Resistance to Kernel Infection by *Fusarium moniliforme* in Inbred Lines of Sweet Corn and the Effect of Infection on Emergence

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ABSTRACT

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Kernels from silk-inoculated and noninoculated sweet corn ears were evaluated for incidence of symptomatic infection of kernels by *Fusarium moniliforme* (i.e., the percentage of kernels showing signs or symptoms of *F. moniliforme*), incidence of asymptomatic infection of kernels, and emergence. A total of 138 inbreds were evaluated in at least 1 yr, and 17 were evaluated in each of 3 yr. Emergence was reduced by kernel infection in many inbreds but was dependent on genotype and may have been influenced by environmental stress. Sources of partial resistance to kernel infection by *F. moniliforme* were identified among sweet corn inbred lines that showed good emergence. On the basis of our data, kernel infection by *F. moniliforme* appears to reduce emergence in particular genotypes under given environmental conditions, although physiological constraints on vigor of seedlings may be of greater importance than *F. moniliforme* in the emergence and performance of sweet corn hybrid seedlings.

Poor emergence and vigor of seedlings are major concerns of the sweet corn (*Zea mays* L.) industry, especially among hybrids with the *shrunken-2* (*sh2*) endosperm mutation that results in high amounts of sugars in kernels (4,27,28,31,35). Fungal pathogens (2,5,8,27), environment (25,35), leakage of electrolytes (26,32,35), carbohydrate metabolism (17,28), and morphology of kernels (1) are factors affecting performance of sweet corn seedlings. The role of fungal pathogens has not been delimited clearly, in part because pathogenesis may be affected by the other factors mentioned.

Although *Fusarium moniliforme* Sheldon (teleomorph: *Gibberella fujikuroi* (Sawada) Wollenw.) is the fungus most commonly isolated from corn seed of all endosperm types, its pathogenicity has been debated (14,27). *F. moniliforme* is a cosmopolitan species that grows well saprophytically and survives in debris but lacks chlamydospores and does not survive for long periods in soil (19,34,36). It does, however, survive well in corn kernels (16,27,29,30).

Kernel infection by *F. moniliforme* reportedly results from airborne conidia that germinate on and grow down corn silks to infect the ear (10,12,33). Infected kernels may become molded in an irregular pattern beginning at the tip of the ear or they may be asymptomatic. Molded kernels often are not germinable. Infected, asymptomatic kernels usually are germinable but may die before emergence or produce blighted seedlings in stressful environments.

Because of the cosmopolitan nature of *F. moniliforme* and its ability to survive in seed and debris, crop rotation and chemical control generally have been ineffective. Genetic resistance offers the greatest potential for control, but sources of resistance in sweet corn backgrounds and the inheritance of this resistance have not been examined.

Progress has been made in breeding for resistance to *F. moniliforme* in dent corn germ plasm. Much of this initial work concentrated on screening inbred lines (6,13,24). More recently, Warren (33) demonstrated that high-lysine inbred lines generally were more susceptible to kernel rot caused by *F. moniliforme* than normal lines, but reactions were dependent on the genotype background. King and Scott (11) observed differences in asymptomatic infection of kernels among inbreds that subsequently were expressed in their hybrids. Scott and King (23) determined that resistance to *F. moniliforme* was conditioned by the genotype of the pericarp, whereas the genotype of the endosperm, embryo, or cytoplasm had little effect on the incidence of *F. moniliforme* in kernels. They hypothesized that the silk could be the actual site of gene action, if the mode of entry of the fungus was through silks. Foley (7) and Lawrence et al (15) have reported that ears may become infected through the shank by systemic invasion of *F. moniliforme*. Systemic infection occasionally may be important, although the results of others (10,12,33) and our preliminary observations (9) suggest that entry through the silks may be of greater epidemiological significance.

In examining the effects of *F. moniliforme* on the emergence of corn seedlings, Valleau (30) found the fungus to be borne internally and observed many symptomless, infected kernels. He also discovered that many infected kernels

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germinated well, but the resultant seedlings often showed stunted, necrotic roots. Thomas and Buddenhagen (29) isolated *F. moniliforme* from 78% of symptomless kernels and observed that asymptomatic infection of kernels was a source of inoculum for seedling blight and stalk rot. Other investigators have questioned the effect of seedborne *F. moniliforme* on germination and emergence (14,20,27). While most of these studies have evaluated dent corn, which is dominant at the *shrunken-2* (*Sh2*), *sugary-1* (*Su*), and *sugary enhancer* (*Se*) loci, the effect of seedborne *F. moniliforme* on the field emergence of sweet corn germ plasm (*sh2, su*, or *se*) has not been reported.

