Resistance to Kernel Infection by *Fusarium moniliforme* in the Sweet Corn Inbred IL125b

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ABSTRACT

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Susceptible and resistant inbreds, F_1, F_2, F_3, and backcross generations from crosses of IL125b (resistant) and Ia2256b or IL783a (susceptible) were evaluated for incidence of symptomatic and asymptomatic infection of kernels by *Fusarium moniliforme*. The results of generation mean analysis indicated that additive and dominant gene actions accounted for most of the variability among generation means. Broad-sense heritability ranged from 0.24 to 0.46 for asymptomatic infection and from 0.32 to 0.55 for symptomatic infection. The estimated number of effective factors ranged from 3 to 12. Distributions of incidence classes for symptomatic and asymptomatic infection in F_2 and backcross families were continuous and highly skewed toward the resistant parent, IL125b. Resistance appeared to be controlled by several genes. A breeding method maximizing the accumulation of alleles for resistance, such as recurrent selection, would be effective in breeding for resistance to *F. moniliforme* kernel infection when using IL125b as a source of resistance.

Fusarium moniliforme J. Sheld. (teleomorph: *Gibberella fujikuroi* (Sawada) Ito in Ito & K. Kimura) commonly colonizes corn (*Zea mays* L.) throughout the world, and has been associated with ear rots, stalk rots, and seedling blights (9,18,20,29). Incidence of kernel infection can be 100% in some seed lots (19). Stalk rots and seedling blights can result from seedborne *F. moniliforme* (1,15,22,27). Interest in Fusarium ear rots has increased recently due to the isolation and characterization of fumonisins, mycotoxins with cancer-promoting activities, from corn and corn-based food and feed infected by *F. moniliforme* (16, 26,32).

Poor stands limit the use of certain sweet corn hybrids. Stand establishment problems have been particularly important in portions of the sweet corn industry where the *shrunken-2* (*sh2*) endosperm mutant with high sugar concentration and low kernel weight is used. Inbreds and hybrids with the *sh2* gene are considered to be relatively susceptible to seedling blight (3,11,31,33). Fungi often associated with seedling blight of sweet corn include seedborne *Fusarium* spp., *Penicillium* spp., and a *Rhizopus* sp., and soil-borne *Rhizoctonia solani* and *Pythium* spp. (2,3,5,11,30). Initially, the seedborne fungal problem in *sh2* corn was attributed primarily to *F. moniliforme* because this fungus was frequently isolated from seed of the elite *sh2* hybrids "Florida Sweet" and "Florida Staysweet," produced from inbreds Fa32 and Fa56 (34,35). Fa32, Fa56, and the *sugary-1* (*su*) lines from which they were derived, Ia2132 and Ia2256, are susceptible to kernel infection by *F. moniliforme* (11). The widespread use of elite *sh2* inbreds derived from Fa32 and Fa56 has increased the problem with *F. moniliforme*. Other seed- and soil-borne fungi also have been implicated (2,5). Improving resistance to ear rot due to *F. moniliforme* is an important breeding objective because it would help improve plant stands of *sh2* sweet corn and it would also improve the quality of corn-based food and feed.

Fusarium ear rot can be controlled by planting resistant hybrids. Smith and Madsen (29) reported differential responses of field corn inbreds following inoculation with *F. moniliforme*. Boling and Grogan (4) used generation mean analysis of a resistant × susceptible cross to determine that additive and dominance gene actions, and an additive × dominance digenic interaction, were important in the inheritance of resistance to *F. moniliforme* in a dent corn inbred, Mp 420. Approximately 1.3 effective factors were involved in the inheritance of the resistance. If the calculated number of gene pairs was a good estimate, they suggested that a backcross breeding method could be used to transfer genes for resistance to *F. moniliforme* (4). De León and Pandey (8) used a modified ear-to-row breeding program to improve resistance to Fusarium ear rots in tropical maize gene pools, with –0.9% progress per cycle (i.e., a decrease in ear rot). They concluded (8) that the inheritance of resistance to ear rot in the intermediate white dent and late yellow flint gene pools was polygenic with additive gene action. King and Scott (17) found that differences among inbreds C190C, Mp 317, and SC170 (resistant to kernel infection by *F. moniliforme*), and Mp68:616, Mp 303, and Mp 440 (susceptible to kernel infection by *F. moniliforme*), were expressed in their hybrids. Scott and King (28) later reported that resistance to kernel infection by *F. moniliforme* in the cross Mp 317 × SC170 was chiefly due to additive gene action, and the pericarp was the site of gene action, with endosperm, embryo, or cytoplasm having no genetic effect on resistance. This agreed with the results of Lunsford et al. (20) for seedling blight caused by *F. moniliforme* on corn inbreds GA172, GEC119A, SC155, and SC270P. Hoenisch and Davis (14) measured pericarp thickness of 12 commercial corn hybrids and found that pericarps were thinner for susceptible than resistant hybrids, allowing easier access of the fungus into kernels, especially through insect wounds. After four decades of investigations of resistance to Fusarium ear rot in corn, no corn line is immune to the disease and mechanisms of resistance are not well understood (7).

