Histopathology of Cotton Boll Rot Caused by *Colletotrichum capsici*

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ABSTRACT

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The histopathological aspects of cotton boll rot caused by *Colletotrichum capsici* were studied by using scanning electron and light microscopy. Class I (10- to 12-day-old) greenhouse-grown cotton bolls of two cultivars were resistant to invasion by *C. capsici* and exhibited a hypersensitive response characterized by restricted lesions, plasmolysis and collapse of host epidermal cells, and pigment accumulation in subepidermal parenchyma. Histochemical tests failed to detect gossypol or hemigossypol associated with the hypersensitive lesions. Class II bolls (34–36 days old) were susceptible to invasion. Nonlignified pericarp cell walls became swollen and did not stain. Lignified pericarp tissues were more resistant to degradation and retained their staining properties. The distinctive appearance of bolls rotted by *C. capsici* is probably due to the differential degradation of pericarp tissues.

Additional key words: *Gossypium hirsutum*

A boll rot of cotton (*Gossypium hirsutum* L.) caused by *Colletotrichum capsici* (Syd.) Butler & Bisby was first reported in India by Chopra et al (5) and in the U.S. by Snow and Mertely (16). Where conditions of constant high relative humidity exist in Louisiana, *C. capsici* has been an important boll-rotting organism, causing up to 50% estimated loss in some fields in the southernmost cotton-producing parishes (16).

Boll rot caused by *C. capsici* is distinguished from cotton anthracnose, which is caused by *C. gossypii* (= *Glomerella gossypii* Edgerton), by the macroscopic appearance of the rotted bolls and by morphological differences between the fungi. Under favorable conditions, lesions produced on bolls infected by *C. capsici* rapidly encompass the entire boll (12) and thus differ from the sunken, restricted, anthracnose-type lesions. Bolls infected with *C. capsici* do not fully dehisce as do healthy bolls, and the lint often remains in the locule as a compact mass, a condition commonly known as "tight-lock" (16). Pericarp, endocarp, lint, and seed are often darkened by conidiomata and other fungal stromatic structures. All that remains of badly rotted boll pericarp tissue is a reticulum of vascular tissue and fibers. Nonlignified tissues are often completely destroyed. The skeletonized remains of rotted pericarp tissue give the boll rot caused by *C. capsici* the distinctive appearance that distinguishes it from boll rots caused by other fungi.

The distinctive appearance and effects of boll-rotting fungi on cotton lint and the behavior of cellulose-decomposing fungi in association with cotton lint were described by Simpson and Marsh (15). They reported that hyphae of boll-rotting fungi that decompose cellulose wind around lint following the spiral of the cell wall, penetrate into lint lumina, and cause helical fissures in the lint cell wall. Similar observations of *C. capsici* associated with cotton lint were made by Roberts and Snow (11).

Inoculation studies with *C. capsici* (12) revealed differences in susceptibility to infection between young (10- and 20-day-old) bolls and older (30- and 40-day-old) bolls similar to those reported for Fusarium boll rot of cotton (17). Bolls ≤20 days old appeared to be resistant to infection, forming only minute, limited, rust-colored lesions that became darker with age but did not enlarge. Bolls over 20 days old were susceptible to infection. Dark, blue-black lesions appeared 3–7 days after inoculation and rapidly encompassed the entire boll.

The purposes of this study were to describe the development of boll rot caused by *C. capsici* using light and scanning electron microscopy, to examine histologically the hypersensitive reaction of inoculated resistant bolls, and to document by histology the changes that result in the distinctive appearance of cotton bolls rotted by *C. capsici*.

MATERIALS AND METHODS

Inoculations. Greenhouse-grown Stoneville 213 cotton bolls 10–12 days old (class I) and 34–36 days old (class II) were excised, and the bracts and bracteoles were removed. The bolls were surface sterilized in 0.9% NaOCl, rinsed twice in sterile distilled water, and placed on glass rings in sterile moist chambers.

Agar plugs bearing conidia used for inoculations were cut with a sterile 6-mm-diameter (no. 3) cork borer from 7-day-old cultures of *C. capsici* (ATCC 24179) grown on half-strength Difco PDA at 28 C under continuous fluorescent illumination. Conidial suspensions were prepared from sporulating agar cultures with distilled water and diluted to obtain 65–68% light transmittance with a Spectronic 20 colorimeter (Bausch & Lomb Co., Rochester, NY 14625) at 500 nm. One drop of suspension from a Pasteur pipette was applied to each boll inoculated by this method.

Fifty bolls of each age class were inoculated without wounding by placing agar plugs or drops of conidial suspensions on or between capsular sutures. One drop of sterile distilled water was placed on each agar plug to provide adequate moisture for conidial germination. Ten bolls of each age class were surface sterilized but not inoculated and served as controls. Cauquil and Ranney (4) and Roncadori (13,14) reported the occasional presence of an internal mycoflora in intact green bolls. To assure within reason that any histological changes observed in association with fungi were caused by *C. capsici*, any inoculated or control boll that was visibly contaminated with other fungi was discarded. Moist chambers were held at 28 C under continuous fluorescent illumination for 14 days. Additionally, class I and II bolls from glandless Sampson cotton plants were inoculated and treated as described above.

A nonspecific test (3) was employed to detect the presence of tannins in boll pericarp tissues. Homogenized boll pericarp tissue in phosphate buffer was centrifuged and the liquid was decanted. To this crude plant extract (~10.0 ml) several drops of 1% gelatin solution were added. A precipitate indicated that tannins were present in the crude extract.

Tissue preparation for light microscopy. Small pieces of pericarp tissue taken at 4-hr intervals for 36 hr after inoculation from inoculated and control class I bolls were fixed in 1:1 (v/v) 8% glutaraldehyde and 10% formalin in 0.1 M phosphate buffer at pH 7.2. Fixed tissue pieces were washed twice in 0.1 M phosphate buffer, dehydrated in a graded acetone series and infiltrated under vacuum at 6 C for 4–5 days with catalyzed JB-4 plastic monomer (DuPont Company, Newton, CT 06470). The monomer was polymerized at room temperature by the addition of N-N'-diethyl aniline. After curing for several weeks, blocks were sectioned at 1.5–3.0 μm with glass knives on a JB-4A microtome (DuPont Company). Sections were placed on drops of distilled water on clean glass slides, allowed to dry, stained with dilute toluidine blue, and permanently mounted with Polymount resin (Polysciences, Inc., Warrington, PA 18976).