This study was designed to identify sources of resistance to kernel infection by *F. moniliforme* in sweet corn inbred lines and to determine the effect of infection on emergence of seedlings.

MATERIALS AND METHODS

All studies were done at the Agronomy/Plant Pathology South Farm, Urbana-Champaign, Illinois. In 1986, 115 inbred lines were planted on 9 May. These were primarily *sugary* (*su*) and *sugary enhancer* (*se*) lines developed in the Department of Horticulture, University of Illinois. A few *shrunken-2* (*sh2*) and dent (*Su*) lines were included, plus lines received from Charles Boyer of Pennsylvania State University, William Tracy of the University of Wisconsin, Vern Gracen of Cornell University, and Gene Scott of Mississippi State University. The experimental design was a randomized complete block with two replicates. Plots consisted of 15 plants in single rows spaced 76 cm apart. Within each row, eight plants were self-pollinated, four of which were silk-inoculated with *F. moniliforme*. The four remaining self-pollinated plants served as noninoculated controls.

Plants were inoculated twice with a microconidial suspension of *F. moniliforme*, 4–7 days 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 distilled water and rubbed with a glass rod to dislodge conidia. The inoculum concentration was determined with a hemacytometer and adjusted to approximately 10^6 microconidia per milliliter. The inoculum was a mixture of 12 isolates collected from corn the previous year. Each isolate was derived from a single spore to allow accurate species identification (3,18). Approximately 5 ml of the inoculum suspension was sprayed onto corn silks on each plant after the tassel bag had been removed from a self-pollinated ear. The inoculated ears then were covered with a plastic freezer bag and re-covered with the tassel bag. Plastic bags were removed 48 hr later, and the tassel bags were left on the ears until harvest. Tassle bags also were left on noninoculated ears from pollination until harvest.

In 1987, 49 inbred lines selected for evaluation were planted on 13 May. Experimental design and inoculation techniques were the same as those used in 1986 except that 17 different isolates of *F. moniliforme* were used as inoculum. In 1988, 20 inbred lines were planted on 17 May with four replicates, and 13 different isolates of *F. moniliforme* were used as inoculum. The same inoculation procedures were used. Thus, in the course of 3 yr, a total of 42 isolates of *F. moniliforme* were used to evaluate a total of 138 inbred lines in at least 1 yr. Seventeen of the inbred lines were evaluated in all 3 yr.

In each year, the four inoculated and the four noninoculated ears were hand-harvested 3–4 wk after the second inoculation. Harvested ears were dried with ambient forced air before shelling. Inoculated and noninoculated ears from each row were bulked by inoculation treatment. Kernels were evaluated for incidence of symptomatic infection, incidence of asymptomatic infection, and emergence in the following year.

For determination of incidence of symptomatic infection, 100-kernel samples were taken at random from inoculated and noninoculated samples in each plot. The number of kernels showing signs or symptoms of *F. moniliforme* were counted. Incidence of symptomatic infection then was converted to percentage. The incidence of asymptomatic infection was based on the number of healthy appearing kernels (those with no signs or symptoms of infection or insect damage) from which the fungus was isolated. In 1986 and 1987, 20 healthy appearing kernels of each inbred for each replication were plated—10 from inoculated ears and 10 from noninoculated ears. In 1988, the sample size was doubled. Kernels to be plated were surface-sterilized by soaking for 2 min each in 0.8% sodium hypochlorite and 90% ethanol followed by two rinses in sterile distilled water. Kernels were plated on PDA amended with tetracycline and streptomycin and on a *Fusarium*-selective medium containing pentachloronitrobenzene (PCNB) (21). Incidence of fungi and germination of kernels were recorded 5–6 days after plating, and incidence of asymptomatic infection was converted to a percentage.

