Maternal Influence on the Resistance of Sweet Corn Lines to Kernel Infection by *Fusarium moniliforme*

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ABSTRACT

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Sweet corn inbred lines resistant and susceptible to kernel infection by *Fusarium moniliforme* were hand pollinated to produce parental, F_1, F_2, F_3, and backcross generations with reciprocals to examine maternal effects on resistance. Silks of half of the hand-pollinated plants in each generation were inoculated with *F. moniliforme*. Incidence of symptomatic and asymptomatic infection of kernels by *F. moniliforme* was determined from inoculated and uninoculated ears. Factors operative in maternal tissues had a strong influence on the response of sweet corn lines to kernel infection by *F. moniliforme* and were not influenced by the cytoplasm, endosperm, or embryo. Infection of kernels produced on resistant parents (P_R, F_1, and backcross generations) was less than that of kernels produced on susceptible parents (P_S, F_1, and backcross generations). Infection of kernels produced on the same ear parent was not significantly different regardless of generation. Maternal tissues that may influence the resistance to kernel infection include the silk, pericarp, and closing layer. Silks that grew actively for several days after pollination inhibited kernel infection by *F. moniliforme* and therefore appeared to be the maternal tissue most likely to influence kernel infection. The implications of these findings in breeding for resistance to kernel infection by *F. moniliforme* are to select inbred parents for delayed senescence of silks and to use identified sources of resistance, such as IL125b, as the ear parent in seed production.

Additional keywords: *Gibberella fujikuroi*, *shrunken-2*, *Zea mays*.

In the past 10 yr, sweet corn (*Zea mays* L.) with the *shrunken-2* (*sh2*) endosperm mutation has supplanted traditional sweet corn with the *sugary-1* (*su*) endosperm mutation as the type preferred by many consumers. In the homozygous recessive condition, the *sh2* mutation results in greatly reduced levels of adenosine 5′-diphosphate glucose pyrophosphorylase (3,11). This enzyme deficiency leads to the accumulation of sucrose in the kernels at the expense of water-soluble polysaccharides and starch, thus producing a very sweet taste (3).

Compared to sugary hybrids, homozygous *sh2* hybrids have been hampered by poor stands, reduced seedling vigor, and a high incidence of seedling blight. Kernel infection by *Fusarium moniliforme* Sheldon (teleomorph: *Gibberella fujikuroi* (Sawada) Wollenw.) has been reported as one of the causes of these seedling problems (1,4,5,19,20). Chemical and cultural controls of *F. moniliforme* have been relatively unsuccessful because *F. moniliforme* is a cosmopolitan species that grows well saprophytically and survives effectively in live, asymptomatic corn kernels. Genetic resistance to kernel infection offers the most potential for control.

Resistance to kernel infection by *F. moniliforme* has been studied in starchy endosperm backgrounds by several methods, which have produced variable results (2,9,12,13,18). Boling and Grogan (2) used generation means analysis of a resistant × susceptible cross to determine that epistatic, dominance, and additive gene effects were important in resistance to ear rot caused by *F. moniliforme*. Lunsford, Futrell, and Scott (12,13) determined that maternal effects and additive gene action were more important than dominant gene action in the inheritance of resistance to seedling blight caused by *F. moniliforme*. King and Scott (9) found that differences among inbreds in asymptomatic infection of kernels were expressed in their hybrids; however, responses of the reciprocal crosses necessary to assess maternal effects were not reported. Scott and King (18) later suggested that the site of action of resistance to kernel infection by *F. moniliforme* was the pericarp and that the genotype of the endosperm, embryo, or cytoplasm had little effect on resistance. They hypothesized, however, that the maternal effect could be due to silk tissue, if the mode of entry of *F. moniliforme* into the kernels was through silks.

Many authors consider colonization of silks to be the primary mode of entry of *F. moniliforme* into developing kernels (5,7,10, 14,21). Moisture content of silks at the time of inoculation reportedly affects the incidence of *F. moniliforme* in kernels (6,21). Marsh and Payne (15) also demonstrated that the degree of silk senescence was important in colonization of kernels by *Aspergillus flavus* Link ex Fries.