Small pieces of pericarp tissue were taken from inoculated and control class II bolls at 4-hr intervals for the first 24 hr and at 24-hr intervals thereafter until sporulation was observed on the boll surface. The tissue was fixed in 4% glutaraldehyde in 0.1 M phosphate buffer at pH 7.2, rinsed twice in buffer, dehydrated in a graded acetone series and infiltrated with and embedded in Spurr's (18) resin. Sections were cut at 3.0 μm with glass knives on a Sorvall MT-2 ultramicrotome (DuPont Company), floated on distilled water drops at 60 C on a gelatin-coated slide, and allowed to dry. Sections were stained with toluidine blue or Paragon stain (toluidine blue and basic fuchsin in 30% ethanol), and permanently mounted with Polymount resin.

Microscopic observations and photomicrographs were made with either a Zeiss Standard or Leitz Dialux 20 photomicroscope.

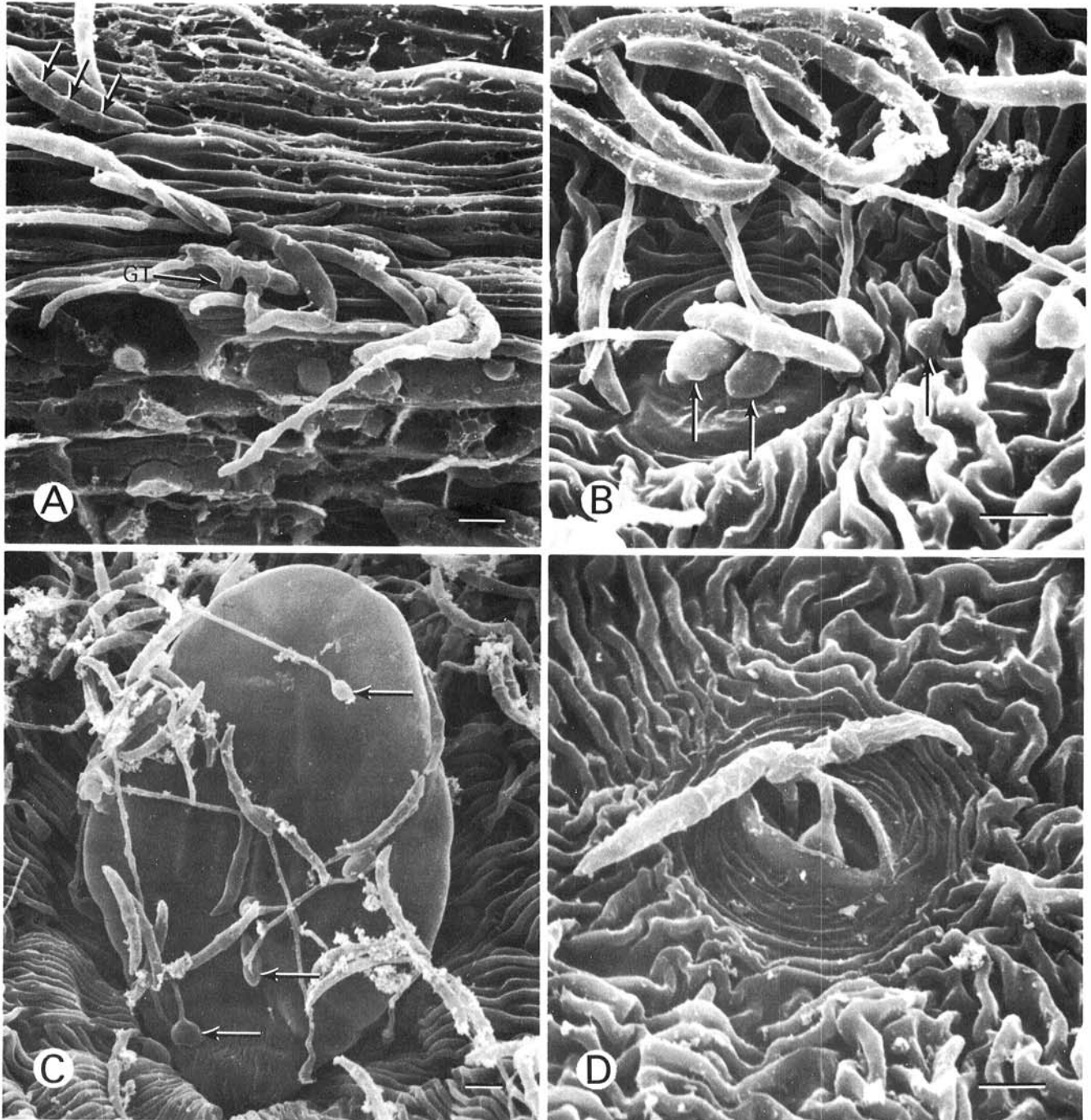**Tissue preparation for scanning electron microscopy.** For

Fig. 1. Germination and penetration of *Colletotrichum capsici* on immature cotton bolls. **A,** Three-septate conidium (arrows) and a conidium that produced three germ tubes, one of which (GT) appears to penetrate the cuticle without producing an appressorium. **B,** Appressoria produced on the cuticle of epidermal and guard cells. **C,** Appressoria (arrows) produced on a multicellular trichome (arrows). **D,** Direct stomatal penetration by germ tube and formation of appressorium in stomatal cavity. Bars = 5 μm.

scanning electron microscopy, inoculated and control pericarp tissues were taken at 4-hr intervals through 36 hr and fixed in the glutaraldehyde/formalin mixture, dehydrated in acetone, and placed in three changes of spectrograde acetone for 5 min each. Tissue was critical-point dried, mounted on aluminum stubs with double-stick tape and sputter-coated with approximately 20-nm (200-angstrom) gold-palladium. Observations were made with an Hitachi S-500 scanning electron microscope operating at 20 or 25 kV.

