Histopathology of Sweet Corn Seed and Plants Infected with *Fusarium moniliforme* and *F. oxysporum*

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ABSTRACT

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Hyphae of *Fusarium moniliforme* were not found in stained and sectioned kernels of two sweet corn inbred lines. However, spherical to amorphous bodies occurred throughout the parenchymatous tip cap tissue and occasionally in the endosperm between the tip cap and the embryo. The fungus grew rapidly through the root system and crown of greenhouse-grown plants but made little additional growth until anthesis, when the rest of the plant including the cob and kernels was colonized rapidly. The root or mesocotyl epidermis was penetrated either directly or through wounds. The outer cortical layers collapsed and intercellular materials and globular bodies were present prior to fungal invasion. Hyphae were mainly intercellular and eventually penetrated throughout the tissues. In the stem, protoxylem vessel elements became occluded, intercellular material accumulated in the parenchyma, parenchyma cells around vascular bundles collapsed, and hyphae penetrated throughout the cells. Occluded xylem vessel elements also occurred in the leaf and shank. Mycelium was evident in the outer edge of the cob. Tissues of the floret or the fertilized ovary, or both, collapsed. Globular bodies were found throughout the infected tissue.

Additional key words: Zea mays, Gibberella fujikuroi.

Corn, *Zea mays* L., is susceptible to attack by *Fusarium moniliforme* Sheldon, the imperfect state of *Gibberella fujikuroi* Saw., and several other *Fusarium* species that can cause stalk rot, leaf spot, kernel rot (7,32), seed rot, damping-off, and seedling blight (8). Usually sweet corn is more susceptible than dent corn to seed rot and seedling blight (32). Dent seed corn infected with *F. moniliforme* usually results in a lower yield than does uninfected seed (35). Edwards (4) reported that *F. moniliforme* also is responsible for reduced germination of dent corn seed.

Corn plants in the field may become infected through wounds (17) or internodal cracks (2). The European corn borer (*Ostrinia nubilalis* Hbn.) has been reported to carry *F. moniliforme* conidia and may inoculate plants when feeding (31). Spores frequently lodge between the leaf sheath and the stalk, an area providing ideal conditions for spore germination (14), and penetration of the stalk at this point has been reported (7,17,35).

Some researchers (17,19,21) believed that planting dent corn seed infected with *F. moniliforme* is not important in the disease cycle while others (7,30,35) felt that it is important. Valleau (34) suggested that the dormant stage of the fungus in seed is hyphal in both sweet and dent corn while Naqvi (23) and Lucado (19) reported a mycoplasmal stage as described by Eriksson (5) in sweet corn seed.

Several researchers have reported that infection is seminal (15,16,20,24). Koehler (15,16) indicated that the pathogen entered in the region of the silks, spread to the bracts and pedicels through the vascular cylinder, and finally spread into the shank. Internal kernel infection did not become established until the ears were approaching maturity.

Other research workers also considered that ear infections probably occur by systemic infection. Christensen and Wilcoxson (2) suggested that a combination of local and systemic infection probably occurs. Kingsland and Wernham (14) suggested that fungal invasion may occur through vascular tissues leading from the main stalk to the rudimentary ears. When windborne spores lodge between the leaf sheath and stalk, invasion of the vascular parenchyma of the leaf and ear could occur since the leaf and stalk tissues are continuous at the sheath base. The presence of *F. moniliforme* inside the kernel and its wide ramification in all parts of seemingly healthy mature plants suggests that infection is systemic (7). Salama and Mishricky (30) suggested that immature seeds became infected with *F. moniliforme* through the placentochalazal region.

Usually the plant is infected during or immediately after seed germination (35). The fungus may enter the cotyledonary plate region when the stem bud breaks through the pericarp or when the plumule breaks through the apex of the coleoptile. Direct

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penetration of emerging adventitious roots and of the primary radicle when they break through the coleorhiza also has been reported (35).

Over the past 20 yr the incidence of stalk rot in sweet corn seed fields has increased with a concomitant decrease in yield and seed quality. Since *F. moniliforme* is readily isolated from such plants, this study was initiated to determine: when and how sweet corn seed is infected with *F. moniliforme*, the form and location of the fungus in the infected seed, and the extent of the invasion from the seed into tissues of the seedling and the mature plant by the fungus.

