Cob and Kernel Infection by *Aspergillus flavus* and *Fusarium moniliforme* in Inoculated, Field-grown Maize Ears

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ABSTRACT

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Significantly more pedicel than apical portions of transversely cut maize (*Zea mays*) kernels were infected with *Fusarium moniliforme*. In contrast, *Aspergillus flavus* was detected more frequently in apical sections. In addition, *A. flavus* was isolated more frequently from the middle portions of inoculated kernels than from the pedicel end. Some ears (needle-inoculated with *A. flavus*) contained profuse fungal growth and sporulation on silk residues and kernel surfaces, but there was little penetration of uninjured kernels. The recovery of *A. flavus* and *F. moniliforme* from apical and pedicel portions of kernels was not affected by genotype or method of inoculation with *A. flavus* (pinbar or needle inoculation). *A. flavus* colonized a high percentage (83%) of the cobs in both inoculated and control ears of all maize genotypes. The fungus was recovered from a higher percentage of placental and sclerenchymatous tissue segments than pith tissue segments. *F. moniliforme* was isolated from cobs less frequently than *A. flavus* but appeared to be more uniformly distributed throughout them. Although the correlation (*r* values) of kernel infection by *A. flavus* with colonization of the placenta and sclerenchyma in the cob was statistically significant, the *r* values were quite low. The relatively low level of pedicel infection of kernels by *A. flavus* (7%) compared to combined infection of other kernel segments (45%) leads us to conclude that *A. flavus* penetrates maize kernels mainly through the pericarp.

Research on the infection of maize (*Zea mays* L.) by *Aspergillus flavus* Link:Fr. and subsequent aflatoxin production has centered on the presence of the fungus or the toxin in kernels (8). The association of insect damage to ears and kernels with infection by the fungus and the presence of aflatoxin in damaged kernels has been well documented (1,6,7,10,11,15). In several recent reports, however, infection by *A. flavus* and aflatoxin contamination were independent of physical damage to kernels (12,13,16). *A. flavus* can colonize silks (9,12,13) or damaged kernels and can penetrate undamaged kernels.

Hesseltine and Bothast (2) found that as silks senesced, they became a suitable medium for microbial growth and could provide entry for fungi into the ear. Jones et al (3) reported that exposed silk tissue may be colonized by *A. flavus* and provides a suitable infection court for inoculation of intact seeds. Marsh and Payne (9) found that conidia of *A. flavus* deposited on silks remained viable throughout pollination. Jones et al (3) showed that *A. flavus* can colonize silks, infect kernels, and produce aflatoxin in developing ears under insect-free conditions. Zummo and Scott (16) developed a method for inoculating ears with *A. flavus* that did not appear to injure kernels and could be used to select for resistance to kernel infection.

Koehler (5) reported that *Fusarium moniliforme* Sheld., the incitant of Fusarium ear rot, enters the ear through the silk channel, spreads within the ear on the silks, and infects isolated single kernels or groups of kernels in localized areas of the ear. Growth cracks in the pericarp or other damage enhances the infection of kernels by the pathogen, but the fungus can penetrate the pedicels of kernels.

Several other pathogens of kernels—*Gibberella zeae* (Schwein.) Petch, *Cephalosporium acremonium* Auct. non Corda, *Monilia* spp., and *Penicillium* spp.—also can colonize the ear via the silks (5). Two additional pathogens, *Diplodia zeae* (Schwein.) Lév. and *Nigrospora* spp., were more prevalent in the butt portion of the cob than in the kernels, indicating that these fungi probably invaded the ear through that portion of the cob. Thus, fungi may infect kernels or ears in the field via several possible avenues.

Attempts to identify sources of resistance to *A. flavus* in the field in the past have been constrained because the infection process is not clearly understood. There is evidence concerning the role of silk and the kernels but not on the role of the cob in the infection process. We undertook the studies described here to determine how kernels in maize ears inoculated with *A. flavus* become infected and to evaluate the role of the cob in that process in the presence of *F. moniliforme* in the field.