RESULTS

Inoculations. After 16 hr of incubation, tiny rust-colored lesions less than 0.25 mm in diameter were observed on the surfaces of all 50 inoculated class I Stoneville 213 cotton bolls. These lesions enlarged slightly and became darker by 24 hr, but did not enlarge further during the incubation period. Inoculated class I Sampson cotton bolls exhibited a similar hypersensitive reaction by 24 hr after inoculation.

Blue-black lesions appeared on the surfaces of 45 of 50 inoculated class II Stoneville 213 cotton bolls 3–7 days after inoculation. These lesions enlarged rapidly and eventually encompassed entire bolls. Inoculated class II Sampson bolls exhibited a similar susceptibility. Control bolls remained green throughout the incubation period.

Scanning electron microscopy. Conidia germinated and produced appressoria on the cuticles of class I and II Stoneville 213 bolls within 4 hr after inoculation. Upon germination, conidia became one- to three-septate and produced a germ tube at one or both ends and/or from the middle of a conidium (Fig. 1A). Well-
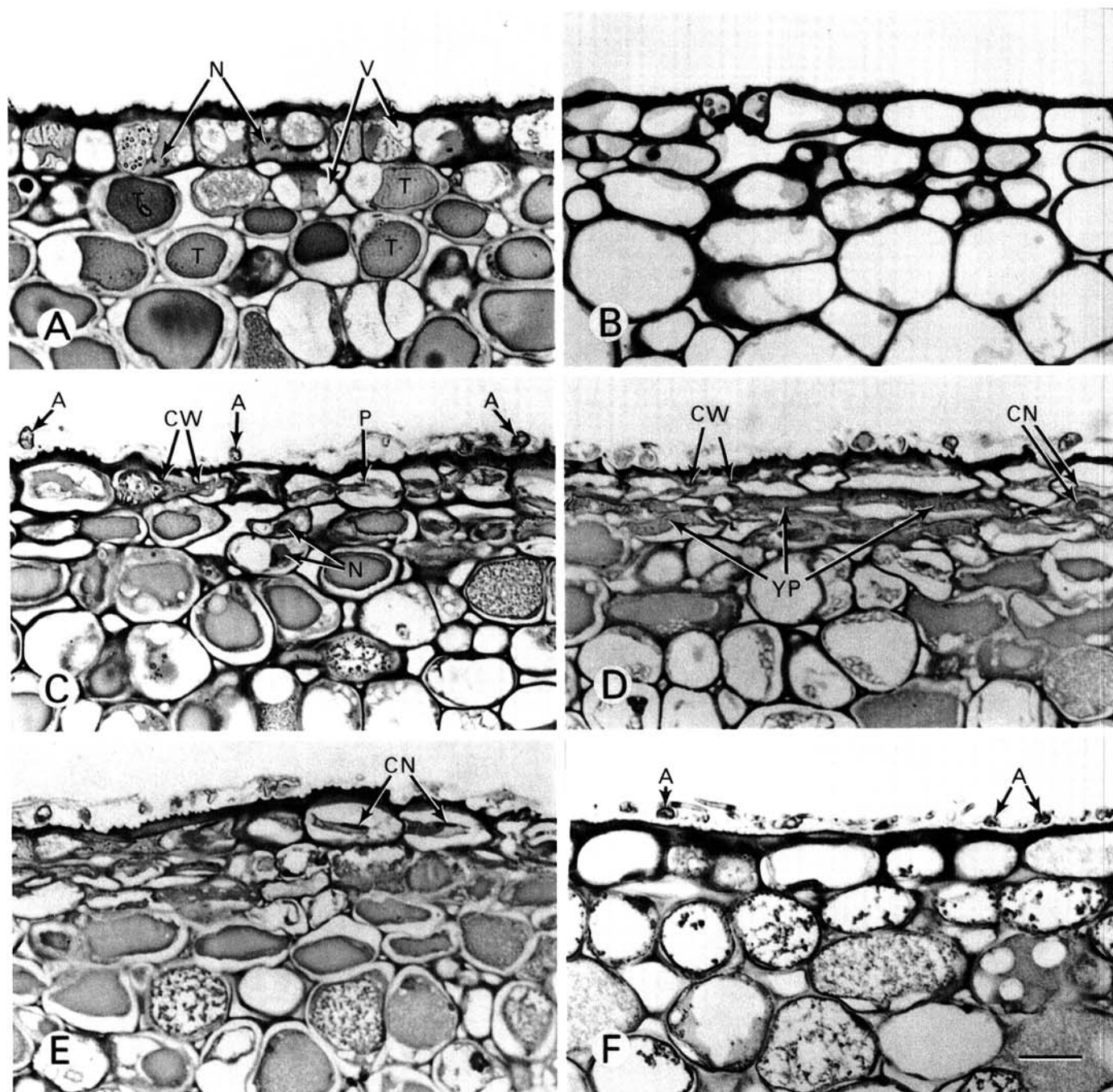

Fig. 2. Hypersensitive response of class I bolls to *Colletotrichum capsici*. **A,** Class I check boll pericarp. **B,** Class II check boll pericarp (Spurr's embedment). **C,** Inoculated class I pericarp 16 hr postinoculation, showing plasmolysis and cell wall collapse in epidermal cells. **D,** Inoculated class I pericarp 24 hr postinoculation, showing collapsed nuclei, collapsed cell walls, and accumulation of a yellow-brown pigment. **E,** Inoculated class I pericarp 24 hr postinoculation. Note flattened nuclei. **F,** Inoculated class II pericarp 24 hr postinoculation. YP = yellow-brown pigment, N = nucleus, V = vacuole, A = appressorium, CW = collapsed wall, P = plasmolysis, CN = collapsed nucleus, and T = tannins. Bars = 20 μm.

developed germ tubes without appressoria were observed and in some instances appeared to directly penetrate the cuticle (Fig. 1A). Appressoria were usually delimited from the germ tube by a septum, but were occasionally produced without an obvious germ tube. Appressoria were formed on the cuticle of stomatal guard cells, epidermal cells, and multicellular trichomes (Fig. 1B and C). Some appressoria were formed in stomatal cavities, and some stomata were penetrated by germ tubes without appressorial formation (Fig. 1D). No conidia were observed on the cuticles of control bolls.