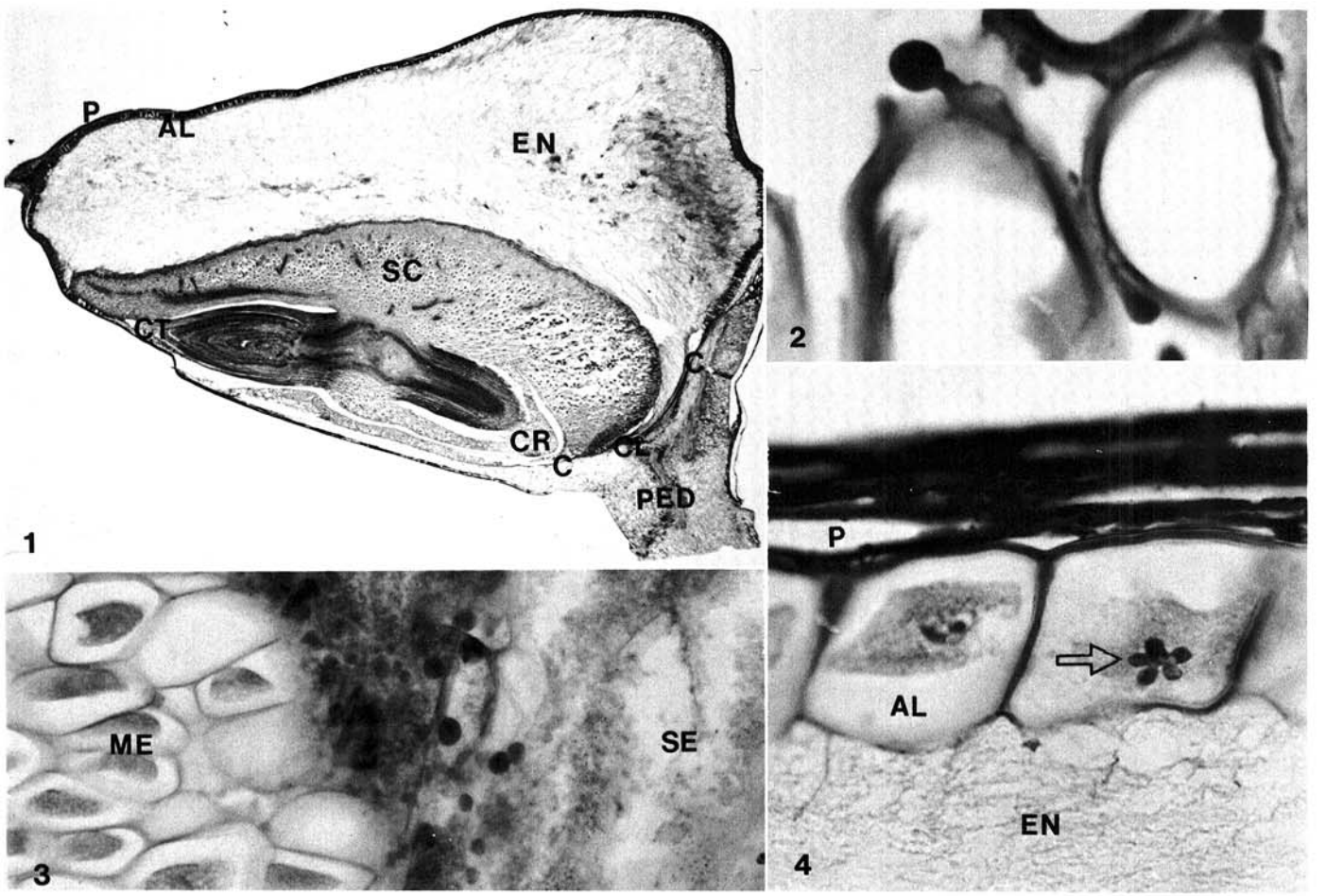
MATERIALS AND METHODS

Kernel histology. Seed of two widely used inbred lines of sweet corn obtained from the Crookham Company, Caldwell, ID 83605, were used in this study. One line, 5Q, is considered to be highly susceptible to stalk rot caused by *F. moniliforme* and the second inbred, I453, is considered resistant because it normally remains green and erect several days longer than more susceptible material of the same maturity class.

Dried kernels of both inbred lines were placed in beakers of sterile distilled water at 6 C for 2 wk, and cut either transversely above the embryo or longitudinally on either side of the embryo to allow better infiltration of fixative. The kernels were placed in formalin-aceto-alcohol fixative (FAA) formulated with 50% ethanol (12) for 3 days and dehydrated in a tertiary butyl alcohol series (12). Infiltration was done in a vacuum oven at −1.4 to −1.7 bars and the tissues were then embedded in Paraplast (Lancer, Sherwood Medical, St. Louis, MO 64500). Embedded specimens were softened 48 hr in a 10% glycerol solution with 1% Dreft (sodium lauryl sulfate) (1) prior to sectioning at 10 μm on a rotary microtome. Sections were mounted on chemically cleaned slides with Haupt's adhesive (12). Stains used included Johanson's quadruple stain, safranin and fast-green, and Fleming's triple stain (12). In Fleming's triple stain, 1% aniline blue in 95% alcohol was substituted for the aqueous methyl violet with appropriate changes in the preceding alcohol rinses.

Selected sections were tested for pectin by the iron absorption method (29). The presence of cellulose was determined by the zinc-chlor-iodine and the IKI–H_2SO_4 methods (29). The lacmoid reaction was used to test for callose (29). Phloroglucinol was used to determine the formation of wound gum and lignin (29). The presence of lignin was determined by the Clorox–$Na_2SO_2 \cdot 7H_2O$ method (12).

Seedling infection. Surface disinfested kernels were planted, three per pot, in 30.5-cm-diameter clay pots filled with a steam-treated mixture of peat, perlite, and soil (1:1:1, v/v) and grown in the greenhouse. Kernels of each inbred line were surface disinfested in 10% Clorox (5.25% sodium hypochlorite) plus 0.01% Dreft for 2 min and rinsed twice with distilled water. An equal number of kernels were left untreated. Beginning 3 wk after planting, three or four plants from the surface-disinfested treatment and the nondisinfested treatment were sampled weekly up to 9 wk, then again at 13 or 15 wk. The whole plant or pieces up to 30.5 cm long were surface disinfested as described above. Pieces 5 mm long were removed aseptically from 27–31 plants in each treatment at regular intervals and numbered. Alternate pieces were placed in FAA in vials or plated on modified Nash's medium (22). After 5 days, fungi