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MATERIALS AND METHODS

Field plots, harvesting, and handling methods. Kernels and cobs assayed in this study were grown in replicated single-row plots at the Plant Science Research Center, Mississippi State, MS. Plots were 5 m long, were spaced 1 m apart, and were overseeded and thinned to 20 plants spaced about 25 cm apart. The top ear of each plant was harvested 60 days after midsilk. Immediately after harvest, the ears were dried at 42 C for 7 days in a forced-air drier and then shelled mechanically or by hand. Kernels and whole shelled cobs from each plot were bulked, placed separately in paper bags, and stored at 6 C and 45% relative humidity until assayed.

Plots were harvested 60 days after midsilk because this allowed the ears to mature completely with minimal exposure of mature grain to an unfavorable environment. Because ears were dried immediately after harvest in a forced-air drier, then shelled and stored under conditions unfavorable for the growth of *A. flavus* and *F. moniliforme,* we assumed that all infection with either fungus occurred in the field and not in storage. We tested this assumption by assaying kernels from inoculated and uninoculated ears at harvest and after periods of storage at 6 C and 45% relative humidity. We did not find any increase in the percentage of infection during storage.

Inoculum source and production, inoculation techniques, and assays. Inoculum was produced from a lyophilized culture of *A. flavus* obtained from D. T. Wicklow, Northern Regional Research Center, Peoria, IL. The culture was grown for 14 days in 500-ml Erlenmeyer flasks containing 50 g of corncob grits (Grit-O'-Cobs, Maumee,

OH) and 100 ml of water. Conidia were washed from the surface of the grits with sterile distilled water containing two drops of Tween 20 (polyoxyethylene sorbitan monolaurate) per 100 ml. All inoculum for each year's inoculations was prepared at one time and stored at 4 C as an aqueous suspension of 10^8 conidia per milliliter. Inoculum was diluted to the desired concentration on each day of inoculation and kept on ice in the field until used.

Two inoculation techniques were used with *A. flavus.* In the pinbar technique described by King and Scott (4), a single 100-mm-long row of 35 stainless steel pins mounted in a plastic bar with 6 mm of the pointed ends exposed was dipped into a suspension containing 2×10^6 conidia per milliliter, lined up with the ear axis, and pressed through the husks into the kernels beneath. The pinbar injured only the husks and one row of kernels; the cob was not penetrated. Ears were shelled by hand, and only kernels in the row adjacent to the inoculated row were assayed. The needle-inoculation technique used a tree-marking gun fitted with a 14-gauge hypodermic needle 35 mm long with the tip opening plugged and three 1-mm holes drilled 6, 8, and 10 mm from the tip as described by Zummo and Scott (16). The needle was inserted through the husks, and 3.4 ml of inoculum containing 9×10^6 conidia per milliliter was injected over the kernels without visibly damaging them. Ears were mechanically shelled, kernels were bulked, and a random sample was selected for assay.

Only undamaged kernels were assayed for incidence of *A. flavus* and/or *F. moniliforme* on Czapek solution agar (CSA) amended with 7.5% NaCl (CSAS), which supports growth of both *A. flavus* and *F. moniliforme* but inhibits growth of bacteria and most other fungi. To eliminate surface microbes, kernels were dipped momentarily in 70% ethanol, submerged in 1.25% NaOCl for 3 min, and rinsed in sterile distilled water. Kernels were either cut in half transversely with small, sterile wire clippers and plated on CSAS in 100-mm petri plates (six kernels per plate) or cut transversely into five pieces approximately 2 mm long that were plated serially 10 mm apart (four complete kernels per petri plate). The plates were incubated for 5 days at 28 C.

Whole, undamaged cobs were immersed for 3 min in a 1.25% solution of NaOCl and cut into four equal parts aseptically. Three pieces of tissue approximately 4 mm^2 in area were taken from each of three freshly cut surfaces of the placenta (the membranous tissue on the surface of shelled cobs), sclerenchyma (the hard tissue between the placenta and the pith cylinder), and pith (Fig. 1). These tissue pieces were plated on CSAS and incubated at 28 C for 7 days.