Unlike field corn, sweet corn has been the subject of only a limited number of studies on resistance to *F. moniliforme*. Headrick and Pataky (11) found different responses among 138 sweet corn inbreds evaluated for reaction to kernel infection by *F. moniliforme*. The reactions were strongly affected by factors operative in maternal tissues such as silk and pericarp (12). Incidence of infection was lowest for lines with silks that grew actively for several days after pollination (12). Conversely, Reid et al. (25) did not observe consistent relationships between resistance to *F. graminearum* and the rate of silk senescence, as the resistant field corn inbred, CO272, and the susceptible field corn inbred, CO266, had the same rate of silk senescence. Headrick and Pataky (11) and Headrick et al. (13) identified an *su* inbred, IL125b, that had the green silk characteristic, consistently lower incidences of symptomatic and asymptomatic infection of kernels by *F. moniliforme*, and good emergence in 3 years of trials. This line has been used by commercial sweet corn

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breeders as a source of resistance to common rust, caused by *Puccinia sorghi* Schwein. (24). Additional information about the inheritance and mechanisms of resistance to kernel infection by *F. moniliforme* in IL125b may enhance its effective use.

The objective of these studies was to evaluate resistance to *F. moniliforme* in IL125b.

MATERIALS AND METHODS

Parents and crosses. IL125b, a sweet corn inbred resistant to kernel infection by *F. moniliforme*, and two inbreds susceptible to kernel infection by *F. moniliforme*, Ia2256b and IL783a, were used as resistant (Pr) and susceptible (Ps) parents. IL125b and Ia2256b are *su* endosperm mutants. IL783a is an *sh2* inbred selfed from Florida Sweet. The following generations were produced by hand pollination in 1989 when IL783a was the susceptible parent, and in 1992 and 1993 when Ia2256b was the susceptible parent: Pr, Ps, Pr × Ps, (Pr × Ps)F_2, (Pr × Ps)F_3, (Pr × Ps) × Pr, (Pr × Ps) × Ps, and their reciprocals. Seed produced in these experiments were one generation advanced from the plant on which they were produced. For example, F_1 hybrid seed were produced on inbreds (Ps or Pr), F_2 kernels were produced on F_1 plants (Ps × Pr or Pr × Ps), etc. Thus, F_1 kernels had maternal tissues (silk, pericarp, placento-chalazal region) from the ear parent, whereas, F_2 and BC (backcross) kernels received a maternal component from each inbred parent. Throughout this article, we use generation of the ear parents to refer to the genotype of the plant on which the seed was produced (i.e., genotype of maternal tissues) and generation of kernels to refer to nonmaternal tissues of kernels.

Trials in 1993. Two experiments in 1993 included IL125b and Ia2256b. The first experiment included three generations of ear parents: Pr, Ps; Pr × Ps, Ps × Pr; (Pr × Ps)F_2, and (Ps × Pr)F_2. All plants were pollinated by hand to produce four generations of kernels (P, F_1, F_2, and F_3) and backcrosses (Table 1). The experimental design was a randomized complete block with two replicates of all generations of kernels including backcrosses. An experimental unit included 30 plants in a two-row plot. Rows were spaced 76 cm apart. The second experiment included the three generations of ear parents listed above and two backcross ear parents: (Ps × Pr) × Pr and (Ps × Pr) × Ps. The experimental design, experimental units, and replicates were the same as those in the first experiment except that all plants were open-pollinated in the second experiment. Both experiments were planted 26 May 1993.

F. moniliforme was isolated from field-infected maize kernels and confirmed by P. E. Nelson, Pennsylvania State University, University Park. Isolates were maintained at –80°C in a 15% glycerol solution. Inoculum was produced from a mixture of eight isolates.

Cultures (5 to 7 days old) grown on potato dextrose agar in petri plates were blended in water. Concentration of inoculum was measured with a hemacytometer and adjusted to approximately 10^6 to 10^8 propagules per ml. Plants were inoculated twice, 10 and 24 days after pollination. Approximately 5 ml of inoculum was sprayed onto silks of each plant. Tassel bags were removed momentarily from hand-pollinated ears in order to inoculate. Inoculated ears then were covered with a plastic bag that was removed after 48 h. Tassel bags remained on hand-pollinated ears until harvest.