Light microscopy. Epidermal cells and subepidermal parenchyma of class I uninoculated pericarp tissue remained turgid throughout the incubation period, and possessed large nuclei with numerous nucleoli, relatively small vacuoles, and dense cytoplasm (Fig. 2A). Subepidermal parenchyma cells contained large amounts of tanninlike material that stained more darkly than other cytoplasmic components (Fig. 2A). The presence of tannins in pericarp tissue was confirmed (3). Epidermal cells and subepidermal parenchyma of uninoculated class II bolls were larger and more highly vacuolate than corresponding cells of class I bolls (Fig. 2B).

By 16 hr after inoculation, the presence of appressoria on the cuticles of class I bolls coincided with areas of severe plasmolysis in epidermal tissue. Anticlinal epidermal cell walls collapsed, and nuclei were smaller and stained more deeply (Fig. 2C) than in corresponding cells of control bolls. At 24 hr, further nuclear disintegration and collapse of anticlinal cell walls in the epidermis and subepidermal parenchyma were observed. A yellow-brown pigment that did not stain and that appeared to replace the darkly staining tannins was seen in collapsed areas in subepidermal tissue several cell layers deep (Fig. 2D). After 24 hr of incubation, no additional collapse of cell walls was observed, but nuclei in affected cells appeared as flat, darkly stained structures (Fig. 2E). No penetration pegs, intracellular hyphae, or intercellular hyphae were observed in pericarp tissue of class I bolls.

Conidia, germ tubes, and appressoria were observed at 24 hr on the cuticles of inoculated class II bolls without the accompanying plasmolysis, collapse of cell walls, nuclear disintegration, and pigment accumulation observed in class I bolls (Fig. 2F). Appressoria produced penetration pegs approximately 0.75 μm in diameter (Fig. 3A). No histological changes were observed at 2 days, but by 3–4 days after inoculation, subcuticular hyphae 0.75–4.0 μm in diameter were associated with areas of swollen epidermal cell walls that did not stain (Fig. 3B). Hyphal proliferation within the outer anticlinal epidermal cell walls and beneath the cuticles preceded penetration into underlying tissue. By 6 days after inoculation, anticlinal and periclinal epidermal cell walls were swollen, and intracellular and intercellular hyphae penetrated pericarp epidermis into underlying tissues (Fig. 3C and D). Hyphae 0.75–5.2 μm in diameter rapidly invaded pericarp parenchyma, vascular tissue, endocarp, and lint. No hyphae were observed within the lumina of lysigenous gossypol glands even though surrounding tissue showed extensive fungal colonization (Fig. 3E).

By 9 days after inoculation, sporulation was observed on boll surfaces. Pericarp parenchyma ramified by hyphae was swollen, collapsed, and did not stain compared to uninoculated pericarp parenchyma (Fig. 3F). Xylem elements, sclerified fibers, and endocarp cells were penetrated by hyphae, but their lignified cell walls did not appear to be degraded, and retained their affinity for stain even when adjacent to highly degraded parenchyma that did not stain (Figs. 3G and 4A and B). Hyphal penetration of xylem elements occurred primarily through the pits (Fig. 4A).

Endocarp tissue was ramified by hyphae without pronounced loss of structural integrity but was often bound to the tight-locked mass of lint and became separated from the pericarp when the lint was removed from an infected locule (compare Fig. 4B with Fig. 4C).

Lint was collected from directly beneath inoculation sites on class II bolls and preserved in FAA. The lint was removed from FAA and mounted in lactophenol or lactophenol-cotton blue for microscopic observation.

Spiral-shaped fissures in the secondary cell wall of lint cells removed from inoculated bolls were often directly associated with spiraling hyphae of *C. capsici* (Fig. 4D). Prolonged association of the hyphae and lint caused the fissures to reach the lint lumina, causing the lint to unwind (Fig. 4E). Lint cells were bound together by hyphae that successively penetrated adjacent cells (Fig. 4F). Hyphae were observed in the lumina of lint from inoculated bolls. The fibrillar nature of the cell wall subunits was apparent in highly degraded lint (Fig. 5A). Fungal stromatic tissue was produced within lint cell lumina and upon the exterior of lint cells. Conidiogenous cells, conidia, and setae often developed from the stromatic tissue (Fig. 5B).

Intracellular and intercellular hyphae were observed in epidermis, hypodermis, sclerenchyma, and parenchyma tissues of seeds removed from infected bolls (Figs. 5C–D and 6A–B). Hyphae were often swollen where cell walls were encountered, narrowed as they passed through, and enlarged again on the distal side of penetrated cell walls (Fig. 6A). No hyphae were observed in nucellar or embryo tissues (Fig. 6B). Plasmolysis was observed in the nucelli of infected seed.

Conidiomatal development in pericarp tissue was initiated when hyphae aggregated within the swollen remains of periclinal epidermal cell walls. Hyphal aggregations developed into pseudoparenchymatous stromata that, with developing conidiophores and conidia, caused the remains of anticlinal epidermal cell walls and cuticle to bulge outward and rupture (Figs. 6C and D). Stromatic tissue often continued to develop following rupture of cuticle and epidermal walls, causing some conidia to be produced above the epidermal remains. Development of conidiomata was intermediate relative to the pulvinate and hypostromatic developmental types described by Sutton (19). Conidia were also produced beneath the intact cuticle and within degraded pericarp parenchyma cells (Fig. 6D).

Gossypol and/or hemigossypol was detected in lysigenous glands in freehand sections of class I and II Stoneville 213 cotton bolls treated with $SbCl_3$–$HClO_4$ by the method of Mace et al (8). Sections from class I and II inoculated and check Sampson bolls treated similarly had no detectable gossypol in pericarp tissues. The hypersensitive response was observed in both Stoneville 213 and Sampson class I inoculated bolls.