Distribution of *A. flavus* and *F. moniliforme* in apical and pedicel fractions of kernels. In 1984, 120 kernels were sampled from each of 31 maize genotypes. The kernels were cut in half transversely and assayed on CSA and CSAS to determine the percentage of apical and pedicel portions that were naturally infected by *F. moniliforme.*

In 1987, 50 kernels were sampled from each of three corn hybrids that had been needle-inoculated with *A. flavus.* The kernels were cut in half transversely and assayed to determine the percentage of each kernel section that was infected with *A. flavus* and/or *F. moniliforme.* The test was a split-plot design with six replications; whole plots were a factorial of hybrids and inoculation treatments (inoculated or not inoculated), and subplots were kernel parts. One hybrid (Mp313E × SC54) had been previously identified as resistant to kernel infection by *A. flavus* (14), and the other two (GT106 × SC212 and Pioneer Brand 3369A) had been classified as susceptible. The test was repeated in a crop planted 30 days after the first one.

Distribution of *A. flavus* and *F. moniliforme* in various portions of kernels. In 1988, 48 kernels per ear were sampled from ears of Pioneer Brand 3369A that had been inoculated in one of four positions. Each kernel was cut transversely into five pieces and assayed for the presence of *A. flavus* and *F. moniliforme* as described above. The inoculation sites were in the silk channel or at the top, base, or shank of the ear. The test was a split-plot design with six replications; the inoculation treatments were whole plots, and kernel segments were subplots. The test was repeated in a crop planted 15 days after the first one.

Distribution of *A. flavus* and *F. moniliforme* in cobs of resistant and susceptible maize genotypes. In 1987, cobs were collected from ears that had been needle-inoculated. Five cobs per plot were assayed to determine the distribution of *A. flavus* and *F. moniliforme* in cob parts. In addition, cobs from two maize hybrids (Mo18W × Mp313E and SC54 × Tx601) previously identified as resistant to kernel infection by *A. flavus* and two hybrids (Mp68:616 × SC212 and GT106 × T202) identified as susceptible (16; N. Zummo and G. E. Scott, *unpublished*) were assayed to determine the distribution of *A. flavus* and *F. moniliforme* in cob parts. The test was a split-split plot design with six replications; inoculation treatments (needle or pinbar) were whole plots, hybrids were subplots, and cob parts were sub-subplots.

Cobs were also collected from plots of the 1988 tests with Pioneer 3369A described above. Five cobs per plot were assayed to determine the distribution of *A. flavus* and *F. moniliforme* in cob parts.

Statistical treatment of the data. The average percentages per plot of kernels, kernel parts, cobs, or cob parts infected with *A. flavus* or *F. moniliforme* were subjected to an analysis of variance appropriate to the experimental design. Least significant differences (LSDs) were computed.

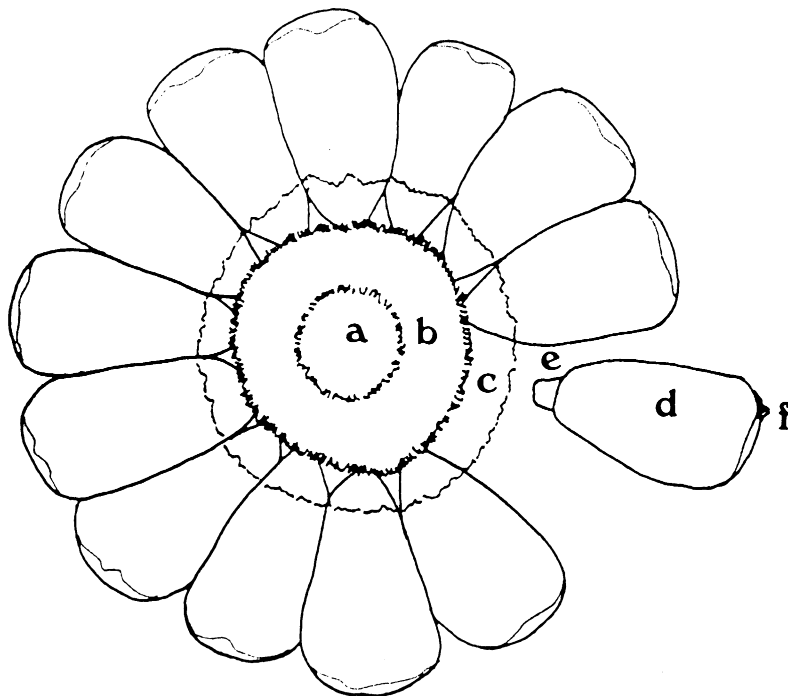

Fig. 1. Cross section of maize ear, showing parts of cob and kernel attachment: pith (a), sclerenchyma (b), placenta (c), detached kernel (d), pedicel (e), and silk scar (f).