Trials in 1994. Two experiments in 1994 included IL125b and both susceptible parents, Ia2256b and IL783a. In the first experiment, four generations (P, F_1, F_2, and F_3) and backcrosses of the cross IL125b × Ia2256b were used as ear parents (Table 1). All plants were hand-pollinated to produce five generations of kernels (P, F_1, F_2, F_3, and F_4) and backcrosses (Table 1). The experimental design was a randomized complete block with two replicates of generations of kernels. An experimental unit included six rows spaced 76 cm apart with 15 plants per row. The experiment was planted 26 May 1994. Eleven isolates of *F. moniliforme* were used for inoculum. Plants were inoculated once 10 days after pollination following the same procedures as in 1993 except that plastic bags were removed 7 days after inoculation.

The second experiment included parental, reciprocal F_2 and F_3, and backcross ear parents of the cross of IL125b × IL783a (Table 2). The experimental design, planting date, and inoculation procedures were the same as the first experiment of 1994.

Disease assessment and data analysis. Ears were harvested by hand 35 to 40 days after inoculation each year. Each experimental unit was kept separately. Ears were dried with ambient forced-air prior to shelling kernels from the cob.

Kernels from individual ears were evaluated for incidence of symptomatic and asymptomatic infection. Symptomatic

Table 1. Incidence (%) of *Fusarium moniliforme*–infected kernels in 1993 and 1994 for different generations of kernels from the cross between the resistant parent IL125b and the susceptible parent Ia2256b

	1993[t]		1994	
Ear parent and crosses[u]	**Symptomatic infection[v]**	**Asymptomatic infection[w]**	**Symptomatic infection[v]**	**Asymptomatic infection[w]**
Pr ear parent				
Pr	2.6 d[x]	8.7 fg[x]	4.6 defg	19.3 efg
Pr × Ps	3.1 d	16.4 de	5.9 cd	24.4 def
Pr × (Pr × Ps)	3.1 d	22.0 cd	5.8 cde	21.3 def
Pr × (Ps × Pr)	3.0 d	19.5 cde	7.8 cd	25.0 def
Ps ear parent				
Ps	18.3 b	26.6 c	45.3 a	56.3 c
Ps × Pr	. . .	. . .	8.8 c	28.3 de
Ps × (Pr × Ps)	29.1 a	53.9 a	32.2 b	87.8 a
Ps × (Ps × Pr)	31.4 a	36.9 b	33.3 b	69.8 b
(Pr × Ps) ear parent				
(Pr × Ps) × Pr	6.3 cd	23.8 cd	1.2 gh	5.3 ij
(Pr × Ps) × Ps	5.2 cd	17.5 de	2.4 efgh	7.1 ij
(Ps × Pr) ear parent				
(Ps × Pr) × Pr	5.9 cd	17.6 de	0.4 h	4.4 j
(Ps × Pr) × Ps	1.5 d	8.2 g	1.8 fgh	6.8 ij
(Pr × Ps) F_2	1.2 d	7.3 g	1.6 fgh	7.3 ij
(Ps × Pr) F_2	2.5 d	12.6 efg	4.6 def	12.1 ghi
Other ear parents[y]				
(Pr × Ps) F_3	5.5 cd	23.3 cd	4.8 defg	11.2 hij
(Ps × Pr) F_3	9.8 c	21.9 cd	4.4 defg	18.3 fgh
(Pr × Ps) F_4	. . .	. . .	6.5 cd	25.8 def
(Ps × Pr) F_4	. . .	. . .	4.8 defg	14.3 ghi
(Pr × Ps) × Pr ⊗	. . .	. . .	3.4 defg	10.2 hij
(Pr × Ps) × Ps ⊗	. . .	. . .	8.1 cd	44.5 c
(Ps × Pr) × Pr ⊗	. . .	. . .	2.0 efgh	12.5 ghi
(Ps × Pr) × Ps ⊗	. . .	. . .	5.2 defg	32.4 d
Grand mean	6.7	18.7	6.0	19.6
Standard error	0.5	0.7	0.4	0.7
Midparent[z]	10.5	17.7	25.0	37.8

[t] Data in 1993 from hand-pollinated trial only.
[u] Pr = IL125b, Ps = Ia2256b
[v] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[w] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[x] Numbers followed by the same letter do not differ significantly at $P = 0.05$ according to Fisher's least significant difference comparisons of double square-root transformed data.
[y] Other ear parents include F_2, F_3, BCr, and BCs.
[z] Midparent = (Pr + Ps) / 2.