Figs. 1–4. 1, Longitudinal section of a kernel of sweet corn, *Zea mays,* inbred line 5Q taken from a 15-wk-old plant (×11). **2,** Portion of a longitudinal section through the outer edge of the tip cap of a kernel of sweet corn inbred line 5Q. Note the varying shapes of the globular bodies and the large stalked globular body (×696). **3,** Portion of a longitudinal section of a kernel of sweet corn inbred line 5Q showing globular bodies in the starchy endosperm next to the marginal endosperm (×465). **4,** Portion of a longitudinal section of a kernel of sweet corn *Zea mays*, inbred line I453 showing the aleurone layer with abnormal nuclei (arrow), pericarp, and endosperm (×435). Chalaza (C), closing layer (CL), pedicel (PED), pericarp (P), aleurone layer (AL), endosperm (EN), coleoptile (CT), scutellum (SC), coleorhiza (CR), starchy endosperm (SE), marginal endosperm (ME).

that grew from the plated pieces were identified (33).

Plant histology. The fixed pieces were dehydrated in a tertiary butyl alcohol series (12), infiltrated, and embedded in Paraplast. After being softened for 24 hr in a solution of 1% Dreft in 10% glycerol (1), the embedded specimens were sectioned, mounted, stained, and photographed in the same manner as the kernels. Selected slides were tested for the presence of wound gum, lignin, and pectin (29).

RESULTS

Kernel histology. The anatomy of the kernels of both inbred lines was the same. Johann's (11), Kiesselbach and Walker's (13) and Randolph's (28) descriptions of dent corn seed could apply equally well to sweet corn seed (Fig. 1).

Fungal hyphae were not visible in any part of the kernels; however, spherical to amorphous bodies occurred throughout the parenchymatous tip cap (pedicel) tissue. Globular bodies appeared to be more frequent in the susceptible line 5Q than in the resistant line I453. Cap tissue with these bodies did not differ anatomically in other respects from cap tissue without them. The bodies showed no evidence of nuclei or cytoplasm, and in all cases their outer surfaces were smooth. When they were contiguous with the cell wall their shape was amorphous; otherwise, they were stalked (Fig. 2) with the upper part of the body being spherical. Occasionally these bodies also were present below the embryo between the starchy and marginal endosperm (Fig. 3). Globular bodies were found on the cell walls of the scutellum, and in some cases, many were found in the scutellum. These globular bodies were found in the embryo or the endosperm only if they also were present in the tip cap.

Nuclei in the aleurone layer of seeds with globular bodies occasionally appeared abnormal in both inbred lines. At maturity, nuclei of normal aleurone layer cells do not divide (28) and contain one spherical nucleus per cell. However, some aleurone layer cells in tissue with globular bodies contained up to four nuclei per cell. Abnormal nuclei consisting of a cluster of lobes were observed more frequently than were multiple nuclei (Fig. 4). Cells with normal nuclei often were adjacent to cells with abnormal nuclei.

Histochemical tests. Histochemical tests did not provide conclusive evidence as to the nature of the globular bodies. Tests for gum and lignin were negative. Pectin and callose tests ranged from negative to weakly positive depending on how the surrounding tissue reacted. If the cell walls with which the bodies were associated reacted positively, the globular bodies also gave a positive reaction. If the plant cell walls did not react, the globular bodies did not react. In the test for cellulose, the globular bodies remained clear even though the surrounding tissues of both the tip cap and embryo stained light blue. In the IKI–H_2SO_4 test for cellulose, the globular bodies turned light yellow, which is the color reaction for lignin. Since in the majority of histochemical tests the globular bodies reacted in the same manner as did the surrounding plant tissues, this suggests a relationship between the bodies and the plant tissue.

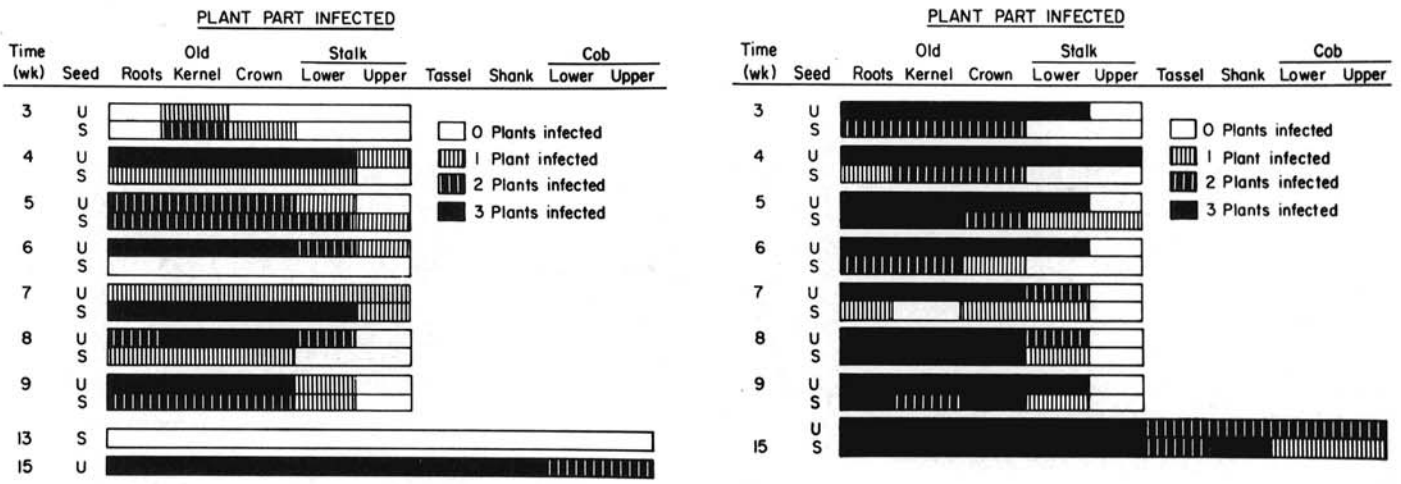
Seedling isolations. In the greenhouse test, 53% of the plants from surface-disinfested seed of inbred line 5Q were infected with *F. moniliforme* and 20% were infected with *F. oxysporum* (Schlect.) Snyder & Hansen while 14% of the plants from similarly treated seed of inbred line I453 were infected with *F. moniliforme* and 38% were infected with *F. oxysporum*. All plants from nondisinfested kernels of inbred line 5Q were infected with *F. moniliforme* and 12% also were infected with *F. oxysporum*. When grown from nontreated seed, 65% of the plants of inbred line I453 were infected with *F. moniliforme* and 12% with *F. oxysporum*.