In spring of 1987 and 1988, kernels harvested the previous year were planted to evaluate emergence. These experiments were arranged as split-split plots of a randomized complete block, where main plots were inbreds, subplots were kernels from inoculated or noninoculated ears, and sub-subplots were treatments (infection levels). In 1987, the two treatments were asymptomatic kernels or kernels selected at random. In 1988, symptomatic kernels were added as a third treatment. There were two replicates per trial in each year. Sixty kernels were planted per experimental unit twice in each year—20 April and 14 May 1987 and 12 April and 17 May 1988. Stand counts were made twice for each trial, with the exception of the early planting in 1988 (4 and 12 May 1987, 22 and 30 May 1987, 10 and 26 May and 3 June 1988).

Data were subjected to analysis of variance. Percent emergence was regressed on incidence of symptomatic and asymptomatic infection of kernels by *F. moniliforme*.

RESULTS AND DISCUSSION

Incidence of *F. moniliforme*. Silk inoculation resulted in approximately twice the incidence of asymptomatic infection of kernels by *F. moniliforme* as that occurring with noninoculation in all 3 yr (Table 1). However, a significant inbred × inoculation interaction

Table 1. Means of all inbreds for incidence (%) of asymptomatic and symptomatic infection of kernels by *Fusarium moniliforme* and emergence (%) of sweet corn inbreds in 1986, 1987, and 1988

	1986			1987			1988	
Treatment	Asymptomatic infection[w]	Symptomatic infection[x]	Emergence[y]	Asymptomatic infection	Symptomatic infection	Emergence	Asymptomatic infection	Symptomatic infection
Noninoculated	33.5 a[z]	14.8 a	79.3 a	28.6 a	6.7	74.9 a	9.1 a	10.6
Inoculated	54.5 b	21.5 b	71.2 b	54.0 b	8.5	71.6 b	17.2 b	14.2

[w] Percentage of healthy appearing kernels showing growth of *F. moniliforme* on PCNB medium.
[x] Percentage of kernels showing signs or symptoms of infection by *F. moniliforme*.
[y] Emergence evaluated the following spring. Percentages are from the late stand count in the late planting date in 1987 and 1988; kernels were chosen at random.
[z] Differences among inoculated and noninoculated treatments indicated by letters in columns are based on ANOVAs in Tables 1 (infection) and 4 (emergence).

occurred for asymptomatic infection in 1986 and 1987, indicating that individual inbreds did not respond equally to inoculation (Table 2). Inoculation with *F. moniliforme* increased the incidence of asymptomatic infection in susceptible inbreds but had less effect on the most resistant inbreds (Fig. 1), which supports the observation of King and Scott (11) that there are genotypic differences in levels of asymptomatic infection.

Incidence of symptomatic infection of kernels was higher among inoculated ears than among noninoculated ears in 1986 (Tables 1 and 2). Incidence of symptomatic infection also differed among inbreds in all 3 yr (Tables 2 and 3). The inbred × inoculation interaction for symptomatic infection was significant only in 1988, possibly because of the more extreme resistance levels of the selected inbreds or the confounding effects of high incidences of *Penicillium* and *Aspergillus* species.

The incidence of asymptomatic and symptomatic infection of kernels by *F. moniliforme* from noninoculated and inoculated ears was significantly positively correlated among inbreds in each year, although correlation coefficients varied from 0.32 to 0.80 (Figs. 1 and 2). These relatively low correlation coefficients indicate considerable variation among inbred response to inoculation and natural infection. Sources of this variation might include spatial and temporal distribution of natural inoculum, microenvironmental conditions, and plant maturity. Also, the relatively small sample size used to estimate asymptomatic infection of kernels may have affected these values. Scott and King (22) recommended a sample size of 65 kernels and six replications to estimate plot means for kernel infection by *F. moniliforme* and to reduce standard errors of means, respectively. Thus, our estimates of symptomatic infection of kernels, which were based on 100 kernels per experimental unit, were probably more reliable than our estimates of asymptomatic infection, which were based on 10 or 20 kernels per experimental unit. Slope values for regressions of incidence from noninoculated ears on incidence from inoculated ears were considerably less than one, which reflected the effects of inoculation on the incidence of *F. moniliforme*.