infection was assessed by counting the number of kernels exhibiting signs or symptoms of fungal infection from a 100-kernel sample taken at random from each ear. Asymptomatic kernels, those with no signs or symptoms of infection or insect damage, were sorted from each ear, and a sample of 50 asymptomatic kernels was assayed. Samples were surface sterilized by soaking for 2 min in 90% ethanol and 2 min in 10% commercial Clorox, rinsed twice with sterile distilled water, and plated, 10 kernels per plate, on a *Fusarium*-selective medium containing pentachloronitrobenzene (PCNB) (23). Incidence of fungal growth from asymptomatic kernels was recorded 5 to 7 days after plating.

Since the endosperm and embryo of seed are one generation advanced from the plant on which they are produced and have a different genotype than the ear parent plant, two groups of generations were analyzed: the generations of the ear parent (maternal tissues of seed), and the generations of kernels (embryo and endosperm). In 1993, when data were collected from trials with hand-pollinated and open-pollinated plants, data from both trials were used in the analyses of generations of the ear parents, but only the data from the hand-pollinated trial was used in the analyses of generations of kernels. Data were subjected to analysis of variance using the appropriate procedures of SAS (Statistical Analysis System, SAS Institute, Cary, NC). Double square-root transformations were used to create homogeneous variances. Fisher's least significant difference (LSD) values were used to test significance of differences among means of generations. Correlations between symptomatic and asymptomatic infection of kernels were calculated using data from each experimental unit, and phenotypic correlations were computed from means of generations.

The notation and procedures of Hayman (10) were used in a generation means analysis for generations of ear parents. Increasingly complex models were fitted by least squares regression to the generation means: $Y_k = m + \alpha_k d + \beta_k h + \alpha_k^2 i + 2\alpha_k\beta_k j + \beta_k^2 l + e_k$ where Y_k is the mean of the k^{th} generation, m is the mean of the F_2 generation, α_k and β_k are coefficients determined by the degree of genetic relationship of the k^{th} generation, d is the pooled additive genetic effects, h is the pooled dominance genetic effects, i is the pooled additive × additive effects, j is the pooled additive × dominance effects, and l is the pooled dominance × dominance effects. The coefficients appropriate for the generations of this study are given by Hayman (10). The results of the regression analyses are presented in terms of the proportion of the corrected regression sum of squares for generations due to additive effects (d) alone, due to dominance effects (h) after adjusting for d, and due to digenic epistatic effects after adjusting for d and h.

Broad-sense heritability (h^2) estimates in the F_2 generation of the crosses IL125b × Ia2256b and IL125b × IL783a were calculated by the formula $(\sigma^2_p - \sigma^2_e) / \sigma^2_p$, in which σ^2_p is the phenotypic variance of individuals in the F_2 generation and σ^2_e is the environmental variance between individuals of the same genotype, estimated by pooling the within-plot variances of the inbred parents and the F_1 generation. Estimates of the minimum number of effective factors (k) controlling resistance were calculated according to the Castle-Wright formula as modified by Mather and Jinks (21) in which $k = (\text{phenotypic range in } F_2)^2 / 8 \times (\sigma^2_p - \sigma^2_e)$.

RESULTS

Incidence of infection in generations of kernels. In the generations of kernels of IL125b × Ia2256b, the Pr and Ps differed by 16 and 18% in 1993 and by 39 and 37% in 1994, for symptomatic and asymptomatic infection, respectively (Table 1). Seed produced on the resistant parent, i.e., Pr, Pr × Ps, Pr × (Pr × Ps), and Pr × (Ps × Pr), had equally low incidence of symptomatic and asymptomatic infection of kernels (Table 1), except for a lower amount of asymptomatic infection of the Pr in 1993. Seed produced on the susceptible parent, i.e., Ps, Ps × Pr, Ps × (Pr × Ps), and Ps × (Ps × Pr), had higher incidences of symptomatic and asymptomatic infection than that produced on the Pr except for Ps × Pr in 1994 (Table 1).

For the IL125b × IL783a cross, seed produced on the resistant parent had equally low incidences of symptomatic and asymptomatic infection of kernels (Table 2). Seed produced on the susceptible parent had the highest incidences of symptomatic and asymptomatic infection of kernels although each generation was not equal in reaction to kernel infection (Table 2). Within the F_2, F_3, and F_4 generations, reciprocals did not differ from each other for symptomatic or asymptomatic infection except for a lower incidence of asymptomatic infection in the (Ps × Pr) F_4 than in the (Pr × Ps) F_4 of IL125b × Ia2256b in 1994 (Tables 1, 2).