The movement of the fungi through the plants showed the same pattern in plants of each inbred line whether or not the seed had been surface disinfested (Figs. 5 and 6). The fungi ramified rapidly through the root system and the crown area. The lowest quarter of the stalk also was invaded early. By 9 wk after planting, and prior to tasseling, the fungi had made little progress beyond the lower portion of the stalk. The plants appeared to outgrow the fungus until anthesis when the entire plant appeared to be invaded by the fungus. The fungi reached the same height in the cob and stalk at identical times.

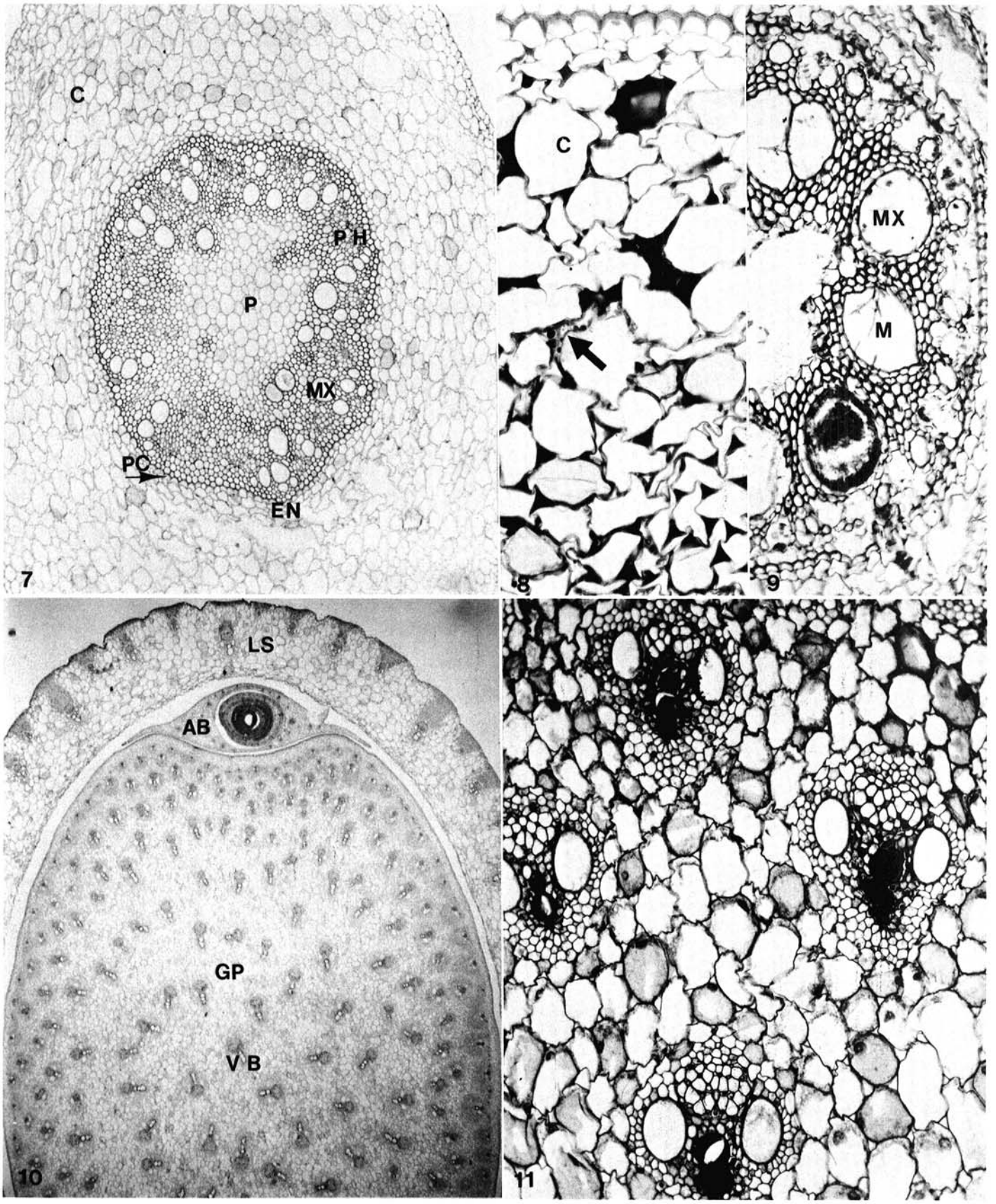
Plant histology. Since the anatomy of all plant parts of both inbred lines was identical and their reactions to *F. moniliforme* and *F. oxysporum* also were the same, the development of the two fungi in the two inbred lines will be presented jointly.