Sources of resistance to kernel infection by *F. moniliforme* recently were identified in sweet corn backgrounds (5). Resistant lines were observed to have silks that actively grew for a longer period of time after pollination than did silks of susceptible lines (6). Differences between silks of resistant and susceptible genotypes may account for the maternal effect on resistance to *F. moniliforme*, if the characteristics of silks are maintained in hybrid combination and these characteristics are not affected by the genotype of the endosperm. More information regarding the inheritance and mechanism of resistance to kernel infection by *F. moniliforme* in sweet corn is needed to enhance the effective use of sources of resistance.

The objectives of this study were to examine the maternal influence on resistance to kernel infection by *F. moniliforme* in sweet corn and to evaluate the effects of endosperm mutations on kernel infection by *F. moniliforme* and emergence.

MATERIALS AND METHODS

Genotypes and generations tested. Genotype and cytoplasm combinations were developed to differentiate the effects of endosperm, embryo, cytoplasmic, and maternal plant tissues on resistance to kernel infection by *F. moniliforme* (Table 1). Kernels produced in these experiments were one generation advanced from the plants on which they were produced. For example, F_1 hybrid kernels were produced on inbred plants, F_2 kernels were produced on F_1 plants, etc. Thus F_1 kernels had maternal tissues from one inbred parent (i.e., the ear parent). Likewise, F_2 and backcross generations received a maternal component from each inbred parent.

Two experiments were done. In experiment 1, a sweet corn inbred resistant to kernel infection by *F. moniliforme* (IL125b) and six susceptible sweet corn inbreds (FA56a, I2123, I2256b, I5125, IL783a, and IL784a) were evaluated. IL125b is a sugary inbred that exhibited partial resistance to kernel infection by *F. moniliforme* and good emergence (5). FA56a, IL783a, and IL784a are shrunken inbreds that are susceptible to *F. moniliforme*. I2256b, I2123, and I5125 are susceptible, sugary inbreds. Also, FA56a is a shrunken conversion of I2256b (22). The resistant inbred parent, P_R (IL125b), and the susceptible inbred parents, P_S (FA56a, I2123, I2256b, I5125, IL783a, and IL7841), and all reciprocal F_1 generations, $P_R \times P_S$ and $P_S \times P_R$, were produced by hand pollination and evaluated in 1987 at Urbana, IL. Reciprocal F_2 generations, $(P_R \times P_S)F_2$ and $(P_S \times P_R)F_2$, were produced by hand pollination in Hawaii during the winter of 1987–1988 in the breeding nursery of Illinois Foundation Seeds, Inc. In 1988 and 1989, the inbreds IL125b, FA56a, and IL783a were evaluated. The above generations as well as reciprocal F_3 generations, $(P_R \times P_S)F_3$, $(P_S \times P_R)F_3$, and backcross generations, $P_R \times (P_R \times P_S)$, $P_R \times (P_S \times P_R)$, $P_S \times (P_R \times P_S)$, $P_S \times (P_S \times P_R)$, $(P_R \times P_S) \times P_R$, $(P_R \times P_S) \times P_S$, $(P_S \times P_R) \times P_R$, and $(P_S \times P_R) \times P_S$, were produced by hand pollination at Urbana for IL125b (P_R) and FA56a and IL783a (P_S). The ear parent is listed first in all hybridizations.

In experiment 2, the starchy inbred, B14, which had been identified as resistant to kernel infection by *F. moniliforme* (21), was crossed to FA56a and I2256b. Parental and reciprocal F_1 generations were produced in 1986. Parental and reciprocal F_1 and F_2 generations were produced in 1987. Parental and reciprocal F_1, F_2, F_3, and backcross generations were produced in 1988 and 1989. All plants were hand pollinated at Urbana.

In both experiments, kernels of parental lines and reciprocal F_1 hybrids were produced in single rows with 15 plants per row. Kernels of segregating generations were produced in paired rows with 15 plants per row. Both experiments were planted twice in 1988 and 1989.

Many of the crosses involved combinations of endosperm mutants. For example, the crosses of sugary (*susuSh2Sh2*) × shrunken-2 (*SuSush2sh2*) inbreds produced F_1 kernels that were starchy (*Su–Sh2–*) and F_2 kernels that segregated in a 9:3:3:1 ratio of starchy, sugary, shrunken, and sugary-shrunken endosperm phenotypes, respectively. Kernels were separated by endosperm phenotype in the F_2 generation before selfing to produce the F_3 generation. The double-recessive sugary-shrunken phenotypic class, *sush2*, was the only class in which the endosperms were genotypically identical at the *su* and *sh2* loci because endosperms in the remaining three classes had one to three dominant alleles at the *su1* or *sh2* loci (11). Similarly, kernels from the crosses with B14 were separated by endosperm phenotype before producing the next generation.