The backcrosses of IL125b × Ia2256b did not differ significantly for incidence of kernel infection when the Pr or Ps inbreds were used as the pollen source except for

Table 2. Incidence (%) of *Fusarium. moniliforme*–infected kernels in 1994 for different generations of kernels from the cross between the resistant parent IL125b and the susceptible parent IL783a

Ear parent and crosses[u]	Symptomatic infection of kernels[v]	Asymptomatic infection of kernels[w]
Pr ear parent		
Pr	4.6 efgh[x]	19.3 ghi[x]
Pr × Ps	1.9 h	5.7 i
Pr × [(Pr × Ps) × Pr]	4.3 fgh	18.2 ghi
Pr × [(Ps × Pr) × Pr]	4.5 efgh	17.2 ghi
Ps ear parent		
Ps	70.9 a	73.8 b
Ps × Pr	44.6 b	66.0 bc
Ps × [(Pr × Ps) × Ps]	72.6 a	51.7 cde
Ps × [(Ps × Pr) × Ps]	75.0 a	100.0 a
(Pr × Ps) × Pr ear parent		
(Pr × Ps) × Pr ⊗	4.7 efgh	14.9 hi
[(Pr × Ps) × Pr] × Pr	2.2 gh	12.0 hi
(Pr × Ps) × Ps ear parent		
(Pr × Ps) × Ps ⊗	26.4 c	42.9 def
[(Pr × Ps) × Ps] × Ps	26.4 c	55.0 bcd
(Ps × Pr) × Pr ear parent		
(Ps × Pr) × Pr ⊗	3.3 fgh	13.4 hi
[(Ps × Pr) × Pr] × Pr	1.3 h	6.2 i
(Ps × Pr) × Ps ear parent		
(Ps × Pr) × Ps ⊗	15.2 de	50.5 cde
[(Ps × Pr) × Ps] × Ps	20.6 cd	54.4 bcd
Other ear parents[y]		
(Pr × Ps) F_3	7.5 efgh	28.8 fgh
(Ps × Pr) F_3	10.5 defgh	26.9 fghi
(Pr × Ps) F_4	13.1 def	32.0 efgh
(Ps × Pr) F_4	12.9 defg	37.3 defg
Grand mean	11.3	26.9
Standard error	0.7	1.0
Midparent[z]	37.8	46.6

[u] Pr = IL125b, Ps = IL783a.
[v] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme.*
[w] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[x] Numbers followed by the same letter do not differ significantly at $P = 0.05$ according to Fisher's least significant difference comparisons of double square-root transformed data.
[y] Other ear parents include F_2 and F_3.
[z] Midparent = (Pr + Ps) / 2.

less asymptomatic infection of the (Ps × Pr) × Ps in 1993 (Table 1). For the backcrosses of IL125b × IL783a with IL125b as the pollen parent, symptomatic and asymptomatic infection ranged from 1 to 5% and from 6 to 15%, respectively, whereas for the backcrosses with IL783a as the pollen parent, symptomatic and asymptomatic infection ranged from 15 to 26% and 43 to 55%, respectively (Table 2). Similarly, selfed ears of (Pr × Ps) × Pr had a significantly lower incidence of kernel infection than selfed ears of (Pr × Ps) × Ps (Table 2).

Incidence of infection in generations of ear parents. In the generations of ear parents of IL125b × Ia2256b, the Pr and Ps differed by 17 and 25% in 1993, and by 28 and 49% in 1994, respectively, for symptomatic and asymptomatic infection (Table 3). Incidence of kernel infection did not differ for Pr, Pr × Ps, and Ps × Pr in 1993; but the Pr had a significantly higher incidence of asymptomatic infection of kernels than Pr × Ps and Ps × Pr in 1994 (Table 3). Symptomatic and asymptomatic infection of the Ps ranged from 21 to 33% and from 43 to 69% whereas symptomatic and asymptomatic infection of Ps × Pr ranged from 2 to 4% and from 8 to 14%, respectively (Table 3). In backcrosses with IL125b as the pollen parent in 1993 and 1994, incidence of symptomatic and asymptomatic infection of kernels ranged from 2 to 3% and from 10 to 13%, respectively, whereas for two backcrosses with Ia2256b as the pollen parent, incidence of symptomatic and asymptomatic infection ranged from 5 to 8% and from 32 to 45%, respectively (Table 3). Incidence of symptomatic and asymptomatic infection was lower (6 and 23% in 1993, and 5 and 11% in 1994, respectively) for (Pr × Ps) F_2 than for (Ps × Pr) F_2 (10 and 32% in 1993, and 4 and 18% in 1994, respectively) (Table 3). In the F_3 families, (Pr × Ps) F_3 and (Ps × Pr) F_3 were not different for incidence of symptomatic infection, but asymptomatic infection was higher for (Pr × Ps) F_3 than for (Ps × Pr) F_3.