Asymptomatic and symptomatic infection of kernels were positively correlated in 1986 and 1987 (r = 0.49 and 0.31, respectively; Fig. 3). Symptomatic infection was not always indicative of asymptomatic infection, however, because asymptomatic infection among inbreds varied greatly. For example, several inbreds showed 100% incidence of asymptomatic infection of kernels by *F. moniliforme* in 1986, but the incidence of symptomatic infection for these inbreds ranged from 10 to 95% (Fig. 3A, Table 3). Also, the incidence of symptomatic and asymptomatic infection varied among years, depending on the inbreds evaluated and the environment (Table 1, Fig. 3). For example, both symptomatic and asymptomatic infection were reduced in 1987 compared with 1986 because of the selection of several resistant inbred lines for evaluation in 1987. Also, in the extremely hot, dry year of 1988, asymptomatic infection was much lower than in the previous 2 yr and symptomatic and asymptomatic infection were not correlated.

Emergence. Emergence of kernels from inoculated ears was less than that of kernels from noninoculated ears, regardless of infection level (Table 1). The analyses of variance for emergence indicated the same significant main and interaction effects for planting dates and stand count dates within years (Table 4). Emergence was delayed and very poor in the early planting in 1988, probably because of cold temperatures. The inbred responses to inoculation and infection were the same as those observed in the later planting, however. In both years, inoculation decreased the emergence of susceptible inbreds but did not affect the emergence of the most resistant inbreds, which was indicated by the significant inbred × inoculation interaction. Infection level was significant in 1987, but interactions involving infection level were not. Thus, inbreds varied in response to inoculation, but emergence was reduced for inbreds that became infected. In 1988, there was a significant inbred × infection interaction, apparently due to a variable inbred emergence response to asymptomatic infection.

The relationship between emergence and incidence of symptomatic infection was significant, negative, and consistent among years and planting dates, despite lower incidence of symptomatic infection among the selected inbreds in 1988 and the overall poor emergence in the early planting in 1988 (Fig. 4). The relationship between emergence and asymptomatic infection also was significant and negative in 1987 but not in 1988 (Fig. 5). When emergence data from all four trials (2 yr, two planting dates) were regressed on incidence of symptomatic and asymptomatic infection, higher incidence resulted in significant reductions in

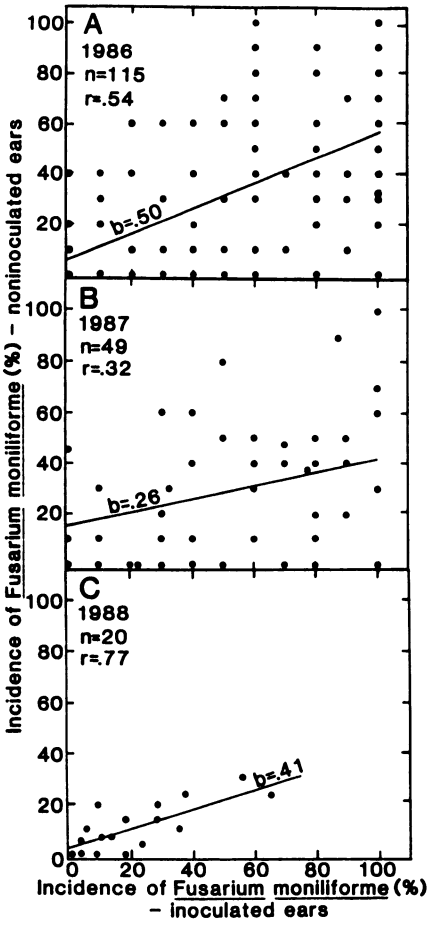

Fig. 1. Incidence in **(A)** 1986, **(B)** 1987, and **(C)** 1988 of asymptomatic infection of kernels by *Fusarium moniliforme* from noninoculated ears of sweet corn inbreds plotted on incidence from inoculated ears. Each point represents an inbred.

Table 2. Main and interaction effects for incidence of asymptomatic and symptomatic infection of sweet corn kernels by *Fusarium moniliforme* in 1986, 1987, and 1988

	1986		1987		1988	
Source of variation	Asymptomatic infection[x]	Symptomatic infection[y]	Asymptomatic infection	Symptomatic infection	Asymptomatic infection	Symptomatic infection
Inbred	***[z]	***	***	*	***	***
Inoculation	***	***	***	*	**	NS
Inbred × inoculation	***	NS	***	NS	NS	**

[x] Percentage of healthy appearing kernels showing growth of *F. moniliforme* on PCNB medium.
[y] Percentage of kernels showing signs or symptoms of infection by *F. moniliforme*.
[z] NS = not significant, * = $P < 0.10$, ** = $P < 0.05$, *** = $P < 0.01$.