Inoculation. Silks of three or four of the hand-pollinated plants in each row were inoculated with a suspension of microconidia of *F. moniliforme* for 4–7 and 18–21 days after pollination. The fungus was grown on potato-dextrose agar (PDA) in petri plates at room temperature. Cultures were flooded with water and gently rubbed with a glass rod to dislodge conidia. The inoculum concentration was determined with a hemacytometer and adjusted to approximately 10^6 microconidia/ml. In 1987, 1988, and 1989, the inoculum was a mixture of 17, 13, or 19 isolates from infected corn kernels. About 5 ml of the inoculum suspension was sprayed onto the silks after the tassel bag had been removed. Inoculated

TABLE 1. Genotype and cytoplasm of parents and reciprocal F_1, F_2, F_3, and backcross generations when the parents have different cytoplasms and also differ for a single nuclear gene for resistance to kernel infection[a]

Generation	Silk and pericarp	Endosperm	Embryo	Cytoplasm
P_R (resistant)	AA	AAA	AA	X
P_S (susceptible)	aa	aaa	aa	Y
$P_R \times P_S$[b]	AA	AAa	Aa	X
$P_S \times P_R$	aa	Aaa	Aa	Y
$(P_R \times P_S) \times P_R$	Aa	AAA Aaa	AA Aa	X
$(P_R \times P_S) \times P_S$	Aa	AAa aaa	Aa aa	X
$P_R \times (P_R \times P_S)$	AA	AAA AAa	AA Aa	X
$P_S (P_R \times P_S)$	aa	Aaa aaa	Aa aa	Y
$(P_S \times P_R) \times P_R$	Aa	AA Aaa	AA Aa	Y
$(P_S \times P_R) \times P_S$	Aa	AAa aaa	Aa aa	Y
$P_R \times (P_S \times P_R)$	AA	AAA AAa	AA Aa	X
$P_S \times (P_S \times P_R)$	aa	Aaa aaa	Aa aa	Y
$(P_R \times P_S)\ F_2$	Aa	AAA AAa Aaa aaa	AA Aa aa	X
$(P_S \times P_R)\ F_2$	Aa	AAA AAa Aaa aaa	AA Aa aa	Y
$(P_R \times P_S)\ F_3$	AA Aa aa	AAA AAa Aaa aaa	AA Aa aa	X
$(P_S \times P_R)\ F_3$	AA Aa aa	AAA AAa Aaa aaa	AA Aa aa	Y

[a] From Scott and King (18).

[b] Ear (maternal) parent of hybridization listed first, pollen parent second.

silks then were covered with a plastic freezer bag and the tassel bag to insure adequate moisture for fungal growth. Plastic bags were removed 48 hr later. Tassel bags were left on the ears until harvest. In experiment 1 in 1987, only the sugary × sugary crosses were inoculated. In 1988 and 1989, all generations in each family were inoculated. In experiment 2, all generations of all crosses were inoculated each year. In each experiment, inoculated and uninoculated ears were harvested 3–4 wk after the second inoculation. Harvested ears were dried with ambient forced air before shelling. Ears from each row were bulked by inoculation treatment. Kernels were evaluated for the incidence of symptomatic and asymptomatic infection by *F. moniliforme*.

Symptomatic infection. Symptomatic infection was determined from samples of 100 kernels taken at random from inoculated and uninoculated treatments in each plot. The kernels exhibiting signs or symptoms of *F. moniliforme* were counted. Incidence of symptomatic infection then was converted to a percentage. In segregating generations, the kernels were separated by endosperm phenotype before assessment, and the percentage of symptomatic infection was determined from 100 kernels of each phenotype per inoculation treatment per row.