In the IL125b × Il783a cross, the two parents differed by 60 and 48% for symptomatic and asymptomatic infection of kernels, respectively (Table 4). Incidence of symptomatic and asymptomatic infection of kernels in the backcross generations with IL125b as the pollen parent ranged from 3 to 4% and from 11 to 14%, respectively, whereas in the backcross generations with IL783a as the pollen parent, symptomatic and asymptomatic infection of kernels ranged from 18 to 26% and from 49 to 53%, respectively (Table 4). The reciprocal F_2 and F_3 families of IL125b × IL783a did not differ in their reactions to kernel infection.

Correlation of symptomatic and asymptomatic infection. Correlations between the incidence of symptomatic and asymptomatic infection of kernels for both populations (IL125b × Ia2256b and IL125b × IL783a) in both years were moderate, ranging from 0.40 to 0.58 when data from experimental units were used. Phenotypic correlations of generations were higher, ranging from 0.87 to 0.99.

F_1 hybrid and midparent values. In both categories of generations (ear parents and kernels), in the IL25b × Ia2256b population, Pr × Ps and Ps × Pr had lower incidences of symptomatic and asymptomatic infection of kernels than the midparent values (Tables 1, 3). In the generation of kernels of IL125b × IL783a population, incidences of symptomatic and asymptomatic infection for Pr × Ps were 35 and 40% lower than the midparent values, whereas Ps × Pr exceeded the midparent values by 7 and 21%, respectively (Table 2).

Generation mean analyses. From the generation mean analyses, the pooled additive effects and the pooled dominance effects for the two populations studied were significant for both symptomatic and asymptomatic infection of kernels (Tables 5, 6). Significant additive × additive, additive × dominance, and dominance × dominance epistatic effects for symptomatic infection were detected in the cross IL125b × Ia2256b in 1994 (Table 5). The additive × additive epistatic effects for symptomatic infection and the additive × dominance epistatic effects for asymptomatic infection were detected in the cross IL125b × IL783a (Table 6).

For the population IL125b × Ia2256b, additive and dominance genetic effects accounted for 54 and 33%, and for 66 and 23% of the variability among generation means, of symptomatic and asymptomatic infections of kernels, respectively, in 1993 (Table 5). In 1994, additive and dominance genetic effects accounted for 41 and 48%, and for 45 and 51% of the variability among generation means, of symptomatic and asymptomatic infection of kernel, respectively (Table 5). The digenic epistatic effects accounted for 0 to 10%, and for 0 to 7% of the variability in generation means, of kernel infection in 1993 and 1994, respectively.

For the population IL125b × IL783a, additive and dominance genetic effects

Table 3. Incidence (%) of *Fusarium moniliforme*-infected kernels for differents generations of ear parents from the cross between the resistant parent IL125b and the susceptible parent Ia2256b

	1993		1994	
Ear parent[v]	Symptomatic infection[w]	Asymptomatic infection[x]	Symptomatic infection[w]	Asymptomatic infection[x]
Pr	3.7 cd[y]	17.7 d[y]	5.2 bcd[y]	19.7 de[y]
Ps	21.3 a	43.3 a	32.8 a	69.1 a
Pr × Ps	4.6 cd	16.9 d	1.9 d	6.7 i
Ps × Pr	3.8 cd	13.6 de	2.2 d	7.6 hi
(Pr × Ps) F_2	5.5 c	23.3 c	4.8 cd	11.2 ghi
(Ps × Pr) F_2	9.9 b	31.5 b	4.4 cd	18.3 ef
(Pr × Ps) F_3	...	...	6.5 bc	25.8 cd
(Ps × Pr) F_3	...	...	4.8 cd	14.3 fg
(Pr × Ps) × Pr	...	...	3.4 cd	10.2 ghi
(Pr × Ps) × Ps	...	...	8.1 b	44.5 b
(Ps × Pr) × Pr	2.2 d	11.1 e	2.0 d	12.5 fgh
(Ps × Pr) × Ps	6.3 c	31.5 b	5.2 bcd	32.4 c
Grand mean	7.0	22.5	5.7	19.1
Standard error	0.4	0.7	0.4	0.7
Midparent[z]	12.5	30.5	19.0	44.4

[v] Pr = IL125b, Ps = Ia2256b
[w] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[x] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[y] Numbers followed by the same letter do not differ significantly at $P = 0.05$ according to Fisher's least significant difference comparisons of double square-root transformed data.
[z] Midparent = (Pr + Ps) / 2.