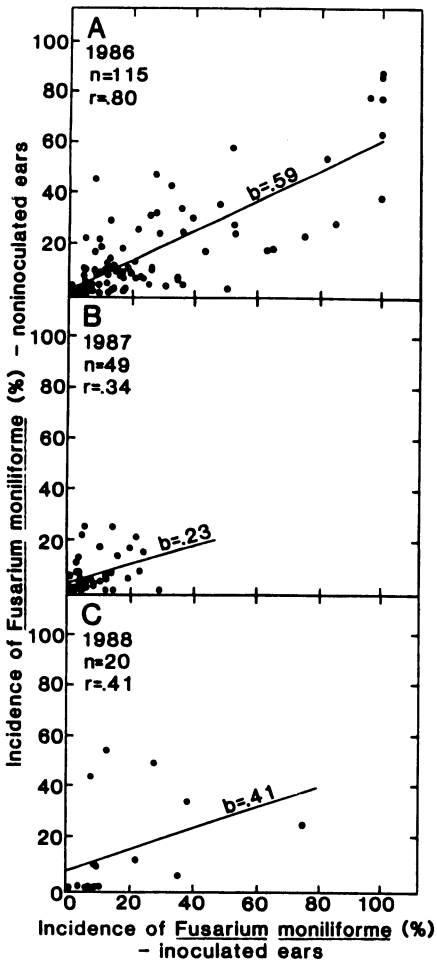

Fig. 2. Incidence in **(A)** 1986, **(B)** 1987, and **(C)** 1988 of symptomatic infection of kernels by *Fusarium moniliforme* from noninoculated ears of sweet corn inbreds plotted on incidence from inoculated ears. Each point represents an inbred.

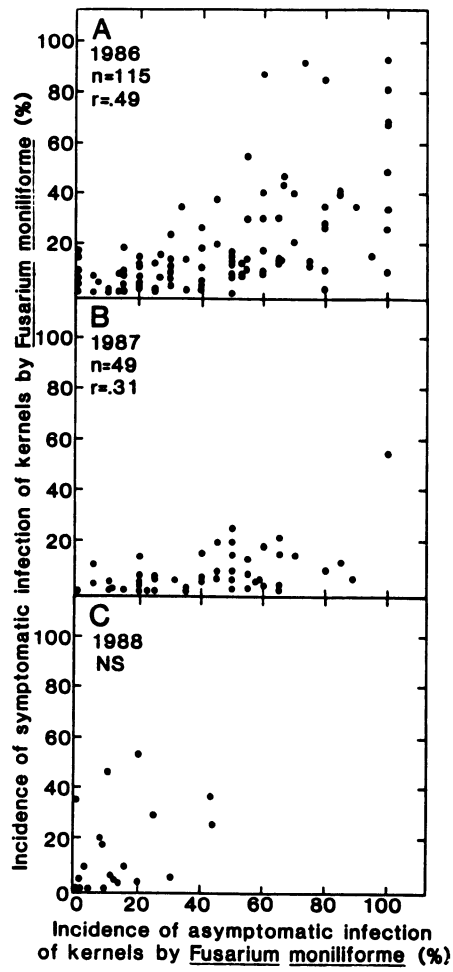

Fig. 3. Correlations of incidence in **(A)** 1986, **(B)** 1987, and **(C)** 1988 of symptomatic and asymptomatic infection of kernels by *Fusarium moniliforme* for inoculated and noninoculated ears. Each point represents an inbred.

emergence (Fig. 6). The observation that incidence of symptomatic infection of kernels by *F. moniliforme* predicted emergence more consistently over years than asymptomatic infection (Figs. 4 and 5) is not unexpected. Kernels that have become molded by the pathogen often are dead before planting, whereas asymptomatic kernels often remain germinable. However, the importance of asymptomatic infection in affecting inbred emergence is illustrated by the intercepts in Figure 6A and B. When incidence of symptomatic infection by *F. moniliforme* was 0%, emergence was about 63%, but when incidence of asymptomatic infection was 0%, emergence increased to about 75%.