DISCUSSION

Conidial germination and the formation of appressoria of *C. capsici* were comparable on class I bolls, which reacted hypersensitively to the pathogen, and class II bolls, which were susceptible. Penetration of class II bolls occurred directly or through stomata from either appressoria or germ tubes. Penetration of class I bolls was not seen, presumably due to the relatively thick and deeply staining methacrylate sections that may have obscured the thin (~0.75 μm diameter) penetration hyphae. The hypersensitive reaction of class I tissue produced severe plasmolysis, collapse of anticlinal epidermal cell walls, and nuclei that appeared smaller and more darkly stained than normal after 24 hr, whereas changes in susceptible tissue were not observed until 3–4 days after inoculation. The pathogen continued to ramify and eventually sporulated in class II bolls but apparently failed to colonize class I bolls and could only be reisolated from approximately 30% of pericarp tissue pieces bearing hypersensitive lesions.

Preparation of boll tissues for light microscopy according to the methacrylate procedure produced sections that stained well and clearly demonstrated cytoplasmic detail. Because the methacrylate monomer is water soluble, the hydrophobic nature of boll cuticles and epicuticular waxes made them difficult to infiltrate and presented a barrier to infiltration of underlying tissues. The polymerized resin consequently separated from the cuticles during the sectioning procedures, making usable sections difficult to attain. The most obvious histological phenomenon observable at the light microscopic level in class II bolls appeared to be the effect of hyphae on pericarp cell walls. Both hyphae and pericarp cell walls stained well in Spurr's resin. Because usable sections were

easier to obtain from Spurr's than from JB-4 embedded tissue, Spurr's resin was used for studies of susceptible boll tissues.

Epidermal cells and subepidermal parenchyma in class I bolls appeared to have denser cytoplasm and were not fully expanded when compared to corresponding cells in class II bolls and were presumably more metabolically active. Veech (20) demonstrated that the production of gossypol can be induced in certain root cells attacked by nematodes. Bell (1) reported the association of gossypol production and fungal infection and proposed a possible

role of gossypol in resistance to fungal boll-rotting organisms. Due to the presence of gossypol glands near the epidermis, the ability of gossypol and related compounds to diffuse through cotton parenchyma (7), and the fungitoxicity of the gossypols (1,6), the gossypols were initially suspected of participating in the hypersensitive reaction observed in class I bolls. Inoculated class I bolls completely lacking gossypol glands (cultivar Sampson), however, exhibited similar restricted lesions within 24 hr after inoculation. The absence of red pigment in freehand sections of
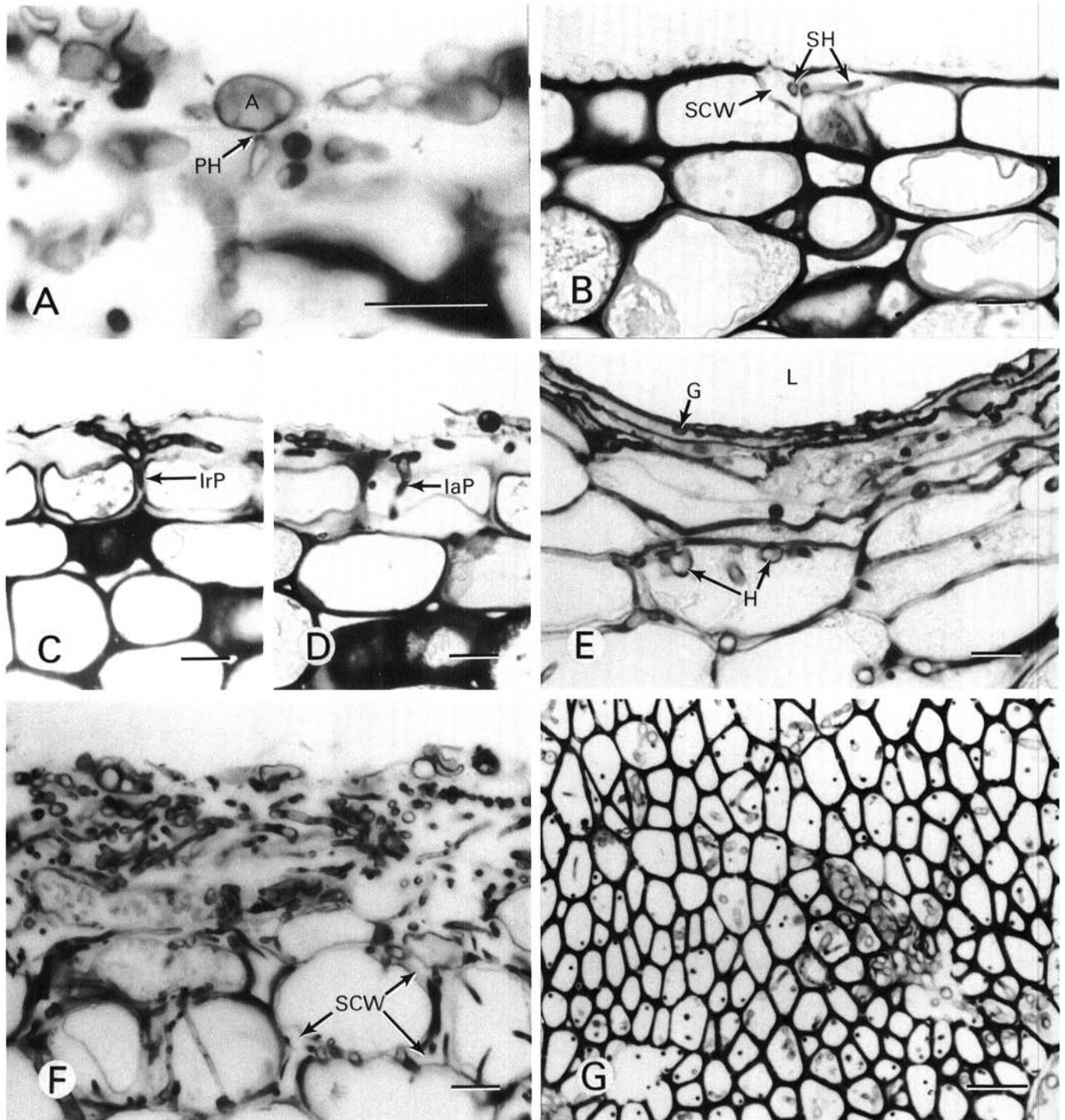

