Infection of Two Endosperm Mutants of Sweet Corn by *Fusarium moniliforme* and its Effect on Seedling Vigor

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ABSTRACT

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Kernels of hybrid sweet corn plants with the recessive *shrunken*-2 (*sh*2) gene became heavily infected with *Fusarium moniliforme* in the field earlier than kernels of hybrid plants with the standard *sugary* (*su*) gene. Mature ears of both mutants inoculated 10 days postpollination (DPP) with *F. moniliforme* had higher levels of rot and seed infection than those inoculated later in development. In *sh*2 seeds, the pathogen appeared to enter through small cracks in the pericarp or by appressoria. The fungus spread throughout pocketlike areas between the pericarp and aleurone layer, and eventually moved into the endosperm and embryo. Infection increased the number of abnormal *su* seedlings and reduced seedling growth of *sh*2 seeds germinated under optimum conditions. Germination of infected *sh*2 seeds was lower in a cold soil test and growth rates were reduced compared to those observed under optimum conditions. Poor seed and seedling vigor of *sh*2 was due, in part, to infection with *F. moniliforme*, but this infection did not appear to be the primary factor involved in the reduction in vigor. Under stress conditions, uninfected *sh*2 seeds had less germination and vigor than uninfected *su* seeds.

Fusarium moniliforme Sheldon commonly infects a wide range of crops throughout the world and is a major parasite of the Gramineae, particularly in tropical and subtropical regions (2). On corn this fungus can cause stalk rot, leaf spot, ear and kernel rot, damping-off, and seedling blight (4,20). Although *F. moniliforme* causes a kernel rot, the pathogen is often found in kernels that appear to be physically undamaged (12,25). Infected seed that is planted may increase the incidence of seedling blight (8) and contribute to systemic infection of plants (7). However, the importance of infected seed as a source of subsequent plant infection seems minimal, because seedlings can be readily infected by *F. moniliforme* from infested soil debris (13).

Corn kernels, as they mature, may become either externally (12) or systemically infected (14). Corn earworms or borers may aid in the development of stalk and ear rots caused by *F. moniliforme* either by causing wounds through which the pathogen can enter later or by carrying inoculum directly into tissues (4,20,21). Kernel infection in the tip half of corn ears before maturity is most common (10). *Fusarium* spp. were isolated from kernels the third week after silk emergence and peaked in occurrence the eighth week (9). King (10) first isolated *F. moniliforme* 2 wk after midsilk, and infection increased to 35–66% by the 10th wk.

Sweet corn hybrids with the recessive mutant gene *shrunken*-2 (*sh*2) in place of the standard recessive *sugary* (*su*) gene had two to three times more sugar and one-third less starch 16 to 28 days postpollination (DPP) (5). These *sh*2 hybrids have not been widely accepted because of their poor seedling vigor (27). The higher sugar content of the *sh*2 kernels has been associated with an increase in rot by pathogens during germination (1). Production of *sh*2 seed in Florida in previous years yielded high percentages of infected seed (R. C. Styer, *unpublished*). Genotypic differences in susceptibility of corn have been reported for kernel rot and asymptomatic kernel infection caused in inbreds (22,28) and hybrids (11) by *F. moniliforme*.

The purpose of the experiments reported here was to determine the susceptibility to infection by *F. moniliforme* during seed development of two endosperm mutants of sweet corn (*sh*2 and *su*) and the effect of this pathogen on subsequent seed viability and vigor.

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MATERIALS AND METHODS

Isogenic lines of sweet corn (*Zea mays* L.) homozygous for *su* and *sh*2 were produced in the field and greenhouse as previously documented (23).

Fungal infection. Seeds of field-grown *sh*2 and *su* corn were surface disinfested for 5 min with 1.8% sodium hypochlorite and rinsed three times with sterile distilled water. Ten replications of five seeds each were plated on acidified potato-dextrose agar (APDA) and incubated for 7 days at 25 C under continuous fluorescent light, 5,000 lux. Fungus-infected seeds were counted and expressed on a percentage basis.