The anatomy of stained, transverse sections of corn roots fits previous descriptions (6) (Fig. 7). The first evidence of fungal infection in the root was intercellular material in the outer cortex (Fig. 8). Although the infection process was not observed, it may have taken place either systemically or through natural openings made when the radicle ruptured the coleorhiza or when the lateral roots grew through the cortex. The cortical parenchyma cells collapsed and mycelium ramified through the cortex. Since the endodermis is heavily suberized this may be a temporary barrier to invasion of the stele (35). However, in young roots with an as yet undeveloped casparian strip, the fungus readily enters the stele (Fig. 9). After ramification of hyphae through the tissue, xylem vessel elements either became filled with pectic material or were invaded by the hyphae.

The anatomy of the healthy mesocotyl is essentially the same as that of the root except that the endodermis contains less suberin (35). The sequence of histological changes that occurs after



Figs. 5–6. Numbers of sweet corn plants and plant parts from which *Fusarium moniliforme* was recovered. The plants were grown either from seed that had been surface disinfested (S) for 5 min or from untreated seed (U) planted in steamed soil in the greenhouse. A minimum of three plants grown from treated seed and three plants grown from untreated seed were examined at each time interval. The plant parts were surface disinfested in Clorox and plated on Nash's Medium. After 5 days the plates were examined, and any fungi present were transferred to other media and identified. **5,** Inbred line I453; and **6,** inbred line 5Q.



Figs 7–11. 7, Portion of a transverse section of a healthy root of a plant of sweet corn inbred line 5Q from a 5-wk-old plant (×95). **8,** Transverse section of a root of a 4-wk-old plant of sweet corn inbred line 5Q naturally infected with *Fusarium moniliforme* showing collapse of some cortical cells. Note globular bodies (arrow) and the large amount of intercellular material present (×165). **9,** Portion of a transverse section of a root of a 7-wk-old plant of sweet corn inbred line 5Q naturally infected with *F. oxysporum* showing collapsed cortex, and metaxylem vessel elements, one of which is occluded and the others contain mycelium (×155). **10,** Portion of a transverse section of a healthy stem of a 7-wk-old plant of sweet corn, *Zea mays*, inbred line I453 showing leaf sheath, axillary bud, ground parenchyma, and collateral vascular bundles (×15). **11,** Portion of a transverse section of a stem from a 7-wk-old plant of sweet corn inbred line 5Q naturally infected with *Fusarium moniliforme* showing occluded protoxylem vessels (×157). Cortex (C), endodermis (EN), pericycle (PC), metaxylem vessel elements (MX), phloem (PH), pith (P), mycelium (M), leaf sheath (LS), axillary bud (AB), ground parenchyma (GP), collateral vascular bundles (VB).

infection follows the same basic pattern as in the root. The cortex is the first tissue affected. The fungus may enter directly through the epidermis or through ruptures produced in the cortex by the emergence of adventitious roots. The outer layers of the cortex appear to collapse first and intercellular material and globular bodies are present prior to fungal invasion. Eventually the hyphae ramify throughout the cortex, mainly intercellulary. As in the root, tissues of the stele eventually become infected. Although xylem vessel elements became occluded, no hyphae were seen.

Anatomy of the healthy corn stalk is described by Esau (6) (Fig. 10). The first observable difference in the anatomy of infected stalks was the occlusion of protoxylem vessel elements (Fig. 11). Later, intercellular material accumulated among the parenchyma cells. The parenchyma cells around the vascular bundles collapsed and the hyphae ramified throughout these cells (Fig. 12). Eventually the hyphae penetrated the sclerenchyma sheath and grew through the xylem vessel elements. Occasionally, at the edge of the stem, the fungus ruptured the epidermis and sporulated.