Fig. 3. Hyphae of *Colletotrichum capsici* in susceptible pericarp tissues. **A,** Appressorium and penetration hypha. **B,** Subcuticular hyphae associated with nonstaining, swollen epidermal cell wall. **C,** Intercellular hyphal penetration of epidermis of class II pericarp. **D,** Intracellular hyphal penetration of class II pericarp. **E,** Lumen of lysigenous gossypol gland is free of hyphae, and cells lining lumen contain gossypol droplets. Surrounding parenchyma is ramified by hyphae. **F,** Class II pericarp 9 days postinoculation showing swollen, nonstaining parenchyma cell walls and extensive fungal colonization. **G,** Cross-section of pericarp fibers, showing retention of ability to adsorb stain even when closely associated with hyphae. A = appressorium, PH = penetration hypha, SCW = swollen cell wall, SH = subcuticular hyphae, G = gossypol droplet, L = lumen, H = hyphae, IrP = intercellular hypha, IaP = intracellular hyphae. **A–F,** Bars = 10 μm. **G,** Bar = 20 μm.

rust-colored lesions when treated with SbCl₃–HClO₄ reagent indicated that induced or diffused constitutive gossypols were not present in concentrations detectable by this technique. Therefore, gossypols are believed not to participate in the hypersensitive response. Bell (1) reported that bolls 35–45 days old were more susceptible to infection by *Verticillium albo-atrum* than bolls 15–25 days old, and produced only 10–30% as much gossypol. If the hypersensitive reaction can be a means of resistance of young bolls to boll rot in the field, the results of this study indicate that gossypol is probably not responsible for the resistance.

The conversion of the blue-staining, tanninlike pigments in epidermal and subepidermal cells into a yellow-brown, nonstaining pigment was closely associated with the collapse and death of those cells and the apparent prevention of fungal colonization of boll

tissues. The precise nature of the tanninlike substance is not known.

Inoculation of class II bolls with conidia of *C. capsici* produced changes in boll tissues distinct from the hypersensitive reaction observed in class I bolls. After penetration of the cuticle, subcuticular hyphae proliferated in the outer periclinal epidermal cell walls for several days before penetrating between and through epidermal cells into underlying boll tissues. The most striking effect on pericarp tissues was the apparent dissolution of the cellulosic portion of parenchyma cell walls, and the concomitant swelling and loss of stainability of those walls. Prolonged incubation of inoculated bolls resulted in the degradation of nearly all nonlignified boll tissues. The degradation of pericarp parenchyma and the persistence of xylem elements and other sclerified tissues give bolls rotted by *C. capsici* their diagnostic macroscopic
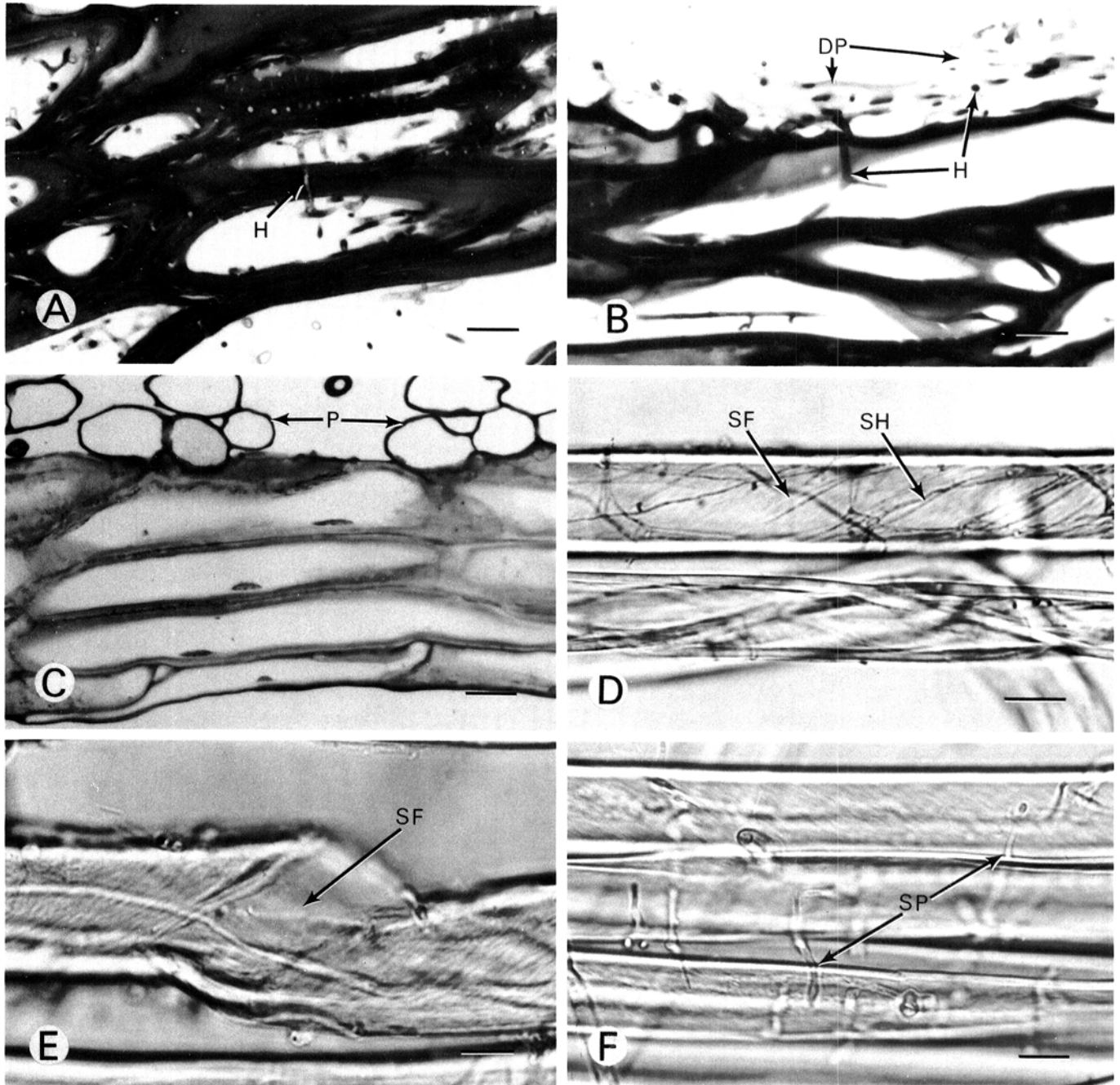