Greenhouse inoculation. Ears of *sh*2 and *su* corn were inoculated with *F. moniliforme* in the greenhouse 10 and 20 DPP. Inoculations were made at the silk end by injecting 0.5 ml of a suspension containing 10^6 spores per milliliter into the silk tuft (15). The spore suspension was prepared by adding 5 ml of sterile water containing one drop of Triton X-100 per 100 ml to five 2-wk-old APDA cultures of *F. moniliforme*. The suspensions were combined and adjusted to the final concentration by using a hemacytometer.

Four ears of each mutant at each inoculation time were harvested 30, 38, and 46 DPP. In addition, four control ears of *sh*2 and *su* corn injected only with sterile water plus Triton X-100 were harvested at 46 DPP. All ears were husked, dried for 2 wk at 30 C, and rated for ear rot. Ear rot was rated on a scale of 1 to 5, with 1 = no rot and $5 \geqslant 75\%$ rot. Only visibly nondiseased seeds were removed from the cob and assayed for fungal infection. Fifty seeds per ear were surface disinfested, incubated, and evaluated as above.

Location of fungi in seeds. Ten seeds each of field-grown *sh*2 (30 and 46 DPP) and *su* (38 and 46 DPP) and inoculated, greenhouse-grown, mature *sh*2 and *su* were separately soaked in water for 24 hr at 25 C. The pericarp, tip cap, endosperm, and embryo were dissected aseptically, surface disinfested for 2 min in 1.8% sodium hypochlorite, rinsed three times in sterile distilled water, and plated on APDA, one seed per plate (16). All plates were incubated for 7 days at 25 C under continuous fluorescent light at 5,000 lux to determine infection by *F. moniliforme*.

Seed viability and seedling vigor tests. Five replications of 20 seeds of mature, infected ($\geqslant$90% of total seeds) and uninfected, greenhouse-grown *sh*2 and *su* corn were tested for viability and vigor by using a rolled towel test and cold soil test as previously described (23).

Scanning electron microscopy. Observations of freshly infected

sh2 seeds were obtained by placing mature seeds on cultures of *F. moniliforme* for 2 days, removing, then treating as previously described (24).

RESULTS

The incidence of fungal infection in the field was considered severe in developing *sh2* seeds almost 2 wk earlier than in *su* seeds (Fig. 1). More than 50% of the *sh2* seeds were infected after 30 DPP. Seeds with the *su* gene did not reach this high a level until after 42 DPP. Observations of previous years (R. C. Styer, *unpublished*) support this determination of an earlier and more severe fungal infection of *sh2* corn. Field-grown seeds were infected predominantly with *F. moniliforme* but also exhibited a low incidence of *Aspergillus flavus*.

When harvested 26 to 28 DPP, greenhouse-grown *sh2* ears had more rot than uninoculated ears (Fig. 2A). Ears of *sh2* corn inoculated 20 DPP had increasingly greater amounts of rot as they matured. All inoculations of *sh2* ears led to ear rot. To compensate for this high infection, only visibly nondiseased seeds from outside the rotted areas on the ear were selected to study the spread of the pathogen. The highest percentages of seed infection (Fig. 2B) were obtained from inoculated *sh2* ears that had the greatest ear rot rating (correlation coefficient, 0.83, $P = 0.01$). Few significant differences in percentage of infected seeds were noted among *sh2* ears, regardless of inoculation and harvest date.

More ear rot was measured in *su* ears ≥4 wk after inoculation compared with uninoculated ears (Fig. 2A). No ear rot was observed 10 days after inoculation, but 38% of the seeds were infected. Seed infection percentages from *su* ears inoculated 10 or 20 DPP were not different between harvest dates (Fig. 2B). Ears of *su* corn that had the most ear rot had more infected seeds than uninoculated ears (correlation coefficient, $r = 0.597$, $P = 0.01$).