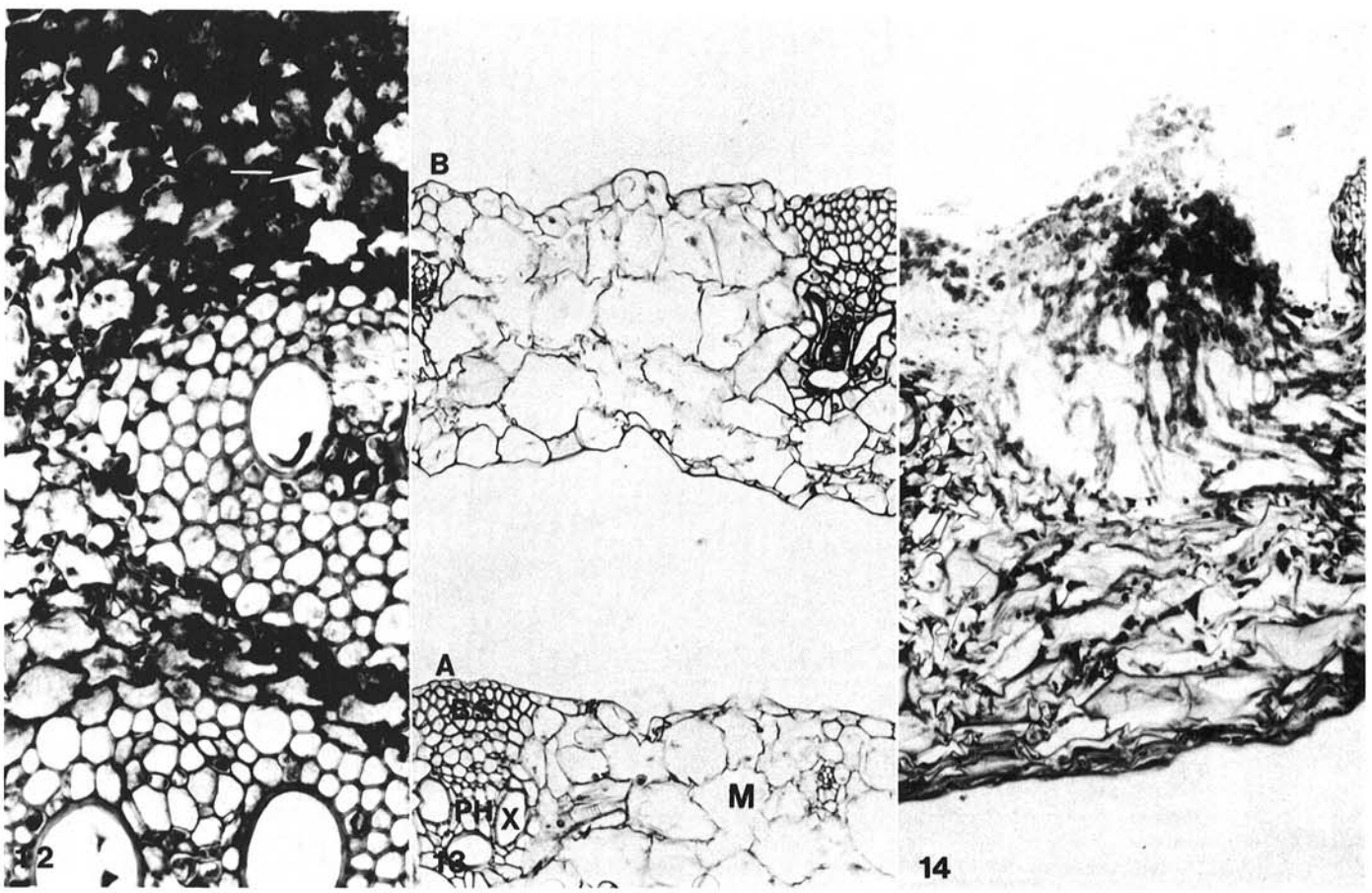
As in the stalks, the first visible evidence of fungal infection in leaves was occluded xylem vessel elements. Next, intercellular material accumulated in the mesophyll, and hyphae became established throughout this region. Eventually parenchyma cells collapsed, first in the outermost leaves and then in the innermost ones (Fig. 13). Sporulation of the pathogen takes place just prior to cell collapse. Hyphae may emerge through a stomate or rupture the epidermis and then sporulate. In some cases it appears as if a stroma is formed (Fig. 14). The spores from these stroma can move upward betweem the unexpanded leaves (Fig. 15), perhaps through the capillary action of free water between the leaves.

The anatomy of the ear shank was nearly identical to that of the stalk. Occluded xylem vessel elements were the first sign of fungal invasion. Globular bodies also were present at sites where there was intercellular material. The reactions of the shank to the fungi were the same as those of the stalk.