Asymptomatic infection. Asymptomatic infection of kernels by *F. moniliforme* was based on healthy appearing kernels (those with no signs or symptoms of infection or insect damage). Sample size differed among years and generations. In 1987, 1988, and 1989, sample size was 40, 80, and 160 kernels, respectively, per endosperm phenotype per generation per family. In 1987, 20 kernels (10 from the inoculated treatment and 10 from the uninoculated treatment) were plated for each endosperm phenotype of each generation of each family for each replicate (planting date). In 1988 and 1989, 20 and 40 kernels, respectively, were plated from inoculated and uninoculated treatments of each endosperm phenotype of each generation of each family for each replicate (planting date). Thus, for example, in 1989 a total of 160 and 640 kernels were plated for the F_1 and F_2 generations, respectively, of IL125b × FA56a (i.e., F_1 = 40 kernels × 2 inoculation treatments × 1 endosperm phenotype × 2 replicates, F_2 = 40 kernels × 2 inoculation treatments × 4 endosperm phenotypes × 2 replicates). Kernels of each endosperm phenotype were plated separately in segregating generations.

Kernels were surface sterilized by soaking for 2 min each in 0.8% sodium hypochlorite and 90% ethanol followed by two rinses with sterile distilled water. In 1987, kernels were plated on PDA amended with tetracycline and streptomycin and on a *Fusarium*-selective medium containing pentachloronitrobenzene (PCNB) (16). In 1988 and 1989, kernels were plated only on the *Fusarium*-selective PCNB medium. Incidence of *F. moniliforme* and other fungi and germination of kernels were recorded 5–6 days after plating and were converted to percentages.

Emergence. The percentage of emerged seedlings from kernels of resistant and susceptible parents, F_1, and F_2 generations was determined for each planting date in experiments 1 and 2 in 1988. The kernels used for planting the experiments were uninoculated and did not exhibit any signs or symptoms of fungal infection. Sixty kernels were plated per experimental unit. Each generation was replicated four times.

Silk notes. In experiment 1 in 1988, the length and color of silks were determined when silks were inoculated with *F. moniliforme* 1 and 3 wk after pollination. Silks were categorized as short, medium, or long based on relative length, and green, green-brown, or brown based on the degree of senescence.

Data analyses. Incidence of symptomatic infection and asymptomatic infection of kernels by *F. moniliforme* was analyzed by analysis of variance for both experiments in each year with inoculation treatments and generations as main effects and endosperm phenotypes nested within generations. Families were combined among P_S of similar endosperm types (i.e., sugary or *shrunken-2*) after preliminary analysis indicated that families derived from various susceptible parents responded similarly. Main effects and interactions were tested with a pooled error term after testing for homogeneity of error variance. Waller-Duncan Bayesian least significant difference values with k = 100 were used for comparisons within inoculation treatments, generations, and endosperm phenotypes. Comparisons of the effect of endosperm phenotype on emergence of seedlings and the effects of length and color of silks on kernel infection by *F. monlliforme* were done by *t*-tests. Analyses were done with untransformed, log-transformed, and arcsine-transformed data.

RESULTS

In all experiments, families derived from the various susceptible parents of similar endosperm phenotype responded similarly; thus, data were combined among susceptible parents with similar endosperm phenotypes (e.g., data were combined for FA56a, IL783a, and IL784a families and for I2256b, I2123, and I5125 families).

For both experiments, inoculations and generations had a significant effect on kernel infection; however, endosperm phenotypes did not affect kernel infection except for symptomatic infection in 1989 when the percentage of symptomatic kernels was 10, 11, 26, and 42% for starchy, sugary, shrunken, and sugary-shrunken endosperms, respectively. Likewise, interactions among inoculations, generations, and endosperm phenotypes were not significant; therefore, data were combined among endosperm phenotypes and inoculation treatments within generations. Analyses were similar for untransformed and transformed data. Data presented in tables are from analyses of untransformed data.

Maternal influence on kernel infection by *F. moniliforme*. A maternal influence on kernel infection by *F. moniliforme* was evident. In experiment 1 in 1987 and 1988, symptomatic and asymptomatic infections by *F. moniliforme* were significantly less for kernels of the resistant inbred (P_R) and F_1 kernels produced on the resistant inbred compared with kernels of the susceptible inbred (P_S) and F_1 kernels produced on the susceptible inbred

TABLE 2. Incidence of symptomatic and asymptomatic infection of corn kernels by *Fusarium moniliforme* for parents and F_1 hybrids of the resistant, sugary inbred line IL125b and susceptible, sugary inbred lines I2256b, I2123, and I5125 or susceptible, shrunken inbred lines IL783a, IL784, and FA56a in 1987