Alternate entrances of *F. moniliforme* into the kernel, other than through the tip cap, were determined by scanning electron microscopy of inoculated, mature *sh2* seeds (Fig. 3). Upon inoculation, the pathogen grew over the surface of the seed (Fig. 3A) and germinating spores were observed (Fig. 3B). The freeze-drying treatment caused the hyphae and spores to collapse. Hyphae entered through very small cracks in the pericarp (Fig. 3C), but also formed appressoria that may have directly penetrated the seed surface (Fig. 3D). However, no further studies were made to determine if penetration actually occurred with appressoria.

After penetrating the *sh2* pericarp, the hyphae grew throughout the area between the pericarp and aleurone layer (Fig. 4). Due to the collapsed nature of *sh2* seeds, the pericarp pulled away from the aleurone layer and small 'pockets' were formed, into which the hyphae grew and sporulated. These 'pockets' were not seen in *su* seeds.

The pericarp, tip cap, endosperm, and embryo of infected groups of *sh2* and *su* corn seeds were aseptically plated on APDA to locate *F. moniliforme* (16) (Table 1). Although seed parts were surface disinfected for only 2 min, the disinfection time appeared to be too long to enable isolation of the fungus from the pericarp and some tip caps. Seeds with infected tip caps also had infected endosperms and embryos. These deep-seated infections were noted in mature (46 DPP), field-grown *sh2* and *su* seeds, and in inoculated, mature,

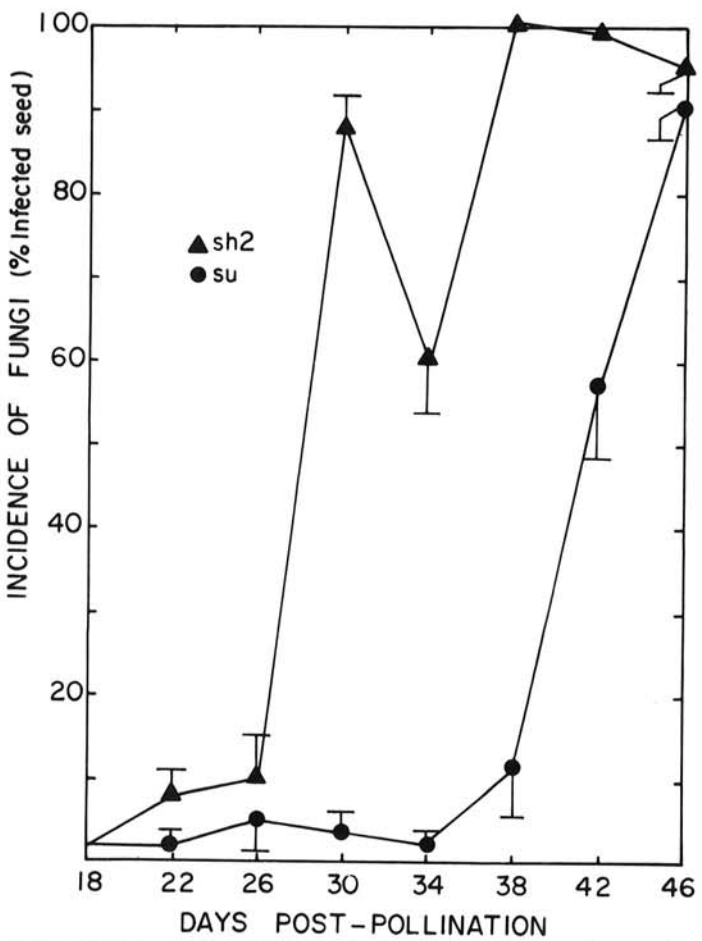

Fig. 1. Incidence of fungal infection (primarily *Fusarium moniliforme*) in seeds of inbred hybrid sweet corn plants with *shrunken*-2 (*sh2*) or *sugary* (*su*) genes harvested at different stages of development. Bars represent standard errors of the means.