RESULTS AND DISCUSSION

Recovery of *F. moniliforme* was greater from the pedicel end (32% on

CSA, 29% on CSAS) than from the apical end (11% on CSA, 4% on CSAS) of kernels from uninoculated ears ($P = 0.05$). This was also true for kernels sampled from ears inoculated with *A. flavus* (Table 1).

In another test with serially cut sections from ears of Pioneer Brand 3369A that had been inoculated with *A. flavus,* significantly more *F. moniliforme* was recovered from lower portions of kernels than from upper portions (Table 2). Infection was significantly greater in kernels from the second planting date, and the interaction between planting dates and kernel parts was significant.

The percentage of recovery of *A. flavus* was significantly higher from apical portions than from pedicel ends of kernels in 1987 (Table 1). Kernel infection was significantly higher in inoculated ears than in uninoculated ears in 1987, but the ratio of fungus recovery from apical and pedicel parts was not influenced by inoculation treatment. Differences in infection levels between planting dates were not significant in this test. In a second test with serially cut sections of kernels (Pioneer Brand 3369A), infection was significantly greater in the upper or apical portions of kernels than in the lower or pedicel portions (Table 2). The interaction between planting dates and kernel parts was significant in this test.

A. flavus seemed to grow out from small, discrete areas of the kernel pericarp and was recovered predominantly from single sections of transversely cut kernels rather than from multiple sections of the same kernel. In two tests with serially sectioned kernels (Table 2), the fungus was recovered from all five portions in only 0.8 and 1.3% of the kernels, whereas recovery from just one section was 7.0 and 7.2%. Moreover, the fungus usually grew out from a single point of the pericarp. With kernels from ears that were pinbar-inoculated, the fungus was recovered from only 0.2% of excised embryos and not at all from the scutellum of dissected kernels (*data not shown*).

In some needle-inoculated ears, *A. flavus* grew and sporulated profusely on silk residues and kernel surfaces inside the husks, but few of the kernels had been colonized. Although the fungus covered more than 25% of the surface area of some ears, fewer than 4% of the kernels were found to be invaded. If *A. flavus* enters through the silk channel of the ear, as has been reported for other fungi (5), it can apparently subsist on senescent silks within the husks (3) and may invade kernels directly through the pericarp, through weak spots in the seed coat such as silk scars or cracks, or through the pedicel sometime before harvest.

In these tests, *A. flavus* was recovered more frequently from the apical or silk ends of kernels than from the pedicel. The fungus was also recovered more frequently from the pericarp near the middle of the kernel than from the pedicel (Table 2). Thus, although *A. flavus* can invade kernels through the silks, it may also colonize the pericarp at random points. In contrast, *F. moniliforme* appeared to penetrate kernels mostly through the pedicel.

We recovered *A. flavus* from relatively high percentages (over 83%) of cobs of both inoculated and uninoculated ears of all maize genotypes. The recovery of *A. flavus* did not differ significantly between cobs sampled from needle-inoculated and pinbar-inoculated ears in 1987 (Table 3). Genotype GT105 × SC212 was colonized to a greater degree than the other two genotypes. Significantly more pieces from each cob tissue were infected with *A. flavus* in the crop planted in April than in that planted in May, but the planting date × cob tissue interaction was not significant. Needle inoculation significantly increased infection by *A. flavus* in all cob tissue compared to the uninoculated control (Table 3). With pinbar inoculation, infection was significantly greater in the sclerenchyma of inoculated cobs than in the uninoculated control; however, the percentage of placental segments infected with *A. flavus* in cobs from uninoculated

Table 1. Percentage of apical and pedicel portions of kernels internally infected with *Aspergillus flavus* or *Fusarium moniliforme* from ears (inoculated or not inoculated with *A. flavus*) of three maize genotypes in 1987[a]

