Genotypic Differences in Maize to Kernel Infection by *Fusarium moniliforme*

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ABSTRACT

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Maize kernels produced by 84 commercial hybrids, six inbreds, and the 15 possible crosses among these inbreds were assayed for percentage of asymptomatic kernels infected by *Fusarium moniliforme*. Infection levels among commercial hybrids ranged from 5–89%. Consistently low or high infection was associated with some hybrids in all 3 yr of testing. Infection of inbreds and of their hybrids were 19–79 and 5–60%, respectively. Crosses between two resistant (R) parents had 11% average infection compared with 55% for crosses between two susceptible (S) parents. R × S crosses averaged 33% infection. The results demonstrate that maize genotypes differ significantly in percentage of healthy-appearing kernels that harbor *F. moniliforme* and that these differences are under genetic control.

Fusarium moniliforme Sheld. infects a wide range of crops throughout the world, particularly in tropical and subtropical regions (1). In maize (*Zea mays* L.), it causes seedling blight and root, stalk, and ear rots (2,5,15,20) and it is the fungus most frequently isolated from maize kernels (6,11–13,19). Although kernel rot is easily observed, infected kernels frequently appear undamaged (7,9,17,18).

The significance of *F. moniliforme* in healthy-appearing kernels is not fully understood. Planting infected seed may increase the incidence of seedling blight (5) and contribute to systemic infection of plants (4). The importance of infected seed as a source of subsequent plant infection, however, seems minimal because seedling plants are readily infected by *F. moniliforme* from infested soil debris (10). *F. moniliforme* produces a toxin in vitro that has a deleterious effect on both plants (3,14,16) and animals (3,12,16). Grain infected by *F. moniliforme* may also be toxic to animals (8).

The purposes of this investigation were to determine the level of *F. moniliforme* kernel infection in grain of commercial maize hybrids, the repeatability of reaction to *F. moniliforme* kernel infection among select maize genotypes, and the heritability of reaction to kernel infection by *F. moniliforme*.

MATERIALS AND METHODS

Kernels produced by 84 commercial maize hybrids, six inbreds, and the 15 possible single crosses among these inbreds were assayed for infection by *F. moniliforme*. The commercial hybrids included many of those either grown or being tested for future use in southeastern USA. Of the six inbreds used, three were considered resistant and three were considered susceptible to kernel infection by *F. moniliforme* in preliminary tests. Tests involving commercial hybrids were grown in four environments including single tests in 1977 and 1979 and two tests in 1978, one planted in early April (1978A) and the other planted in a separate field in late May (1978B). Although most commercial hybrids were grown in at least two environments, relatively few were grown in all four environments. Inbreds and their crosses were grown during both the 1978 and 1979 growing seasons. All plants were grown at Mississippi State, MS. We relied on natural infection by *F. moniliforme*.

A randomized block design with two replications in 1977 and three replications in 1978 and 1979 was used for each experiment. Plots were single rows, 5 m long and 1 m apart, with 15–20 plants. The top ear of each of the first 10 plants in a row was harvested 60 days after the midsilk date for the row. Ears were dried at 40 C for 5–7 days before shelling. Kernels from a given plot were thoroughly mixed and a random sample of ~300 kernels was stored at 6 C and 45% relative humidity until assayed.

Kernels were surface-sterilized by being dipped once in 70% ethyl alcohol, soaked for 3 min in 1.6% NaOCl (Clorox), and

TABLE 1. Percentage[x] of maize kernels infected with *Fusarium moniliforme*[y] for 15 hybrids grown in three tests

Hybrid	Brand	Test 1977	Test 1978A	Test 1978B	Mean
Low group:					
77	Coker	19 a[z]	14 ab	11 a	15 a
XL395	DeKalb	20 a	12 a	16 ab	16 a
511A	Pioneer	30 ab	50 f	30 b–e	37 b–d
56	Coker	38 bc	42 ef	31 b–e	37 b–d
G-4864	Funk's	38 bc	25 a–e	21 a–c	28 ab
G-4776	Funk's	42 bc	18 a–c	35 c–e	32 a–c
G-795W	Funk's	42 bc	54 fg	44 ef	47 b–e
PX715	Northrup King	49 cd	36 b–f	36 c–f	40 b–e
Mean		35	31	28	32
High group:					
3145	Pioneer	58 de	19 a–d	34 c–e	37 b–d
RA2601	Ring Around	63 ef	27 a–e	22 a–d	37 b–d
6663	Wilstar	65 ef	59 fg	37 c–f	54 d–f
75-200	McCurdy	68 e–g	38 c–f	38 c–f	48 c–e
G-4507	Funk's	70 e–g	50 f	40 d–f	53 d–f
PX675	Northrup King	72 fg	41 d–f	54 f	56 ef
TXS114	Trojan	78 g	75 g	53 f	69 f
Mean		68	44	40	51
Mean of all hybrids		50	37	33	40

[x] Values are means of three replications of 260 kernels each.
[y] Infection determined after incubation on Czapek solution agar for 4 days at 28 C.
[z] Values within a column not followed by the same letter differ significantly, $P = 0.05$.