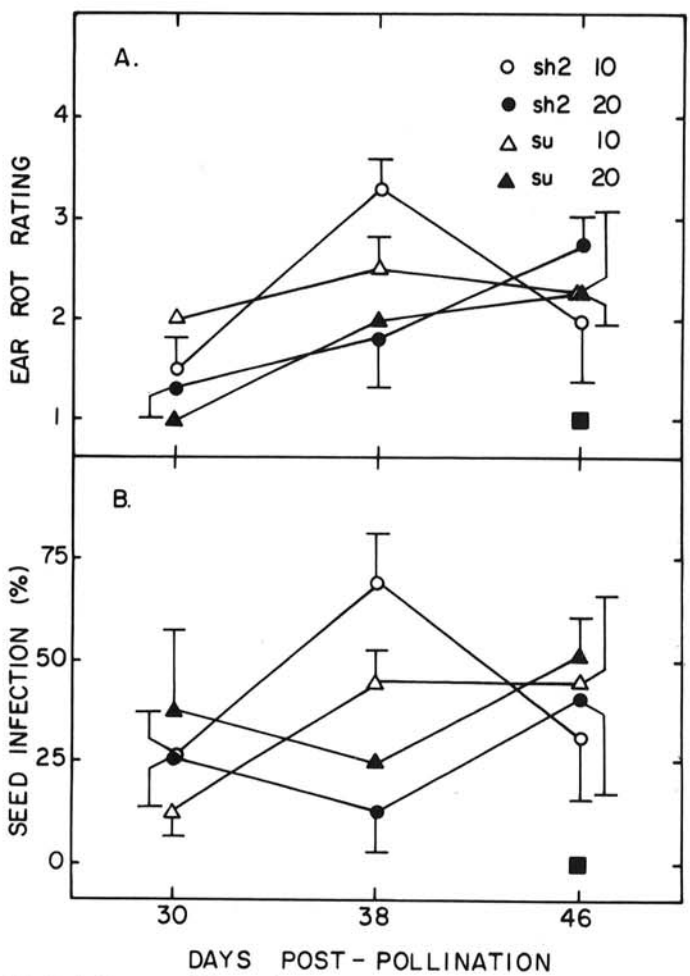

Fig. 2. A, Ear rot and **B,** seed infection in ears of greenhouse-grown inbred sweet corn plants with *shrunken*-2 (*sh2*) or *sugary* (*su*) genes inoculated with *Fusarium moniliforme* and harvested at various stages of development. Ear rot rating was on a scale of 1 to 5, with 1 = no rot and 5 = 75% or greater rot. Controls: ■ = uninoculated *sh2* and *su* ears. Open symbols represent ears inoculated 10 days postpollination (DPP) and closed symbols represent ears inoculated 20 DPP. Each data point represents the average of four ears. Bars represent standard errors of the means.

