited to an analysis of GCA and SCA mean squares and effects because of the fixed-model experimental design (1). Because the GCA was significant and the SCA was not, the reaction of single-cross hybrids to infection by *G. zeae* could be predicted on the basis of the parental inbred reaction to *G. zeae*. This means a breeding population could comprise a population with a higher gene frequency for resistance and that reduced testing of hybrid selections would be necessary.

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