		A. flavus			*F. moniliforme*		
Treatment	**Kernel portion**	**Test 1[b] (%)**	**Test 2[c] (%)**	**Mean (%)**	**Test 1[b] (%)**	**Test 2[c] (%)**	**Mean (%)**
Inoculated	Apex	19.0	27.4	23.2	5.4	4.1	4.8
	Pedicel	15.4	21.6	18.5	11.8	6.7	9.2
Uninoculated	Apex	6.4	5.9	6.1	0.9	3.2	2.1
	Pedicel	6.1	5.7	5.9	7.4	8.3	7.9
LSD[d] ($P = 0.05$)		3.2	2.3	1.8	2.4	2.5	1.8

[a] Each value is the mean of six replications of 50 kernels from needle-inoculated or uninoculated ears of three genotypes. The kernel parts were surface-sterilized, plated on Czapek solution agar amended with 7.5% NaCl, and incubated for 7 days at 28 C.
[b] Planted 16 April 1987.
[c] Planted 15 May 1987.
[d] Least significant difference.

Table 2. Percentage of transversely cut kernel sections infected by *Aspergillus flavus* and/or *Fusarium moniliforme* from replicated plots of Pioneer Brand 3369A inoculated with *A. flavus* in 1988[a]

	A. flavus			*F. moniliforme*		
Kernel part	**Test 1[b] (%)**	**Test 2[c] (%)**	**Mean (%)**	**Test 1[b] (%)**	**Test 2[c] (%)**	**Mean (%)**
Apical end	12.6	9.7	11.2	3.6	9.8	6.7
Upper intermediate	13.2	10.4	11.8	3.1	8.6	5.9
Center	12.3	12.8	12.5	5.7	14.3	10.0
Lower intermediate	9.3	9.1	9.2	9.8	20.2	15.0
Pedicel end	6.4	8.2	7.3	11.5	18.6	15.0
LSD[d] ($P = 0.05$)	2.0	1.8	1.3	1.4	2.0	1.2

[a] Each value is the mean of six replications of 48 kernels. The kernel parts were surface-sterilized, plated on Czapek solution agar amended with 7.5% NaCl, and incubated for 5 days at 28 C.
[b] Planted on 10 April 1988.
[c] Planted on 25 April 1988.
[d] Least significant difference.

Table 3. Percentage of placenta, sclerenchyma, and pith tissue segments infected by *Aspergillus flavus* from cobs of three maize genotypes inoculated with *A. flavus* in the field by the needle or pinbar methods of inoculation in 1987[a]

Inoculation method	**Placenta**	**Sclerenchyma**	**Pith**	**Average**
Needle	64.1	64.6	38.7	55.8
Pinbar	55.7	59.1	27.9	47.6
Control	44.9	37.4	20.3	34.2
Mean	54.9	53.7	29.0	
LSD[b] ($P = 0.05$)	11.5	11.4	11.3	9.8

[a] Each value is the mean of 45 cob pieces from each of 12 replicated plots planted on two dates. The pieces were surface-sterilized, plated on Czapek solution agar amended with 7.5% NaCl, and incubated at 28 C for 7 days.
[b] Least significant difference.

ears of GT106 × SC212 was significantly higher than that of similar tissue from pinbar-inoculated ears (*data not shown*). Infection was significantly higher in placental and sclerenchymatous tissue than in the pith of all genotypes, regardless of inoculation procedure.

Recovery of *A. flavus* from placental tissues of Mo18W × Mp313E and SC54 × Tx601, which are crosses of inbreds considered resistant to kernel infection by *A. flavus* (14,16), was significantly less than in Mp68:616 × SC212 and GT106 × T202, which are considered susceptible to kernel infection (Table 4). The overall percentage of cobs infected with *A. flavus* did not differ significantly for ears in the pinbar (97.5%) and needle (94.0%) inoculation treatments. Significantly more placental and sclerenchymatous tissues than pith tissue were infected by *A. flavus* (Table 4). The main effects of cob portions were significant, as indicated by LSDs. The main effects of hybrids were significant for infection by *A. flavus;* Mo18W × Mp313E and SC54 × Tx601 had significantly less cob infection than the other two hybrids. SC54 × Tx601 had the least kernel infection by *F. moniliforme,* and Mo18W × Mp313E had the highest level of infection. The interaction between hybrid and cob portion was significant for both fungi.