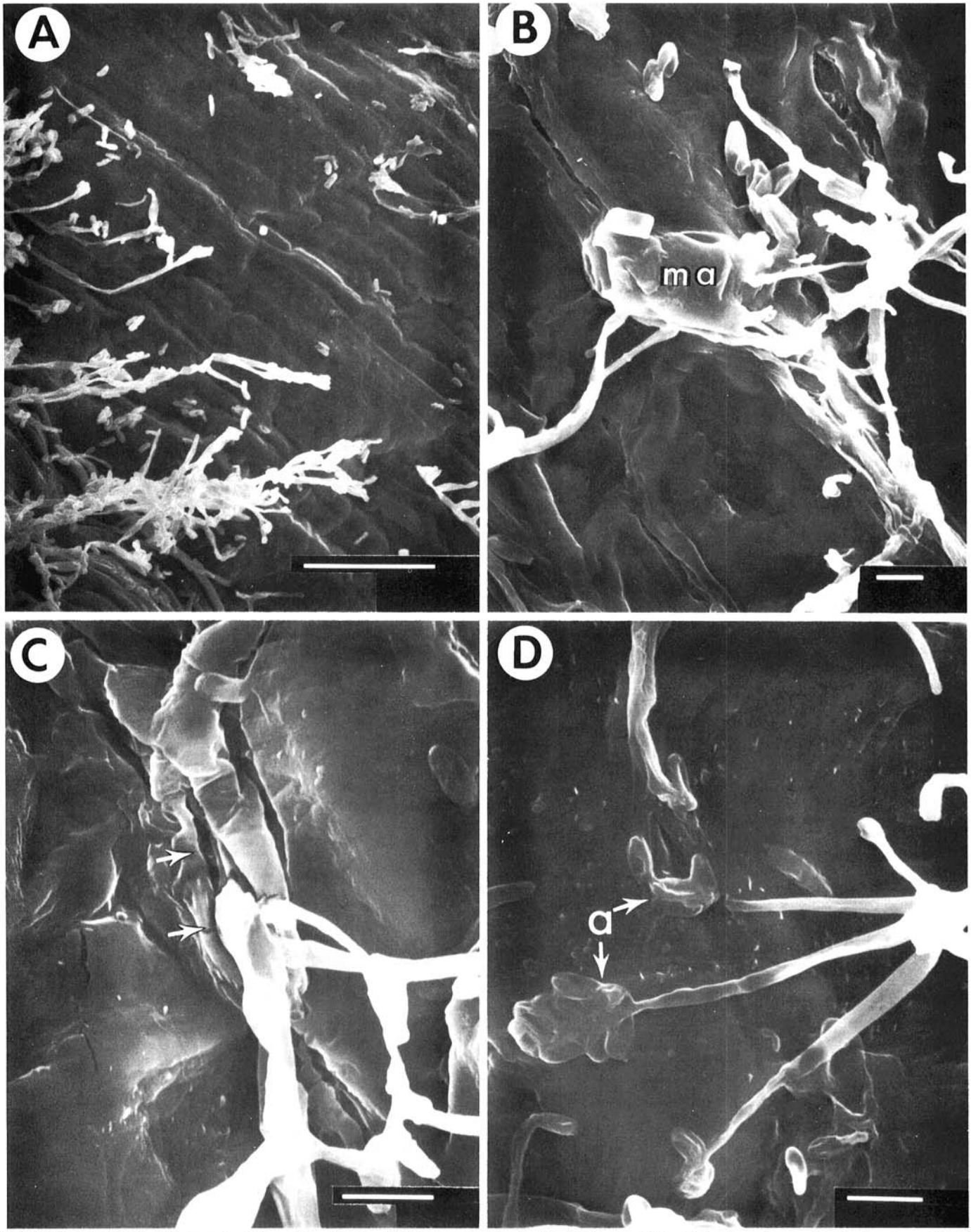

Fig. 3. Scanning electron micrographs of the surface of mature (46 days postpollination [DPP]) seeds of inbred hybrid sweet corn with the *shrunken*-2 (*sh2*) gene following inoculation with *Fusarium moniliforme*. **A,** Hyphae and microconidia. **B,** Germinating macroconidium (ma). **C,** Hyphae entering through a small crack in the seed surface (arrows). **D,** Hyphae directly penetrating the seed surface by forming appressoria (a). Bar for **A** = 50 μm. Bar for **B–D** = 5 μm.

greenhouse-grown *sh*2 and *su* seeds. The fungus was not isolated from embryos or endosperms of field-grown, 38 DPP *su* seeds, but was isolated from embryos of 30 DPP *sh*2 seeds. The infection of the latter two seed ages coincided with the time when field infection by *F. moniliforme* increased greatly (Fig. 1).

Infection by *F. moniliforme* during seed development affected the vigor of mature, greenhouse-grown *sh*2 and *su* seedlings germinated under optimum conditions (Table 2). More abnormal seedlings were noted from infected *su* seeds, whereas radicle and whole seedling lengths were significantly shorter in infected *sh*2 seedlings. Fresh and dry weights of seedlings from infected *su* seeds were significantly less than those from uninfected seeds, whereas only seedling fresh weights were lower in infected *sh*2.