Relatively low coefficients of determination (r^2) for all regressions of emergence on incidence of fungal infection do not negate the conclusion that infection of sweet corn kernels with *F. moniliforme* adversely affects emergence. However, low coefficients of determination indicate that the models (although significant) did not account for a large portion of the variation in inbred response. Low coefficients of determination may have been partially due to the small sample size from which asymptomatic infection was estimated, but this would not account for the low coefficients of determination for regressions of emergence on symptomatic infection that was estimated from a sample size of 100 kernels per experimental unit. Additional experimentation is needed to determine the mechanism(s) of the response. Among the inbreds that deviated from the regression models, several showed low incidence of symptomatic infection, yet

Table 3. Incidence (%) of asymptomatic and symptomatic infection of kernels by *Fusarium moniliforme* and emergence (%) of sweet corn inbred lines evaluated in 1986, 1987, and 1988

		1986				1987				1988	
Inbred	Endosperm[v]	Asymptomatic infection[w]	Symptomatic infection[x]	Early emergence[y]	Late emergence	Asymptomatic infection	Symptomatic infection	Early emergence	Late emergence	Asymptomatic infection	Symptomatic infection
IL781a	*su*	10	4	60	87	0	0	15	93	0	0
IL125b	*su*	0	1	94	90	40	0	67	95	8	0
IL773a	*su*	0	6	80	90	10	0	27	80	2	7
Oh43	*Su*	10	5	74	70	20	2	49	82	15	10
IL776a	*se*	20	7	47	64	30	0	13	73	8	3
IL11d	*su*	0	5	60	80	40	2	40	69	18	0
W6786	*su*	30	4	86	94	50	0	58	85	5	35
IL789a	*su*	30	18	34	60	0	0	46	86	10	46
IL451b	*su*	0	6	63	70	70	0	34	93	22	7
I453	*su*	30	24	86	96	80	2	49	87	2	0
MA83610b	*se*	0	10	64	86	20	5	7	66	74	35
IL676a	*su*	60	36	83	84	70	3	18	62	2	8
I2123	*su*	60	100	13	20	40	5	16	49	18	9
I5125	*su*	80	40	70	86	90	16	22	78	27	5
IL788a	*su*	100	28	56	67	100	19	5	26	10	22
MA83608b	*su*	100	82	26	64	100	76	25	73	37	9
IL783a	*sh2*	100	100	7	0	80	8	13	55	53	7
Population means[z]		54	22	59	71	54	8	25	72	17	14
BLSD (k = 100)		61.2	24.4	13.8	11.2	33.5	17.2	12.0	10.6	15.3	9.1

[v] *su* = *Sugary-1*, *se* = *sugary enhancer*, *sh2* = *shrunken-2*, and *Su* = dent endosperm types.
[w] Percentage of healthy appearing kernels showing growth of *F. moniliforme* on PCNB medium.
[x] Percentage of kernels showing signs or symptoms of infection by *F. moniliforme*.
[y] Emergence evaluated the following spring. Percentages are from the late stand count for each planting in 1987 (seed produced in 1986) and 1988 (seed produced in 1987), with the exception of the early planting in 1988, for which only one count was made; kernels were chosen at random.
[z] Mean of all inbreds evaluated in a given year.

had poor emergence (Fig. 6A). Such a response may be attributable to high levels of asymptomatic infection, infection by other undetected pathogens in the seeds and/or soil, insects, environmental stress, or, perhaps most likely, inherently poor vigor of seedlings. Conversely, some inbreds showed a high incidence of asymptomatic kernel infection by *F. moniliforme* and good emergence (Fig. 6B). Seedlings with asymptomatic infection may emerge well yet develop blight symptoms under conditions of environmental stress (25); this aspect was not examined in this study, however. Alternatively, vigorous seedlings may "outgrow" the fungus, which has been described as only weakly pathogenic (14,30). It also has been suggested that seedborne *F. moniliforme* may become systemic (7,15), in which case asymptomatic infection could ultimately result in ear rot. Variability in responses among genotypes supports the contention that *F. moniliforme* may contribute to the poor performance of seedlings of particular sweet corn genotypes under given environmental conditions, but it is not likely the primary cause of the problem (27).