One cross, SC54 × Tx601, had significantly fewer cobs (13.5%) infected with *F. moniliforme* than the other hybrids. This hybrid also had significantly less infection of placenta and sclerenchyma with *F. moniliforme* than other genotypes (Table 4). On the other hand, these genotypes did not differ significantly in total kernel infection by *F. moniliforme* (range 11–16%; *data not shown*). *F. moniliforme,* though lower overall in incidence in cobs than *A. flavus,* was distributed more uniformly throughout the cob (Table 4).

In 1988, placental and sclerenchymatous tissues of Pioneer 3369A had significantly more infection by *A. flavus* than pith tissue (Table 5). Infection with *A. flavus* did not differ significantly between planting dates, but the interaction between dates and tissues was significant. With *F. moniliforme,* differences were significant for dates and tissues, and the interaction between dates and tissues was also significant.

Correlation coefficients (*r* values) were calculated to determine the association between the incidence of kernels infected with *A. flavus* and the incidence of placenta and sclerenchyma tissues infected by this fungus. Data from two plantings of a test with three hybrids, nine side-needle inoculation treatments plus a pinbar inoculation, and six replications (180 plots per test) were used for these calculations. For the first planting date, *r* values of 0.215 and 0.207 were obtained for correlations between kernel and placenta and kernel and sclerenchyma, respectively. Comparable values from the second planting were 0.452 and 0.320. All *r* values were statistically significant but relatively low.

The relatively low level of infection by *A. flavus* in pedicels of kernels (7%) compared to a relatively high level of combined infection of other kernel segments (45%) leads us to conclude that *A. flavus* invades maize kernels mainly through the pericarp.

Table 4. Percentage of placenta, sclerenchyma, and pith tissue segments infected with *Aspergillus flavus* or *Fusarium moniliforme* from cobs of four maize genotypes inoculated in the field with *A. flavus*[a]

Cob tissue assayed	Genotype	*A. flavus* (%)	*F. moniliforme* (%)
Placenta	Mo18W × Mp313E	57.1	30.0
	SC54 × Tx601	46.1	3.9
	Mp68:616 × SC212	75.8	13.3
	GT106 × T202	86.6	11.6
	Mean	66.4	14.7
Sclerenchyma	Mo18W × Mp313E	59.4	31.1
	SC54 × Tx601	55.0	3.3
	Mp68:616 × SC212	72.6	17.2
	GT106 × T202	65.9	19.4
	Mean	63.3	17.7
Pith	Mo18W × Mp313E	8.3	17.8
	SC54 × Tx601	7.8	1.7
	Mp68:616 × SC212	20.5	15.5
	GT106 × T202	11.1	14.4
	Mean	11.9	12.3
LSD[b] ($P = 0.05$) among hybrid × cob tissue		11.2	5.6
LSD ($P = 0.05$) among cob tissue means		5.6	2.8

[a]Each value is the mean of 45 cob pieces from each of six plots. The pieces were surface-sterilized, plated on Czapek solution agar amended with 7.5% NaCl, and incubated at 28 C for 7 days.

[b]Least significant difference.

Table 5. Percentage of placenta, sclerenchyma, and pith tissue segments infected with *Aspergillus flavus* or *Fusarium moniliforme* from ears of Pioneer Brand 3369A inoculated with *A. flavus*[a]

	A. flavus		*F. moniliforme*	
Cob tissue	Test 1[b] (%)	Test 2[c] (%)	Test 1[b] (%)	Test 2[c] (%)
Placenta	44.6	33.2	26.7	27.3
Sclerenchyma	67.2	37.4	14.8	28.8
Pith	4.3	24.4	2.2	7.5
LSD[d] ($P = 0.05$)	5.6	4.4	4.2	4.0

[a]Each value is the mean of 45 cob pieces from six replications. The pieces were surface-sterilized, plated on Czapek solution agar amended with 7.5% NaCl, and incubated at 28 C for 7 days.

[b]Planted 10 April 1988.

[c]Planted 25 April 1988.

[d]Least significant difference.

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