Germination and emergence of infected *sh*2 seeds in cold soil was

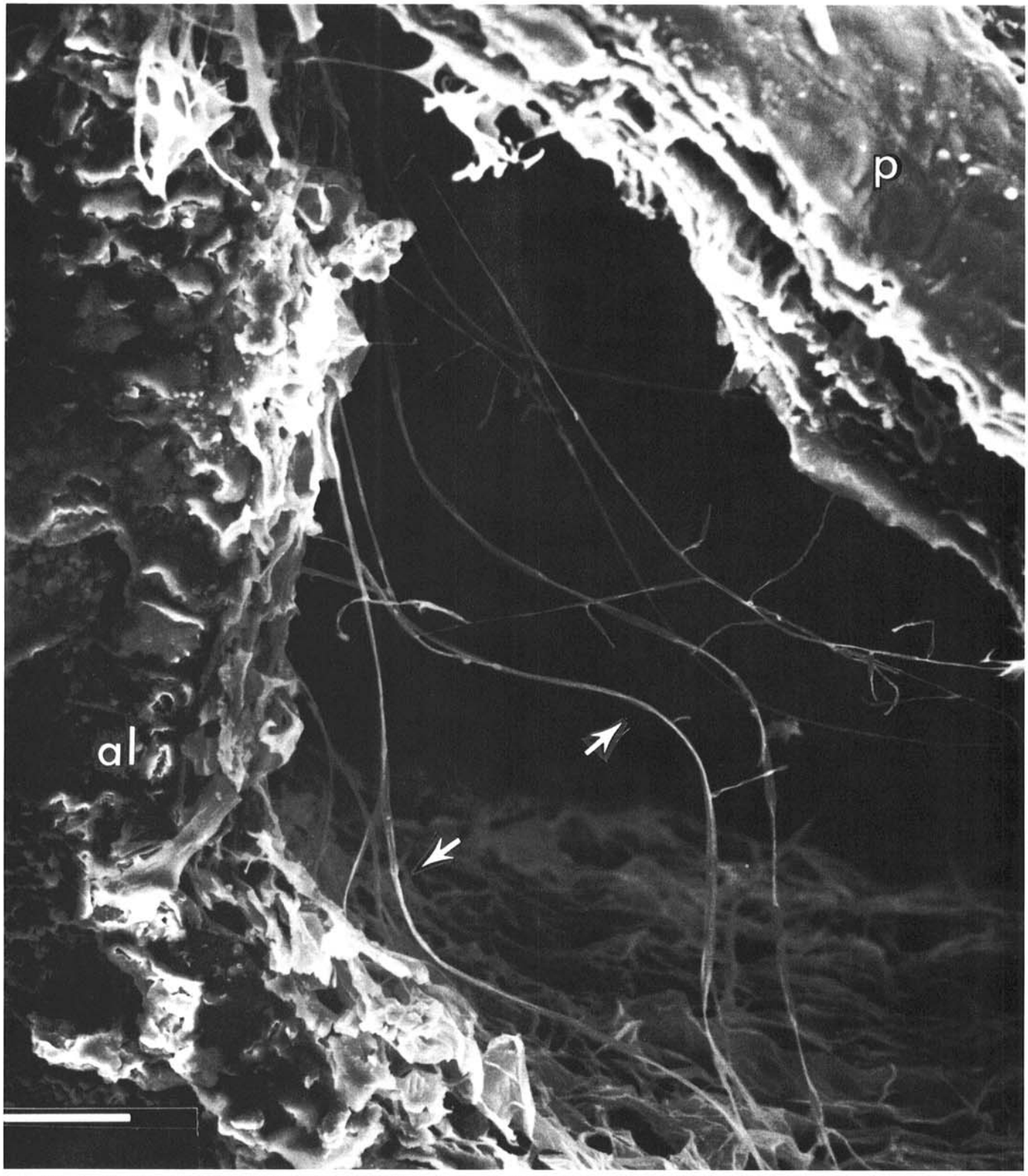

Fig. 4. Scanning electron micrograph of a 'pocket' between the pericarp (p) and the aleurone layer (al) of a mature (46 days postpollination [DPP]) seed of an inbred sweet corn hybrid with the *shrunken*-2 (*sh*2) gene following inoculation with *Fusarium moniliforme*. Hyphae can be seen throughout this area (arrows). Bar = 50 μm.

significantly reduced compared with uninfected *sh*2 seeds (Table 3). No differences in germination and emergence were noted between infected and uninfected *su* seeds. Uninfected *sh*2 seeds had significantly less germination and seedling vigor than uninfected *su* seeds when germinated in cold soil.