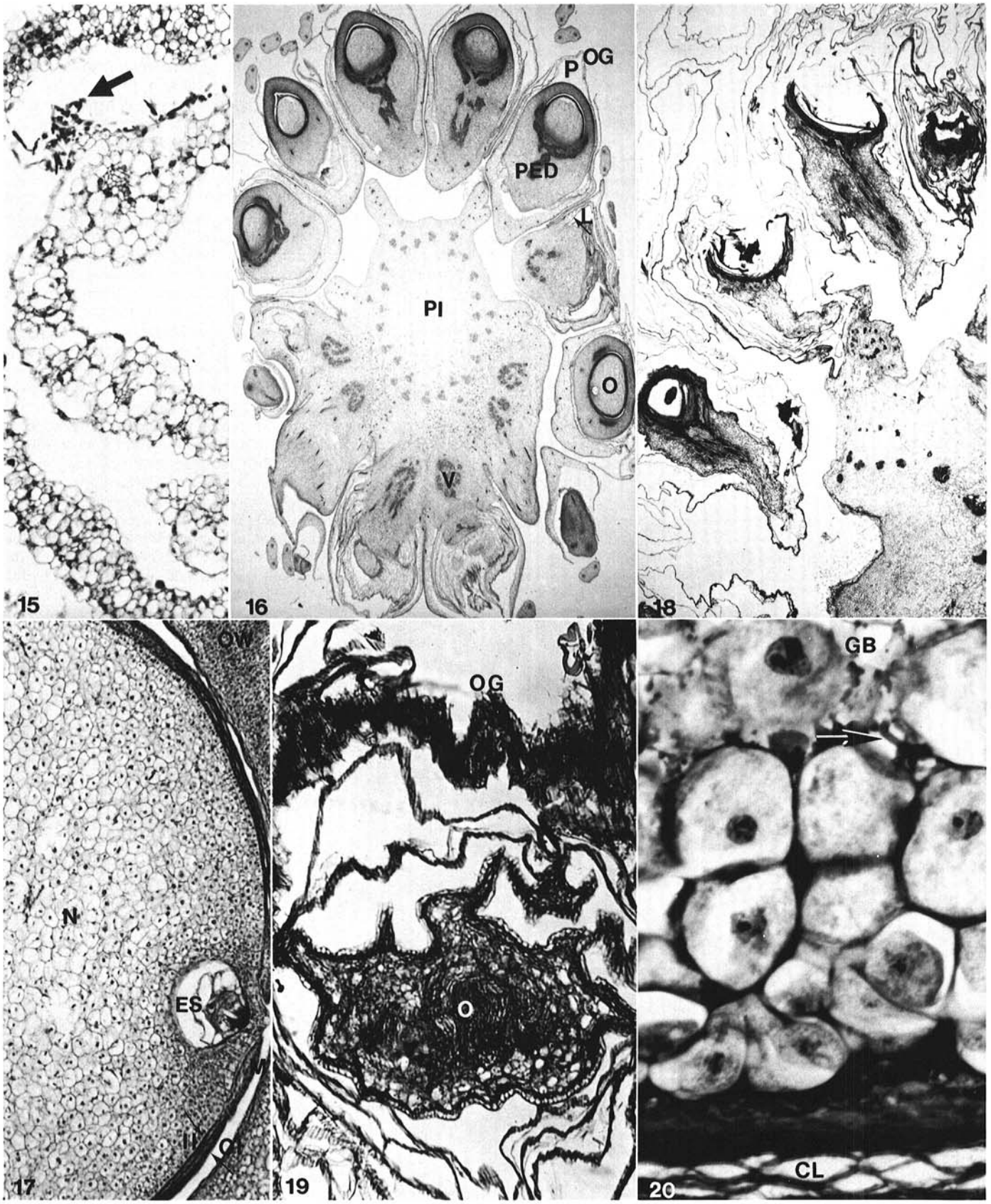
The corn ear consists of an elongated cylinder of hardened tissue (the cob) on which pairs of fertile spikelets are attached, usually in longitudinal rows (Figs. 16, 17, and 20). The effects of natural infection of *Fusarium* sp. on the cob and developing kernels can be striking (Fig. 18). Either the tissues of the floret, the fertilized ovary, or both, collapse (Fig. 19). Mycelium was present around the outer edge of the cob itself, in the collapsing ovaries, and even in the outer glumes. Sporulation occurred on all of these tissues.

The closing layer of the kernel forms by suberization of the integuments (15). If the pathogen is to enter the kernel, it presumably must do so before the closing layer forms, since there is no direct connection between the vascular system of the pedicel and the ovary. Intercellular material is the first sign of fungal invasion. Globular bodies also can be found in the embryo. After this, the hyphae grow intercellularly in the embryo itself (Fig. 20).

The results of the histochemical tests were the same for all the plant tissues. The material occluding the xylem vessel elements gave a strong positive reaction to the iron absorption test for pectin. This substance gave a negative reaction to the phloroglucinol test for gum and lignin, and the orcinol test for gum. The intercellular material gave a weak reaction to the phloroglucinol test and a negative reaction with the iron absorption test for pectin. The globular bodies gave a negative-to-weak reaction with the phloroglucinol and iron absorption tests and a negative reaction with the orcinol test. It appears that the material occluding the xylem is primarily a pectic substance as is the intercellular material,



Figs. 12–14. 12, Portion of a transverse section of a stem from a 7-wk-old plant of sweet corn inbred line 5Q naturally infected with *F. moniliforme* showing intercellular material between the ground parenchyma cells and mycelium (arrow). The cells between the vascular bundles have collapsed (×158). **13,** Transverse sections of a healthy leaf (A) and a leaf naturally infected with *F. oxysporum* (B) from a 7-wk-old plant of sweet corn inbred line I453. The infected leaf shows cell collapse and occluded xylem vessel elements (×190). **14,** Portion of a transverse section of a leaf taken from a 7-wk-old plant of sweet corn of inbred line I453 naturally infected with *F. oxysporum*. The fungus has ruptured the epidermis to form a stroma and sporulate (×203). Xylem vessel elements (X), phloem (PH), bundle sheath (BS), mesophyll (M).



Figs. 15–20. 15, Transection of unexpanded leaves at the apex of a 4-wk-old sweet corn plant of inbred line 5Q naturally infected with *F. moniliforme*. The leaves show no evidence of invasion by fungi. Note the spores (arrow) between the leaves (×197). **16,** Transverse section of a healthy cob taken from a 15-wk-old plant of sweet corn, *Zea mays,* inbred line I453 (×13). **17,** Longitudinal section of a healthy ovary taken from a 13-wk-old plant of sweet corn inbred line I453 ~2–4 days after fertilization (×109). **18,** Portion of a transection of a cob taken from a 15-wk-old plant of sweet corn inbred line 5Q naturally infected with *F. moniliforme* showing occluded xylem vessel elements in the cob and pedicels, collapsed tissue in all areas and necrotic ovaries (×13). **19,** Cross section of an ovary from a 15-wk-old plant of sweet corn inbred line 5Q naturally infected with *F. moniliforme*. All tissues of the ovary are collapsed and necrotic (×103). **20,** Portion of a transverse section at the base of a kernel taken from a 15-wk-old plant of sweet corn infected with *F. moniliforme* showing intercellular material in the scutellum above the closing layer, globular bodies, and a hyphal fragment (arrow) (×555). Pith (PI), vascular elements (V), pedicel (PED), outer glumes (OG), palea (P), lemma (L), ovary wall (OW), embryo sac (ES), nucellus (N), inner integuments (II), outer integuments (OI), micropyle (M), ovary (O), closing layer (CL), globular bodies (GB).

although some gum also is present. The globules seem to be composed of the same material as the walls with which they are associated.