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rinsed in sterile distilled water. Kernels were then placed on Czapek solution agar (Difco) in petri plates, 13 kernels per plate and 260 kernels per sample. After 4 days of incubation at 28 C, kernels were examined for the presence or absence of *F. moniliforme* colonies associated with them. Data on percentage of infected kernels per 260-kernel sample were subjected to a standard analysis of variance.

RESULTS

Commercial hybrids. Of the 84 commercial maize hybrids tested, 55, 67, 25, and 18 were evaluated in the 1977, 1978A, 1978B, and 1979 tests, respectively. All hybrids harbored *F. moniliforme* internally in apparently healthy kernels regardless of the year they were tested. The range of infection during the 3-yr period was from 5 to 89% and the mean infection levels for the four tests ranged from 50% in 1977 to 34% in 1979.

Fifteen commercial hybrids assayed for percentage of kernel infection in 1977 were also assayed in two separate tests in 1978. For purposes of presentation, we have divided the 15 hybrids into a low and a high group, based on percentage kernel infection in the 1977 test (Table 1). The mean infection level of the low group in 1977 was also lower than the high group in both 1978 tests. Overall mean infection for the 15 hybrids declined from 50% in 1977 to 37 and 33% in the two 1978 tests. The repeatability of results for given hybrids over the three tests was not always good. For example, kernel infection for Pioneer Brand 3145 was relatively high (58%) in the 1977 test and relatively low (19%) in the 1978A test.

Seven hybrids were common in tests in each of the 3 yr. When groups were divided into low and high groups, group differences were again consistent over tests. Some hybrids, however, notably Pioneer Brand 3369A and 3009, had considerably less infection in the 1978A test than in 1977 or 1979 (Table 2).

Only five hybrids were common in all four tests. Ranges in average percentage infection of the low group (three hybrids) and the high group (two hybrids) over the four tests were 16–26 and 54–75%, respectively. The relative reactions of these five hybrids to *F. moniliforme* kernel infection were generally consistent over the four tests (Table 3).

Inbreds and crosses among them. Six inbreds, known from previous tests to represent a wide range in reaction to kernel infection, and 15 hybrids produced by all possible crosses among them were assayed for *F. moniliforme* kernel infection in 1978 and 1979. The inbreds averaged 49 and 52% infection in 1978 and 1979, respectively (Table 4), and crosses averaged 30 and 35% (Table 5). Three inbreds, CI90C, Mp317, and SC170, had the lowest 2-yr average infection levels of 31, 20, and 31%, respectively. Inbreds Mp68:616 and Mp303 had the highest average infection levels of 76 and 88%, respectively, and Mp440 had 58% infection (Table 4). Average 2-yr infection levels for the 15 crosses among the six inbreds ranged from 5 to 65%. Results were generally consistent for the 2 yr (Table 5).

Crosses involving at least one of the three resistant inbreds had a 24–25% average 2-yr infection level and crosses involving at least one of the susceptible inbreds had 33–51% infection (Table 6). Average 2-yr infection levels of crosses in which both parents were

TABLE 2. Percentage[x] of maize kernels infected with *Fusarium moniliforme*[y] for seven hybrids grown in three tests

Hybrid	Brand	Test 1977	1978A	1979	Mean
Low group:					
77	Coker	19 a[z]	14 ab	14 ab	16 a
XL395	DeKalb	20 a	12 ab	10 a	14 a
G-4864	Funk's	38 b	25 b	27 c	30 ab
3369A	Pioneer	43 b	5 a	26 bc	25 ab
Mean		30	14	19	21
High group:					
3009	Pioneer	55 c	17 ab	42 d	38 b
PX675	Northrup King	72 d	41 c	65 e	59 c
TXS114	Trojan	78 d	74 d	58 e	70 c
Mean		68	44	55	56

[x] Values are means of three replications of 260 kernels each.
[y] Infection determined after incubation on Czapek solution agar for 4 days at 28 C.
[z] Values within a column not followed by the same letter differ significantly, $P = 0.05$.

TABLE 3. Percentage[x] of maize kernels infected with *Fusarium moniliforme*[y] for five hybrids grown in four tests

Hybrid	Brand	Test 1977	1978A	1978B	1979	Mean
Low group:						
77	Coker	19 a[z]	14 a	11 a	14 ab	15 a
XL395	DeKalb	20 a	12 a	16 a	10 a	15 a
G-4864	Funk's	38 b	25 ab	21 a	27 b	28 b
Mean		26	17	16	17	19
High group:						
PX675	Northrup King	72 c	41 b	54 b	65 c	58 c
TXS114	Trojan	78 c	75 c	53 b	58 c	66 c
Mean		75	58	54	62	62

[x] Values are means of three replications of 260 kernels each.
[y] Infection determined after incubation on Czapek solution agar for 4 days at 28 C.
[z] Values within a column not followed by the same letter differ significantly, $P = 0.05$.