Table 4. Incidence (%) of *Fusarium moniliforme*–infected kernels for different generations of ear parents from the cross between the resistant parent IL125b and the susceptible parent IL783a in 1994

Ear parent[v]	Symptomatic infection[w]	Asymptomatic infection[x]
Pr	5.2 e[y]	19.7 ef[y]
Ps	65.4 a	68.2 a
(Pr × Ps) F_2	7.5 e	28.8 de
(Ps × Pr) F_2	10.5 d	26.9 cde
(Pr × Ps) F_3	13.2 cd	31.9 cd
(Ps × Pr) F_3	12.9 d	37.3 c
(Pr × Ps) × Pr	4.1 e	14.2 fg
(Pr × Ps) × Ps	26.4 b	48.8 b
(Ps × Pr) × Pr	2.6 e	10.9 g
(Ps × Pr) × Ps	18.3 c	52.7 b
Grand mean	10.4	26.2
Standard error	0.7	1.0
Midparent[z]	35.3	44.0

[v] Pr = IL125b, Ps = IL783a.
[w] Percentage of kernels exhibiting signs or symptoms of infection by *F. moniliforme*.
[x] Percentage of healthy appearing kernels exhibiting growth of *F. moniliforme* on medium containing pentachloronitrobenzene.
[y] Numbers followed by the same letter do not differ significantly at $P = 0.05$ according to Fisher's least significant difference comparisons of double square-root transformed data.
[z] Midparent = (Pr + Ps) / 2.

accounted for 79 and 18%, and for 91 and 5% of the variability among generation means, of symptomatic and asymptomatic infection of kernels, respectively (Table 6). The digenic epistatic effects accounted for 0 to 4% of the variability among generation means.

Estimates of broad-sense heritability ranged from 0.24 to 0.46 for asymptomatic infection and from 0.32 to 0.55 for symptomatic infection (Table 7). Estimates of number of effective factors ranged from approximately 3 to 12.

DISCUSSION

Resistance to kernel infection by *F. moniliforme* was expressed in maternal tissues of kernels from crosses of IL125b and Ia2256b or IL783a with significant additive, dominance, and digenic epistatic effects detected.. Additive and dominance effects were of primary importance in the IL125b × Ia2256b population. In the I25b × IL783a population, the additive effect was most important. Significant deviations of F_1 means (generations of ear parents) from the midparent values toward lower incidences of symptomatic and asymptomatic kernel infection constituted additional evidence of dominance in the IL125b × Ia2256b population. These data agree with previous findings (4,12,28) that additive and dominance genetic effects were of primary importance in the inheritance of resistance to *F. moniliforme*.

Assumptions made in using generation mean analysis include the isodirectional distribution of genes between the two parental lines, the absence of linkage between interacting loci, and the absence of selection favoring certain gametes (21). The lack of genes being isodirectionally distributed among the parents would result in significant deviations from the simple additive dominance genetic model (6). Our data showed significant epistatic effects (Tables 5, 6), indicating possible lack of isodirectional distribution of genes among the parents. Therefore, only broad-sense heritabilities were estimated. The lack of isodirectional distribution of genes was also taken into account in modifying the Castle-Wright formula for the estimation of effective factors (6), which ranged from 3 to 12. The formula used to calculate these estimates assumes no epistasis; consequently, they were probably biased downward. Heritability was low to moderate, ranging between 0.24 and 0.55. Since additive and dominance genetic effects were of primary importance, the most effective approach to incorporate this resistance into sweet corn inbreds appears to be a recurrent selection breeding procedure, which maximizes the accumulation of alleles for resistance. De León and Pandey (8) used recurrent selection to improve resistance to Fusarium ear rot in two tropical maize gene pools. The inbred IL125b has been used as a source of the gene *Rp1d,* which has been incorporated into elite sweet corn inbreds through backcrossing. Simultaneous improvement for resistance to *F. moniliforme* would have been difficult during these backcross breeding programs even with inoculation and selection.