DISCUSSION

Early infection of field-grown *sh*2 seeds, primarily by *F. moniliforme*, can be attributed to several factors. Rainfall in late spring and early summer in Florida can delay, to some extent, maturation of field-grown kernels of *sh*2 and *su* corn. The resulting higher moisture content would leave the kernels, particularly those of *sh*2 corn, vulnerable to fungal infection for a longer period of time. Ears maturing in a downward position (*su*) usually have less rot than ears maturing upright, such as those of *sh*2 corn (20). The heavier earworm infestation in *sh*2 ears (R. C. Styer, *unpublished*) would increase pathogen access and the higher sugar content of *sh*2 kernels (24) would provide an ideal substrate for pathogenic growth.

Generally, inoculated ears of both genotypes with the greatest degree of ear rot tended to have the highest number of asymptomatically infected seeds (correlation coefficient, $r = 0.72$, $P = 0.01$). It was previously found that ear infection was greatest when inoculation occurred 4 to 14 DPP, and least when it occurred 21 or more DPP (26,28). The lower ear rot and asymptomatic seed infection produced in *sh*2 and *su* ears inoculated 20 DPP may be attributed to a shorter time for disease development, lower moisture content of silks and kernels (28), or kernels with more resistance because of structural changes (24). However, *sh*2 ears still rotted to some extent within 10 days after inoculation while *su* ears did not. This difference indicates that the high-sugar mutant (*sh*2) was more susceptible to infection with *F. moniliforme*, as shown in the high-lysine *opaque*-2 corn mutant (28). Genotypic differences in susceptibility of corn to *F. moniliforme* have also been reported with inbreds (22,28) and their hybrids (11).

F. moniliforme was present in tip caps, endosperms, and embryos of mature *sh*2 and *su* seeds, whether grown in the field or inoculated in the greenhouse. Deep-seated infections were not obtained in less mature seeds of field-grown *sh*2 and *su* corn. Isolation of *F. moniliforme* from inner tissues of seeds is not uncommon (16,18). Early inoculation of sorghum ears with *F. moniliforme* produced deeply infected kernels (3). These kernels matured earlier and were smaller than normal kernels. Early field infection of *sh*2 kernels might lead to deeply established *F. moniliforme* that could reduce seed size and affect seed vigor.

Isolation of *F. moniliforme* from the tip caps of infected *sh*2 and *su* seeds indicated that the pathogen could enter here. Earworm damage could occur initially at the ear tip (17) with fungi gaining access to the cob at this point. From here, the pathogens could move through the cob and infect the kernels through tip caps (14,18). Alternately, *F. moniliforme* could infect the kernels through the silks (12), entering either through pericarp cracks or by appressoria. Further work is needed, however, to conclusively demonstrate the path of infection.

Examination of seed surface morphology revealed no large cracks in the pericarp of *sh*2 seeds, regardless of maturity (24). The seed surface was fairly smooth and did not appear likely to be able to trap spores of *F. moniliforme* (24). The pathogen did not seem to be primarily a surface contaminant occurring in cracks and natural openings in the pericarp, as suggested by El-Meleigi et al (6). Hyphae entered through very small cracks in the pericarp of *sh*2 seeds. Since dry, mature seeds were used as an example, the pericarp cracking may be an artifact of the drying process. However, cracking may occur in developing *sh*2 kernels after 28–30 DPP, when the severe collapsing of *sh*2 kernels begins (L. C. Hannah, *personal communication*). Direct penetration may also be achieved by formation of appressoria.