Ear parent and generation[w]	IL125b and sugary lines[v]		IL125b and shrunken lines[v]	
	Symptomatic infection[x]	Asymptomatic infection[y]	Symptomatic infection	Asymptomatic infection
P_R ear parent				
P_R (resistant)	0 a[z]	20 a	0 a	20 a
$P_R \times P_S$	0 a	23 a	0 a	0 a
P_S ear parent				
P_S (susceptible)	8 b	77 b	26 b	83 b
$P_S \times P_R$	29 b	100 b	10 b	87 b

[v] IL125b and sugary lines were inoculated as described in the text; IL125b and shrunken lines were uninoculated.
[w] P_R = IL125b. P_S = mean response of I2256b, I2123, and I5125, or mean response of IL783a, IL784a, and FA56a.
[x] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[y] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[z] Values followed by the same letter are not significantly different at the 0.05 level based on BLSD-comparisons within columns.

(Tables 2 and 3). Symptomatic and asymptomatic infections of F_1 kernels did not differ significantly from those of kernels produced on the ear parent of the cross (Tables 2 and 3). In experiment 1 in 1989, symptomatic infection was not significantly different among the P_R, P_S, and F_1 kernels produced on either parent; however, asymptomatic infection was similar to results from 1987 and 1988 when infection was less for P_R and F_1 kernels produced on the resistant parent compared with those produced on the susceptible parent (Table 3). Similarly, kernels from backcross generations produced on the resistant parent, $P_R \times (P_R \times P_S)$ and $P_R \times (P_S \times P_R)$, had significantly less symptomatic and asymptomatic infections than did kernels from backcross generations produced on the susceptible parent, $P_S \times (P_R \times P_S)$ and $P_S \times (P_S \times P_R)$ (Table 3). Symptomatic and asymptomatic infections of kernels from backcross generations produced on the inbred parents did not differ from those of the F_1 kernels that had the same ear parent (Table 3), except for kernels from the $P_S \times (P_S \times P_R)$ backcross for which symptomatic infection

TABLE 3. Incidence of symptomatic and asymptomatic infection of corn kernels by *Fusarium moniliforme* for generations of crosses of the resistant, sugary inbred line IL125b and susceptible, shrunken inbred lines FA56a and IL783a in 1988 and 1989

Ear parent and generation[w]	1988		1989	
	Symptomatic infection[x]	Asymptomatic infection[y]	Symptomatic infection	Asymptomatic infection
P_R ear parent				
P_R (resistant)	5 a[z]	12 a	8 ab	13 a
$P_R \times P_S$	14 a	0 a	11 ab	8 a
$P_R \times (P_R \times P_S)$	0 a	5 a	9 ab	30 abc
$P_R \times (P_S \times P_R)$	...	...	9 ab	20 ab
P_S ear parent				
P_S (susceptible)	72 c	65 d	21 bcd	86 fg
$P_S \times P_R$	62 bc	56 cd	19 bcd	71 efg
$P_S \times (P_R \times P_S)$	...	...	25 cde	90 g
$P_S \times (P_S \times P_R)$	46 b	39 bc	44 f	80 fg
$(P_R \times P_S)$ ear parent				
$(P_R \times P_S) \times P_R$	1 a	4 a	4 a	27 abc
$(P_R \times P_S) \times P_S$	5 a	2 a	12 abc	39 bcd
$(P_R \times P_S)$ F_2	4 a	5 a	15 abc	40 bcd
$(P_S \times P_R)$ ear parent				
$(P_S \times P_R) \times P_R$	10 a	0 a	10 ab	43 bcd
$(P_S \times P_R) \times P_S$	8 a	0 a	12 abc	41 bcd
$(P_S \times P_R)$ F_2	2 a	8 a	13 abc	44 cde
F_2 ear parent				
$(P_R \times P_S)$ F_3	9 a	14 a	30 de	66 def
$(P_S \times P_R)$ F_3	10 a	19 a	36 ef	51 cde

[w] P_R = IL125b. P_S = mean response of FA56a and IL783a. There were no significant differences among endosperm phenotypes within segregating generations in 1988 and 1989; therefore, data were combined among endosperm phenotypes within generations.
[x] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[y] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[z] Values followed by the same letter are not significantly different at the 0.05 level based on BLSD-comparisons within columns.