DISCUSSION

The majority of kernels of line 5Q and some kernels of inbred line I453 grown in the greenhouse appear to have carried the fungus internally. Internal infection of both chickpea seeds with *F. oxysporum* f. sp. *ciceri* (10) and corn seeds with *F. moniliforme* (26) has been reported. Since surface disinfestation lowered the amount of fungus isolated from kernels and from plants grown in the greenhouse, external infestation also may have been a factor.

In kernels from plants grown in the greenhouse, mycelium was present in the basal part of the embryo directly above the closing layer (Fig. 20). The fact that no mycelium was observed in the original seed from Idaho was puzzling; however, vacuum embedding procedures may have caused the mycelium to collapse, thus making it difficult to see. The pathogen grows from infected kernels upward in the plant to the cob where it infects developing kernels. The mycelium would have to grow through the pedicel to enter the ovary since there is no direct vascular connection between the cob and ovule (6). The presence of mycelium in the tip cap of mature, dry seed would be difficult to detect, not only due to the possible effects of the vacuum embedding, but also because the tissue is desiccated and distorted at maturity. Mycelium probably does survive in the tip cap tissue.

The structures Naqvi (23) and Lucado (19) described as the mycoplasmic stage of *F. moniliforme* were observed in the material prepared for this study. The small rounded bodies Naqvi found in the cotyledon appeared to be the same as the globular bodies observed in the scutellar region (Fig. 3). Naqvi stated that when the host cell walls were breaking down, the mycoplasms associated with the cells were increasing in size. The results of the histochemical tests performed in this study indicated that the globular bodies may result from the action of cell wall-degrading enzymes produced by the fungus. Weak positive reactions for pectin, callose, and cellulose and negative results for lignin and cutin were obtained for the globular bodies; the reactions were similar to those of the host cell wall with which they were associated. Paul and Schönbeck (25) observed similar structures that they referred to as globular or spherical bodies associated with *F. moniliforme*-infected corn roots. In a report on the effects of the root-knot nematode *Meloidogyne incognita acrita* on okra seedlings, Littrell (18) reported similar bodies that were either attached to cell walls or suspended in the cytoplasm. The photographs of these masses looked much like the globular bodies present in the kernel and tip cap. The histochemical tests he performed showed that the spherical masses of material reacted in the same way as the cell walls.

Naqvi (23) further described abnormally large multilobed nuclei. Naqvi stated that the mycoplasmal body, which was darker staining, resided in one of the lobes. This description corresponds to the abnormal nuclei present in the aleurone layer of kernels in which globular bodies had occurred in the tip caps. The dark spherical bodies present in the lobed nuclei are probably nucleoli. Abnormal nuclei such as these occur in the giant cells of roots of plants infected with root-knot nematodes (3).

The mode of infection of seedlings would depend on the location of the overwintering mycelium. If mycelium was present in the embryo, hyphae could ramify throughout the seed. In such a case the pathogen could be present in all the tissues even before the time of germination.

Four plants grown in the greenhouse, three of inbred line I453 and one of inbred line 5Q, exhibited seedling blight, stunting, and a poorly developed root system caused by *F. oxysporum*. *Fusarium oxysporum* was isolated more frequently from plants grown from surface-disinfested seed, which suggests that it occurs within the kernel. Only six plants of both inbred lines from seeds that had not been surface sterilized were infected with *F. oxysporum* compared to 13 plants from surface-sterilized seed of both lines. Some researchers (36) considered *F. oxysporum* to function as a root rotting fungus of corn when the roots are wounded and temperatures are relatively high.

If the fungus is on the surface of the pericarp or within the tissue of the tip cap, infection would not occur until during or after germination. Griffiths and Lim (9) suggested that pectolytic enzymes are most important during the early stages of infection, resulting in the occlusion of the xylem vessel elements with pectic substances as an early sign of infection. The enzymes would affect tissues in advance of the hyphae. Because of the occlusion of the xylem vessels with pectic materials, hyphae would not become established there. Only in the roots was mycelium present in the xylem vessel elements (Fig. 9). Since the roots are the first tissues to appear on germination, perhaps hyphae are able to rapidly invade the root, including the xylem vessel elements. Growth upward through the parenchyma tissue takes longer than growth up through the vascular system. Other early signs of enzymatic activity are the presence of intercellular material which gives a positive reaction for both pectic substances and gum, and globular bodies.

This study has shown that sweet corn seed can be infected and infested with *F. moniliforme* and to some degree, infected with *F. oxysporum*. The form of infection appears to be mycelial rather than mycoplasmal. In addition to local infections, corn plants can become systemically infected from infected seed as seen from the greenhouse study. Infested kernels result in plants that become infected during or after seed germination. The fungus grows up through the parenchyma tissues of the stalk and into the cob.

The localization of *F. moniliforme* infection to the basal portion of the corn stalk until the time of flowering, and subsequent rapid ramification of the mycelium throughout the plant, is a pattern that has been reported by other workers (27). If this rapid ramification occurs early in the season, the developing kernels may be destroyed by the pathogen. If the fungus does not reach the ears until the kernels are more mature, the seeds will not be killed although they may carry some internal mycelium. *Fusarium moniliforme* could be carried to uninfested regions of the country in sound-appearing, internally infected sweet corn seed.

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