Fig. 4. Hyphae of *Colletotrichum capsici* in endocarp and lint. **A,** Oblique section of pericarp xylem ramified by hyphae. **B,** Endocarp from inoculated boll, showing degraded parenchyma and hyphal colonization of endocarp cells. **C,** Endocarp from check boll. **D,** Spiral hyphae of *C. capsici* associated with small spiral fissures in the lint. **E,** Lint that became unwound when spiral fissures reached the lint lumen. **F,** Lint bound together by successive hyphal penetration of adjacent lint cells. H = hyphae, DP = degraded parenchyma, P = parenchyma, SF = spiral fissure, SH = spiral hyphae, SP = successive penetration. **A–C and E,** bars = 10 μm. **D and F,** bars = 20 μm.

appearance. The rapid and almost total dissolution of parenchyma cell walls and the fissures in infected lint cells is presumptive evidence that *C. capsici* produces cellulases during the process of boll colonization, and that cellulose breakdown products are probably a primary carbon source for the fungus. Panigrahi and Narain (10) reported that glucose rendered previously resistant green chili fruits susceptible to infection by *C. capsici*. The ability of *C. capsici* to produce cellulases and to utilize cellulose breakdown products likely contributes to the pathogenicity observed on class II cotton bolls, and may be directly responsible for the macroscopic appearance of rotted bolls.

The association of hyphae of *C. capsici* with lint cells from inoculated bolls produced effects that closely parallel those reported for cotton lint digested with a cell-free extracellular cellulolytic enzyme solution prepared from *Myrothecium verrucaria* Ditmar: Fr (2,9). Features of lint degradation described by Simpson and Marsh (15) were observed in lint from bolls degraded by *C. capsici*.

Pericarp parenchyma cells adjacent to the endocarp are thin-walled and are often isolated by large intercellular spaces. The separation of endocarp from pericarp in rotted bolls occurred because the sites of attachment to the pericarp were destroyed when the parenchyma cell walls were degraded.
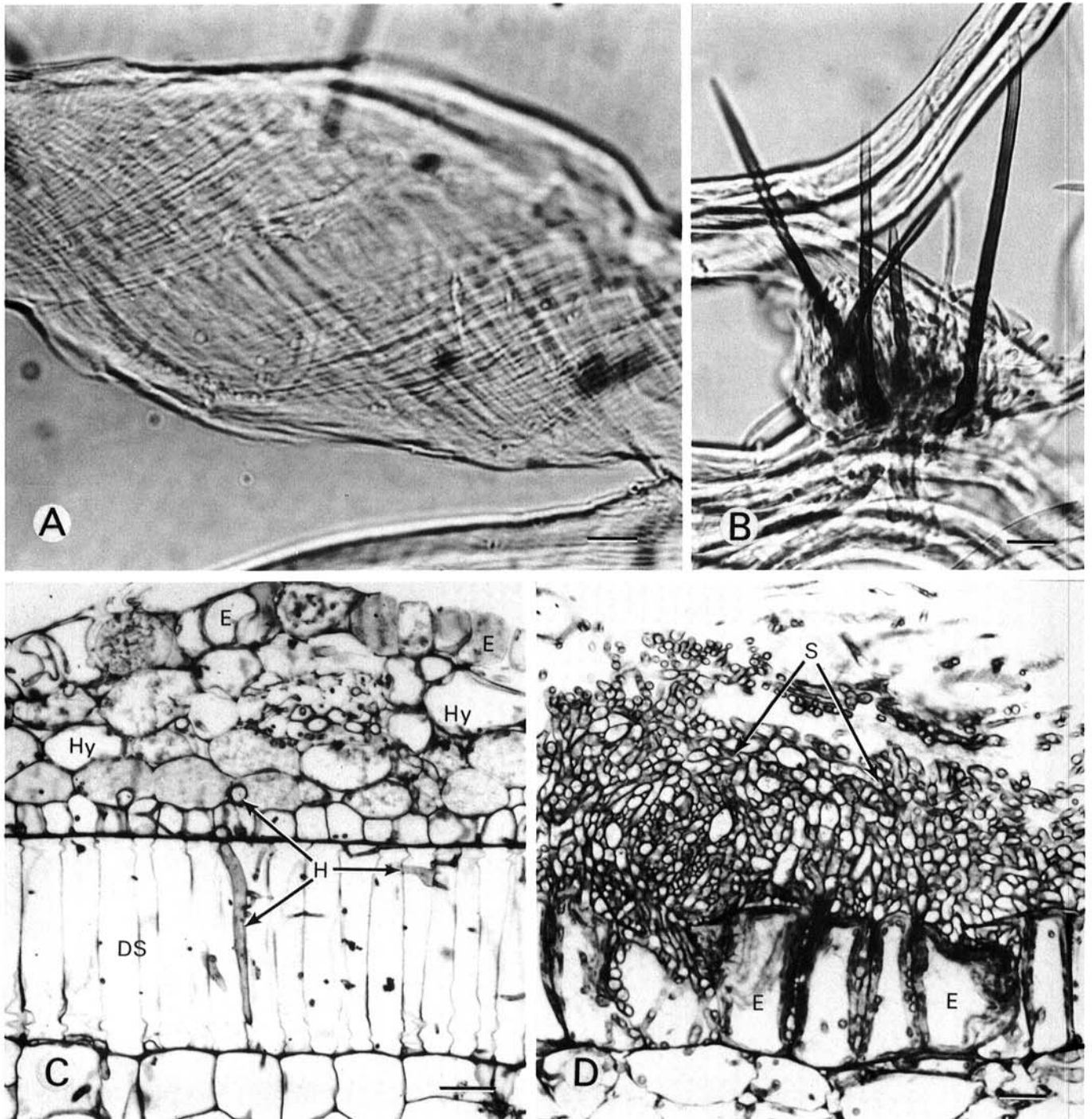