Assuming a single nuclear gene difference between resistant and susceptible parents, Scott and King (28) showed that the genotypes of the pericarp were the same for all crosses using the resistant inbred as the ear parent. If the genotype of the pericarp affects the incidence of kernel infection by *F. moniliforme*, the generations of kernels produced on the resistant parent should have low amounts of infected kernels. Likewise, incidence of kernel infection should be high for generations produced on the susceptible parent. Incidence of symptomatic and asymptomatic infection of kernels was highest when the ear parent was the susceptible parent regardless of the generation of the kernels except for some generations of IL125b × Ia2256b produced on Ia2256b. Similarly, the maternal tissues of the backcross generations with either parent as the pollen source had the same genotype and had similar amounts of kernel infection. These data would suggest an influence of maternal tissues, which agrees with Scott and King (28) and Headrick et al. (12) that the genotype of the ear parent affects the incidence of kernel infection by *F. moniliforme*.

The reciprocal F_1 crosses did not differ in incidence of kernel infection in the ear parent generations of the IL125b × Ia2256b population but differed in the

Table 5. Percent variation (R^2) among generation means of the IL125b × Ia2256b population accounted for by fitting genetic effects

Year and gene effects	Symptomatic infection	Asymptomatic infection
1993		
Total R^2 (model)[y]	59.0	70.0
Additive	54**[z]	66***
Dominance	33**	23*
Additive × additive	3	4
Additive × dominance	0	4
Dominance × dominance	10	3
1994		
Total R^2 (model)	98.7	94.1
Additive	41***	45***
Dominance	48***	51***
Additive × additive	7***	3
Additive × dominance	3***	1
Dominance × dominance	1*	0

[y] Model $Y_k = m + \alpha_k d + \beta_k h + \alpha^2_k i + 2\alpha_k\beta_k j + \beta^2_k l + e_k$. (See text.)
[z] *, **, *** indicate levels of probability (0.05, 0.01, or 0.001) at which the mean square was significant.

Table 6. Percent variation (R^2) among generation means of IL125b × IL783a population accounted for by fitting genetic effects

Gene effects	Symptomatic infection	Asymptomatic infection
Total R^2 (model)[y]	98.5	97.8
Additive	79***[z]	91***
Dominance	18***	5*
Additive × additive	2*	0
Additive × dominance	1	4*
Dominance × dominance	0	0

[y] Model $Y_k = m + \alpha_k d + \beta_k h + \alpha^2_k i + 2\alpha_k\beta_k j + \beta^2_k l + e_k$. (See text.)
[z] *, **, *** indicate levels of probability (0.05, 0.01, or 0.001) at which the mean square was significant.

Table 7. Estimates of broad-sense heritability (h^2) and number of effective factors (k) involved in the resistance to symptomatic and asymptomatic infection of kernels by *Fusarium moniliforme* in two populations of sweet corn

		Broad-sense heritability (h^2)[y]		Effective factors (k)[z]	
Population	Year	Symptomatic infection	Asymptomatic infection	Symptomatic infection	Asymptomatic infection
IL125b × Ia2256	1993	0.55	0.46	5.0	3.1
	1994	0.32	0.30	12.0	6.6
IL125b × IL783a	1994	0.34	0.24	7.8	6.9

[y] Broad-sense heritability (h^2) estimated by the formula $h^2 = [(\sigma^2_{F2} - \sigma^2_e) / \sigma^2_{F2}]$.
[z] Number of effective factors (k) estimated by the formula k = (phenotypic range in F_2)^2 / 8 × ($\sigma^2_{F2} - \sigma^2_e$).

generation of kernels in the IL125b × IL783a population, as expected. These crosses were not available to test in the generation of ear parents for the IL125b × IL783a population. When backcrosses were ear parents, the BCs (i.e., [Pr × Ps] × Ps or [Ps × Pr] × Ps) had significantly greater kernel infection than the BCr. This difference was greater in the IL125b × IL783a population than in the IL125b × Ia2256b population, possibly because the *sh2* endosperm mutation in IL783a also affected the integrity of the pericarp. Near-isolines of IL125b for the *su* and *sh2* endosperm mutation would be useful to evaluate further which maternal tissues are responsible for resistance to *F. moniliforme* and to determine the effect of *sh2* on the susceptibility kernels to *F. moniliforme*.

Phenotypic correlations between symptomatic and asymptomatic infection of kernels based on means of generations were high (r = 0.87 to 0.99), while correlations based on data from experimental units were moderate (r = 0.40 to 0.58). Thus, variation associated with experimental units was relatively high for these two variables.

Resistance to kernel infection by *F. moniliforme* from IL125b or from other sources of resistance should be incorporated into the Fa32-derived *sh2* lines and other susceptible, elite sweet corn germ plasm. However, our study indicates that while IL125b is highly resistant, incorporation of that resistance will require a breeding method, such as recurrent selection, to maximize the accumulation of alleles for resistance.

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