TABLE 4. Incidence of symptomatic and asymptomatic infections of corn kernels by *Fusarium moniliforme* for generations of crosses of the resistant, sugary inbred line B14 and susceptible, sweet inbred lines I2256b and FA56a and 1988 and 1989

Ear parent and generation[w]	1988		1989	
	Symptomatic infection[x]	Asymptomatic infection[y]	Symptomatic infection	Asymptomatic infection
P_R ear parent				
P_R (resistant)	0 a[z]	0 a	3 a	9 ab
$P_R \times P_S$	4 a	5 a	18 abc	10 ab
$P_R \times (P_R \times P_S)$	4 a	3 a	31 bc	5 a
$P_R \times (P_S \times P_R)$	...	...	27 abc	12 ab
P_S ear parent				
P_S (susceptible)	26 a	75 c	32 bc	59 def
$P_S \times P_R$	59 b	78 c	42 c	34 c
$P_S \times (P_R \times P_S)$	...	...	...	...
$P_S \times (P_S \times P_R)$	24 a	41 b	38 c	66 ef
$(P_R \times P_S)$ ear parent				
$(P_R \times P_S) \times P_R$	4 a	5 a	33 bc	57 def
$(P_R \times P_S) \times P_S$	1 a	1 a	43 c	46 cde
$(P_R \times P_S)$ F_2	3 a	5 a	8 ab	46 cde
$(P_S \times P_R)$ ear parent				
$(P_S \times P_R) \times P_R$	...	...	31 bc	29 bc
$(P_S \times P_R) \times P_S$	...	...	38 c	42 cd
$(P_S \times P_R)$ F_2	3 a	23 ab	11 ab	67 f
F_2 ear parent				
$(P_R \times P_R)$ F_3	6 a	13 ab	26 abc	61 def
$(P_S \times P_S)$ F_3	5 a	7 a	30 bc	75 f

[w] P_R = B14. P_S = mean response of I2256b and FA56a. There were no significant differences among endosperm phenotypes within segregating generations in 1988 and 1989; therefore, data were combined among endosperm phenotypes within generations.
[x] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[y] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[z] Values followed by the same letter are not significantly different at the 0.05 level based on BLSD-comparisons within columns.

was higher in 1989.

Symptomatic and asymptomatic infections of the F_2 and F_3 kernels and the kernels of backcross generations produced on F_1 plants did not differ significantly from infections of the kernels of the resistant parent in 1988 (Table 3). In 1989, symptomatic infection was greater for F_3 kernels than for kernels of the resistant parent, but there was no difference among F_2 kernels, kernels from backcross generations produced on F_1 plants, and kernels from the resistant parent. Asymptomatic infection of F_2 and F_3 kernels and kernels from backcross generations produced on F_1 plants was intermediate to that of kernels from the resistant and susceptible parents in 1989.

In 1988 and 1989, results from experiment 2 were similar to those from experiment 1. Kernels produced on the resistant inbred (P_R, F_1, and backcross generations) had less symptomatic or asymptomatic infections by *F. moniliforme* than those produced on a susceptible inbred (Table 4). Means for symptomatic infection also were lower for kernels produced on the resistant parent compared with those for kernels produced on the susceptible parent although differences were not always significant. Infection of kernels produced on F_1 or F_2 plants (backcrosses and F_2 and F_3 kernels) did not differ significantly from that of kernels produced on the resistant parent in 1988 but generally was intermediate to infection of the two inbred parents in 1989.

Effect of condition of silks on kernel infection by *F. moniliforme*. Color and length of silks at the time of inoculation were associated with the incidence of symptomatic and asymptomatic infections of kernels by *F. moniliforme*. Symptomatic infection of kernels was significantly greater when brown silks were inoculated 3 wk after pollination compared with green-brown or green silks (Table 5). Symptomatic infection was not affected by color of silks 1 wk after pollination. Asymptomatic infection of kernels was significantly lower when green silks were inoculated 1 or 3 wk after pollination compared with brown or green-brown silks. Asymptomatic infection also was greater when brown silks were inoculated 3 wk after pollination as opposed to green-brown silks (Table 5). Brown silks were indicative of senescence.

Length of silks was associated with symptomatic and asymptomatic infections of kernels by *F. moniliforme* at both inoculation times. Symptomatic infection of kernels was greatest when short silks were inoculated and least when long silks were inoculated (Table 6). Asymptomatic infection of kernels also was greatest when short silks were inoculated and least when long silks were inoculated; however, no difference existed in infection between medium and long silks 3 wk after pollination (Table 6).