Sources of resistance in sweet corn. Several inbreds showed good emergence and partial resistance to kernel infection by *F. moniliforme*, but none of the inbreds were immune to kernel infection (Table 3). Inbreds IL781a, IL125b, and IL773a had low levels of symptomatic and asymptomatic infection. IL125b showed outstanding emergence in all trials, but emergence of IL781a and IL773a was adversely affected by cold soil temperatures in the early planting in 1988. IL781a is a selection from IL677a × (Funk's G-4455 × IL677a). IL773a is a selection from (IL14h × IL11d) × (Lenha × IL677a). IL125b is a selection from (IL11d × Louisiana Sweet Bantam 2) × (Cuzco × an unidentified yellow dent). Thus, IL781a and IL773a share a common ancestor, IL677a, which was selected from IL442a × (IL44b × BOV1035), and is a source of the *se* gene. IL125b and IL773a share a common ancestor, IL11d, which was derived from a self from a narrow grain open-pollinated dent variety. IL11d was moderately resistant to *F. moniliforme* kernel infection (Table 3). Interestingly, the pedigrees of IL125b, IL773a, and IL781a include floury varieties of South American origin (BOV1035, Cuzco, and Lenha). It is not surprising that the South American varieties may provide resistance to kernel infection, since the coevolution of *Z. mays* and *F. moniliforme* is likely due to a relationship dating to the origin in *Zea* in South America. Expression of genetic variation among tropical sweet corn germ plasm has enabled the selection of satisfactory levels of resistance to *F. moniliforme* in Hawaii (J. L. Brewbaker, *personal communication*). It seems likely that additional sources of resistance may be found in tropical and South American germ plasm.

IL125b is a source of the Rp_1^d gene (from Cuzco) for resistance to common

Table 4. Main and interaction effects of kernel infection by *Fusarium moniliforme* on sweet corn inbred emergence in 1987 and 1988[v]

Source of variation	Emergence[w]			
	1987		1988	
	Early planting[x]	Late planting	Early planting	Late planting
Inbred	*[y]	*	*	*
Inoculation	*	*	*	*
Inbred × inoculation	*	*	*	*
Infection[z]	*	*	*	*
Inbred × infection	NS	NS	*	*
Inoculation × infection	NS	NS	NS	NS
Inbred × inoculation × infection	NS	NS	NS	NS

[v] Year of emergence evaluation; inoculation and harvest done the previous year.
[w] Percentage stand.
[x] There were no significant differences between stand counts within planting dates.
[y] NS = not significant, * = $P < 0.05$.
[z] Visibly infected kernels, symptomless kernels, or kernels chosen at random.

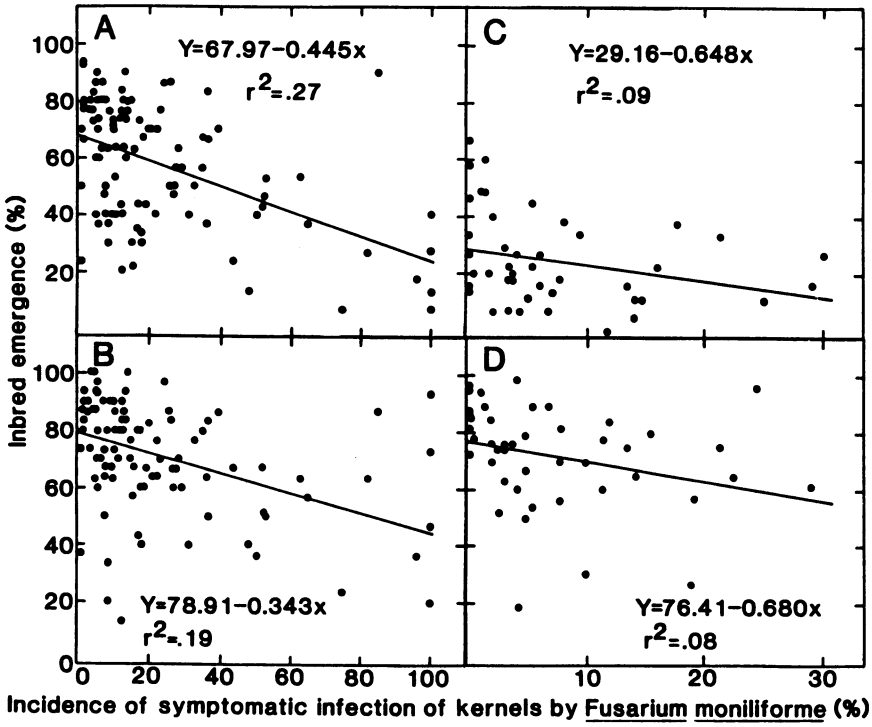

Fig. 4. Effect of symptomatic infection of kernels by *Fusarium moniliforme* on sweet corn inbred emergence. Kernels chosen at random from inoculated ears. **(A)** Early planting, 1987; **(B)** late planting, 1987; **(C)** early planting, 1988; **(D)** late planting, 1988.