TABLE 4. Percentage[x] of maize kernels infected with *Fusarium moniliforme*[y] for six inbreds in 2 yr

Inbred	Year 1978	1979	Mean
CI90C	25 a[z]	37 b	31 ab
Mp317	19 a	20 a	20 a
SC170	29 ab	23 a	31 ab
Mp68:616	73 c	79 cd	76 cd
Mp303	93 c	84 d	89 d
Mp440	47 c	69 c	58 bc
Mean	49	52	

[x] Values are means of three replications of 260 kernels each.
[y] Infection determined after incubation on Czapek solution agar for 4 days at 28 C.
[z] Values within a column not followed by the same letter differ significantly, $P = 0.05$.

TABLE 5. Percentage[x] of maize kernels infected with *Fusarium moniliforme*[y] in all possible crosses among six parents grown in 2 yr

Pedigree	Year 1978	1979	Mean
CI90C × Mp317	5 a[z]	5 a	5 a
CI90C × SC170	15 ab	20 b	18 bc
CI90C × Mp68:616	35 c–e	41 cd	38 de
CI90C × Mp303	33 c–e	51 de	42 d–f
CI90C × Mp440	20 a–c	19 b	20 c
Mp317 × SC170	12 ab	11 ab	12 a–c
Mp317 × Mp68:616	23 a–d	16 ab	20 c
Mp317 × Mp303	36 c–e	57 e	47 e–g
Mp317 × Mp440	26 b–d	37 c	32 d
SC170 × Mp68:616	36 c–e	33 c	35 de
SC170 × Mp303	46 e	60 e	53 fg
SC170 × MP440	5 a	10 ab	8 ab
Mp68:616 × Mp303	71 f	59 e	65 h
Mp68:616 × Mp440	39 de	49 de	44 e–g
Mp303 × Mp440	51 e	59 e	55 gh
Mean	30	35	

[x] Values are means of three replications of 260 kernels each.
[y] Infection determined after incubation on Czapek solution agar for 4 days at 28 C.
[z] Values within a column not followed by the same letter differ significantly, $P = 0.05$.

TABLE 6. Percentage of maize kernels infected by *Fusarium moniliforme* for all crosses involving each of the six parental lines grown in 2 yr

Common parent	Year		Mean
	1978	1979	
CI90C	21	27	24
Mp317	22	25	24
SC170	22	27	25
Mp68:616	41	40	41
Mp303	45	57	51
Mp440	31	35	33

resistant was 11% compared with 55% for crosses in which both parents were susceptible and 33% for crosses in which one parent was resistant and one parent was susceptible.

DISCUSSION

The common occurrence of *F. moniliforme* infection in apparently healthy maize kernels reported here concurs with other reports (7,9,17). Considerable variation in infection levels occurred among commercial hybrids (Tables 1, 2, and 3). Handling and assay procedures were the same for all tests; therefore, differences in infection levels among tests were likely due to environmental factors. Inbreds had considerably more kernel infection than crosses involving them (Tables 4 and 5). The greater susceptibility of inbreds to infection may be related to their low plant vigor in comparison to hybrids. Ears of hybrids usually had less insect damage than ears of inbreds. Although only undamaged kernels were assayed for *F. moniliforme*, insects may have transmitted inoculum to kernels and predisposed plants to subsequent infection by the fungus.

Among commercial hybrids, some, such as Coker 77 and Dekalb XL395, had consistently less kernel infection than others, such as Trojan TXS114 and Northrup King PX675 (Tables 1, 2, and 3). High variability in percentage infection, however, occurred among replicates of most hybrids in each test. Factors that caused this variability are not known. A method to reduce variability among replications would ease the determination of reaction differences among genotypes for *F. moniliforme* kernel infection.

The six inbreds used in this study were selected because they represented differences in reaction to *F. moniliforme* kernel infection in previous tests. These differences were confirmed in tests during 2 yr (Table 4). The data show that the reaction of a hybrid to *F. moniliforme* kernel infection is generally similar to the reactions of its parents, if these are similar, and intermediate to their reactions, if they are dissimilar (Tables 4 and 5). One notable exception was the hybrid SC170 × Mp440, a resistant × susceptible cross, which reacted similarly to resistant × resistant crosses. The reason for the highly resistant response in this hybrid is not known.

From the data obtained, we conclude that high levels of asymptomatic infection of kernels by *F. moniliforme* are likely to occur in commercially produced maize. Genotypic differences have been reported for reaction to *F. moniliforme* kernel rot (15,20) and asymptomatic kernel infection (20). Our results demonstrate for the first time, however, that inbred differences for asymptomatic kernel infection are expressed in their hybrids. Relationships between the occurrence of *F. moniliforme* in asymptomatic kernels and the occurrence of kernel rot caused by this fungus have not been determined.

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