Effect of endosperm mutations on emergence. Differences in emergence of seedlings due to endosperm phenotypes were observed among uninoculated, asympotmatic F_2 kernels of reciprocal crosses between sugary (*susuSh2Sh2*) and shrunken (*suSush2sh2*) inbreds (Table 7). F_2 kernels of all four endosperm phenotypes collected from the same ears of F_1 plants differed in emergence. Emergence of seedlings from sugary kernels ranged from 42 to 83% and did not differ significantly from that of starchy kernels, which ranged from 56 to 92%. Emergence of seedlings from shrunken kernels ranged from 18 to 52% and was significantly less than that from sugary and starchy kernels. The double-recessive phenotype, *susush2sh2*, often was lethal, and emergence of seedlings from sugary-shrunken kernels was significantly less than that from shrunken, sugary, or starchy kernels.

DISCUSSION

A strong maternal influence on the response of sweet corn lines to kernel infection by *F. moniliforme* was evident from our research. Based on the susceptibility of all generations produced on susceptible parents and the resistance of all generations produced on resistant parents, the response seems to result from factors operative in the maternal tissues of the kernels, such as the silk, pericarp, or placento-chalazal region. The cytoplasm does not appear to be the source of the maternal effect because there were no significant differences in kernel infection by *F. moniliforme* between kernels produced on reciprocal F_1 or F_2 plants, or in kernels of backcross generations where reciprocal F_1 plants were used as the ear parent. Likewise, the endosperm and embryo

TABLE 5. Association of color of silk after pollination with symptomatic and asymptomatic infections of kernels by *Fusarium moniliforme* from inoculated ears of several generations of crosses between sweet corn inbreds in 1988

	Time of inoculation and evaluation of silks					
	1 wk after pollination			3 wk after pollination		
Color	Number[w]	Symptomatic infection[x]	Asymptomatic infection[y]	Number	Symptomatic infection	Asymptomatic infection
Brown	4	27.0 a[z]	26.2 b	126	22.7 b	23.2 c
Green-brown	162	18.1 a	22.9 b	218	15.3 a	17.8 b
Green	190	17.7 a	16.3 a	10	18.2 a	3.3 a

[w] Number of genotypes in each category of silk color.
[x] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*. Values represent means of all generations of all crosses.
[y] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene. Values represent means of all generations of all crosses.
[z] Values followed by the same letter are not significantly different at the 0.05 level based on *t*-tests for comparisons within columns.

TABLE 6. Association of length of silk after pollination with symptomatic and asymptomatic infections of kernels by *Fusarium moniliforme* from inoculated ears of several generations of crosses between sweet corn inbreds in 1988

	Time of inoculation and silk evaluation					
	1 wk post pollination			3 wk post pollination		
Length	Number[w]	Symptomatic infection[x]	Asymptomatic infection[y]	Number	Symptomatic infection	Asymptomatic infection
Short	21	42.1 c[z]	43.2 c	39	44.0 c	40.8 b
Medium	228	18.4 b	20.7 b	238	16.0 b	18.5 a
Long	107	12.3 a	11.9 a	77	10.9 a	10.8 a

[v] Length of silks (estimated but not measured): short, <5 cm; medium, , 5–10 cm; long, >10 cm.
[w] Number of genotypes in each category of silk length.
[x] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*. Values represent means of all generations of all crosses.
[y] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene. Values represent means of all generations of all crosses.
[z] Values followed by the same letter are not significantly different at the 0.05 level based on *t*-tests for comparisons within columns.

do not influence the observed response because no differences existed between kernels of the resistant parent and F_1 kernels or kernels of backcross generations produced on the resistant parent. There also were no significant differences in infection of kernels by *F. moniliforme* among endosperm phenotypes except for symptomatic infection in 1989, thus confirming previous results (6) that sugar concentration of the kernels does not affect infection by *F. moniliforme*.