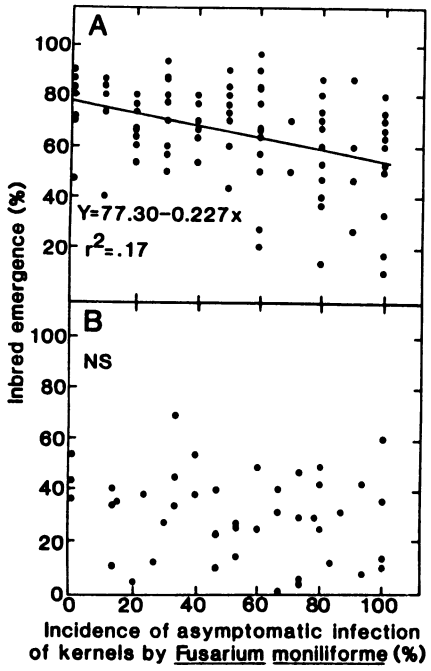

Fig. 5. Effect in **(A)** 1987 and **(B)** 1988 of asymptomatic infection of kernels by *Fusarium moniliforme* on sweet corn inbred emergence. Kernels chosen from inoculated ears.

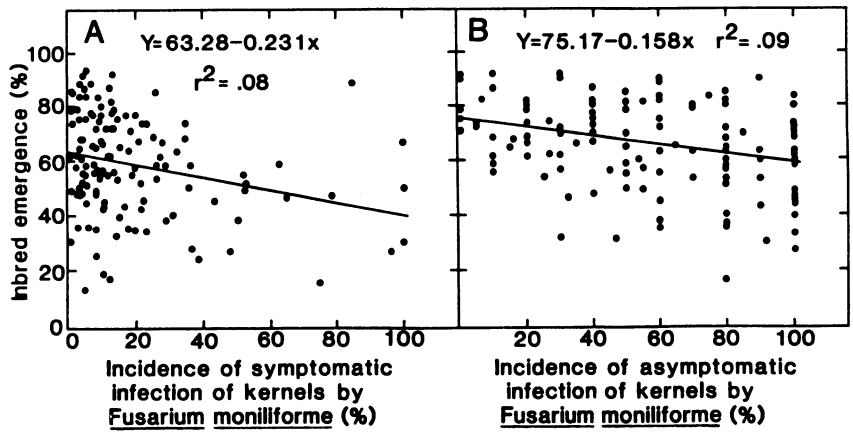

Fig. 6. Mean effects in 1987 and 1988 of kernel infection by *Fusarium moniliforme* on emergence of sweet corn inbreds. Data from the late stand count in the late planting in each year. Incidence of **(A)** symptomatic infection and **(B)** asymptomatic infection.

rust, caused by *Puccinia sorghi* Schwein. It presently is being used by several sweet corn seed companies to incorporate Rp_1^d into elite *sh2* and *su* inbred lines via the backcross method. Thus, the potential exists for simultaneous gains in resistance to *F. moniliforme* and *P. sorghi* when IL125b is used as the source of resistance. On the basis of the maternal inheritance of resistance to kernel infection by *F. moniliforme* (9,23), resistance will not be improved if the susceptible parent is used as the female in production of hybrid seed.

Improvement of the emergence of sweet corn hybrid seed and more vigorous performance of seedlings are most important among the *sh2* endosperm types. The inbreds identified as most resistant to *F. moniliforme* and showing good emergence in this study were *su* endosperm types. This does not preclude the use of *su* lines as sources of resistance, especially since many other traits important in sweet corn hybrids already may be present in these lines. Also, it is noteworthy that many of the most susceptible inbreds in this study were *su* types (Table 3). Thus, these data do not show a distinct correlation between endosperm type (which affects sugar content of kernels) and infection, as has been suggested previously (27). Furthermore, these data provide additional evidence that physiological contraints are as important as seedborne pathogens in vigor of sweet corn seedlings.

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