Infection by *F. moniliforme* adversely affected the seedling vigor of mature, greenhouse-grown *sh*2 and *su* seeds. *F. moniliforme* on or in the seed was the only uncontrolled factor influencing germination and seedling growth under optimum conditions. Mathur et al (16) determined that *F. moniliforme* affected both germination and seedling growth of sorghum germinated on blotters or in soil. Root growth of corn seedlings was inhibited

TABLE 1. Location of *Fusarium moniliforme* in *shrunken*-2 (*sh*2) and *sugary* (*su*) sweet corn seeds

Mutant[a]	DPP[b]	Pericarp	Tip cap	Endosperm	Embryo
*sh*2 F	30[c]	–[d]	–	–	+
	46	–	+	+	+
su F	38[c]	–	–	–	–
	46	–	–	+	+
*sh*2 GHI	46	–	+	+	+
su GHI	46	–	+	+	+

[a] F = field-grown, GHI = greenhouse-grown inoculated.
[b] Days post-pollination.
[c] Developmental stage at which field infection of seeds by the pathogen increased (see Fig. 1).
[d] – = no fungus, + = fungus present as determined by plating of parts isolated from 10 seeds on APDA.

TABLE 2. Viability and vigor of infected (I) and uninfected (UI), mature, greenhouse-grown *shrunken*-2 (*sh*2) and *sugary* (*su*) inbred hybrid sweet corn seeds as determined by rolled towel test

Mutant	Germination (%)	Abnormal seedlings (%)	Germination rate index	Radicle length (cm)	Seedling length (cm)	Seedling wt	
						Fresh (mg)	Dry (mg)
*sh*2 I	88 ab[z]	18 ab	4.7 ab	14.2 b	22.9 b	674 c	96 c
*sh*2 UI	96 a	10 b	5.2 a	17.7 a	28.0 a	814 b	97 c
su I	93 ab	25 a	4.7 ab	17.7 a	27.2 a	870 b	137 b
su UI	84 b	9 b	4.3 b	18.4 a	29.5 a	971 a	144 a

[z] Mean separation in columns by Duncan's multiple range test, $P = 0.05$.

TABLE 3. Viability and vigor of infected (I) and uninfected (UI), mature, greenhouse-grown *shrunken*-2 (*sh*2) and *sugary* (*su*) inbred hybrid sweet corn seeds as determined by cold soil test

Mutant	Emergence (%)	Germination (%)	Abnormal seedlings (%)	Emergence rate index	Radicle length (cm)	Seedling length (cm)
*sh*2 I	13 c[z]	21 c	12 b	0.3 b	5.5 b	12.6 b
*sh*2 UI	23 b	34 b	19 b	0.5 b	6.7 b	13.2 b
su I	70 a	87 a	29 a	1.5 a	12.7 a	21.5 a
su UI	70 a	89 a	28 a	1.5 a	13.0 a	22.0 a

[z] Mean separation in columns by Duncan's multiple range test, $P = 0.05$.

when seeds were inoculated with *F. moniliforme* (8).

The effect of *F. moniliforme* on *sh*2 seed vigor became most noticeable during germination under stress conditions. When *sh*2 seeds were placed in cold soil, germination and emergence of infected seeds were less than uninfected ones, because seeds and seedlings weakened by infection may have died under these conditions. The production of a toxin that inhibits seedling root growth (8,19) would be helped by the presence of *F. moniliforme* within the seed. The high rates of water uptake and leakage from *sh*2 seeds (24) may also stimulate soilborne and seedborne pathogens to attack the seeds during germination.

The association of *F. moniliforme* with *sh*2 seeds during development contributed to, but did not appear to be the primary reason for, the reduction in seed viability and vigor. As illustrated under stress conditions, there still remained a large difference in seed germination and seedling vigor between uninfected *sh*2 and *su* seeds. The small endosperm size of *sh*2 seeds has been associated with slow seedling growth (27). Further work needs to be done to investigate additional causes of poor seed vigor in *sh*2 corn.

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