Our results agree with those of Scott and King (18), who concluded that the pericarp was the site of action for resistance to kernel infection by *F. moniliforme* in starchy inbred lines, Mp317 and SC170. Although the pericarp may be the site of resistance to kernel infection by *F. moniliforme* in sweet corn, the results presented here and those of Scott and King do not exclude other maternal tissues as possible sites of action for resistance. Another maternal tissue that may function in resistance is the placento-chalazal region of the hilar orifice. Infection of kernels by *F. moniliforme* has been thought to result from growth of the fungus along the silks to the surface of the developing kernel, which then is penetrated in the tip cap region (10,17,23). Zummo and Scott (23) recently reported greater recovery of *F. moniliforme* from the pedicel end of kernels than from the apical end of kernels and concluded that *F. moniliforme* appeared to penetrate kernels mostly through the pedicel. The closing layer in the placento-chalazal region beneath the tip cap at the hilar orifice (i.e., the black layer of the kernel) is of maternal origin and may serve as a barrier to fungal ingress (8). It has been observed that the resistant inbred, IL125b, has a dehiscent tip cap and exhibits a more conspicuous closing layer than most other sweet corn inbreds. The importance of the closing layer in the resistance of IL125b to kernel infection by *F. moniliforme* requires further investigation.

Perhaps the most plausible explanation for the observed maternal effects on kernel infection in this study is the role of the silks in resisting fungal invasion. This study and earlier work (6) showed that silks that remained green after pollination were associated with reduced kernel infection by *F. moniliforme*. Longer silks at the time of inoculation also were associated with less kernel infection by *F. moniliforme* compared with shorter silks. Thus, silks that grow actively for several days after pollination may inhibit infection by *F. moniliforme*, compared with those that rapidly senesce. This mechanism of resistance seems consistent with the biology of *F. moniliforme*, which is an opportunistic saprophyte, but only a weak pathogen. Nevertheless, if the resistant genotypes in this study, IL125b and B14, had silks that grew longer and senesced slowly, then one would expect the association observed in this study, whether or not length and color of silks caused resistance to *F. moniliforme*. In the previous study (6), the association between color and length of silks and *F. moniliforme* was observed among 49 different sweet corn inbreds.

In our study, healthy appearing, uninoculated shrunken kernels from several F_2 lines emerged very poorly compared with starchy and sugary kernels from the same ears, even though there were no significant differences in infection by *F. moniliforme* in kernels with different endosperm phenotypes within the same crosses. These observations corroborate earlier conclusions (5,6) that shrunken sweet corn is not more susceptible to infection by *F. moniliforme* as a result of the high sugar concentration of the kernels but that seedling vigor of *sh2* genotypes is inherently poor, due to undetermined causes. The seedling vigor problem was exacerbated in kernels of the double-recessive phenotype, *sush2*, which often were nonviable.

Our results from 1988 also agreed with those of Scott and King (18) in that infection of kernels produced on F_1 plants did not differ significantly from infection of kernels produced on the resistant parent. Thus, resistance to kernel infection by *F. moniliforme* appeared to be dominant because the maternal tissue of the F_1 plant, which received a component from both the resistant and the susceptible parent, exhibited the reaction of the resistant parent. Segregation for susceptibility would be anticipated in the F_3 kernels produced on F_2 plants, and infection of F_3 kernels was higher than infection of F_2 kernels in our study, although the differences were not significant. However, in 1989, asymptomatic infection of kernels produced on F_1 plants was greater than that of kernels produced on the resistant parent. Thus, additional experimentation is required before conclusions can be made concerning the inheritance of resistance.

The implications of our findings in breeding for resistance to kernel infection by *F. moniliforme* are to select inbred parents for delayed senescence of silks and to use identified sources of resistance as ear parents in seed production.

TABLE 7. Percentage of seedlings emerged in 1988 from uninoculated, asymptomatic F_2 kernels of reciprocal crosses between sugary and shrunken sweet corn inbred lines

	Percentage emergence[y] of endosperm phenotype			
Cross[z]	Starchy	Sugary	Shrunken	Sugary-shrunken
IL125b × IL783a	58	55	18	0
IL783a × IL125b	56	42	30	0
IL125b × FA56a	92	83	52	6
FA56a × IL125b	65	74	25	5
W6786 × IL783a	84	75	36	10
IL783a × W6786	56	64	33	7
W6786 × FA56a	80	76	22	5
FA56a × W6786	78	73	22	0

[y] Mean of four rows with about 15 plants per row. Emergences of starchy and sugary kernels were not significantly different within crosses. All other comparisons of endosperm phenotypes within crosses were different at the 0.01 level based on *t*-tests.

[z] IL125b and W6786 are sugary inbreds, which exhibit good emergence. IL783a and FA56a are shrunken inbreds, which emerge poorly.

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