Fig. 5. Hyphae, stromata, and conidioma of *Colletotrichum capsici* associated with cotton lint and seed. **A,** Highly degraded lint demonstrates the fibrillar nature of lint wall subunits. **B,** Conidioma of *C. capsici* produced on infected lint. **C,** Hyphae of *C. capsici* in epidermis, hypodermis, developing sclerenchyma and parenchyma of cotton seed. **D,** Fungal stromatic structures produced on and in epidermis of seed. E = epidermis, Hy = hypodermis, H = hyphae, DS = developing sclerenchyma, S = stromatic structures. **A and D,** Bars = 10 μm. **B and C,** Bars = 20 μm.
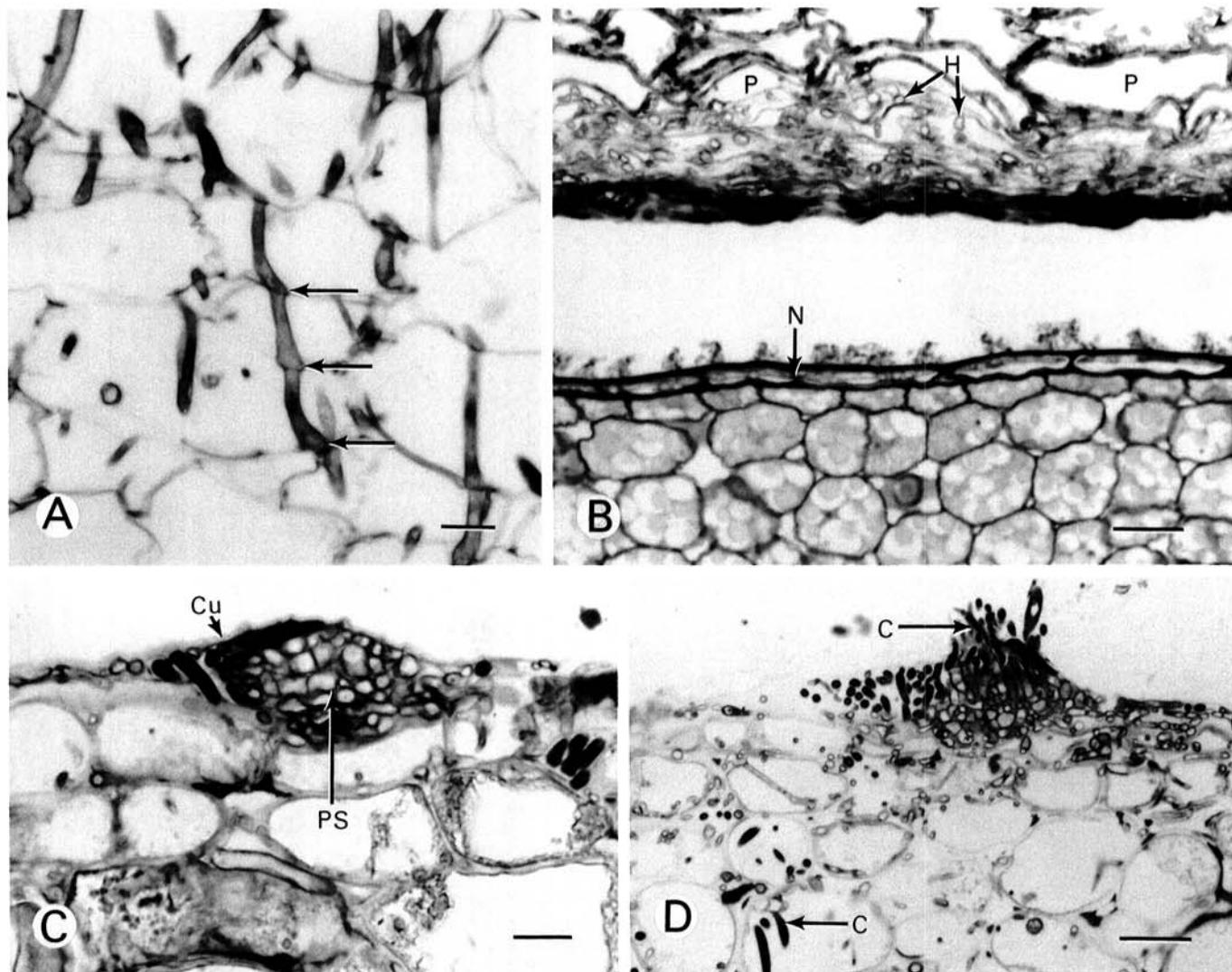
Fig. 6. Hyphae of *Colletotrichum capsici* in seed and conidiomatal development. **A,** Hyphae of *C. capsici* in seed parenchyma. Note alternate swelling and narrowing of hyphae as host cell walls are penetrated (arrows). **B,** Degraded seed parenchyma that became separated from the nucellus and embryo. Note plasmolyzed cells in the nucellus. **C,** Pseudoparenchymatous stroma in periclinal epidermal cell wall caused the cuticle to bulge outward. **D,** Conidioma that ruptured the cuticle, causing conidia to be produced above the epidermis. Note sporulation within pericarp parenchyma. P = parenchyma, H = hyphae, N = nucellus, Cu = cuticle, PS = pseudoparenchymatous stroma, C = conidia. **A and C,** Bars = 10 µm. **B and D,** Bars = 20 µm.

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