FINE MORPHOLOGY OF FUNGAL STRUCTURES INVOLVED IN HOST WALL ALTERATION

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Alteration of host cell walls (HCW) by fungi is probably a key event in pathogenesis, by which pathogens penetrate plant cell walls and secrete specific wall-degrading enzymes. Numerous biochemical, histological, and cytochemical studies of host-fungi interface have revealed that pathogenic fungi produce a wide range of penetrating structures before plant infection and HCW degradation.

This review focuses on fungal structures associated with HCW penetration and degradation in different plant-fungus associations. Particular attention will be given to the morphology and roles played by extracellular fungal sheaths in cell wall alteration and penetration. Unusual structures that may contribute to HCW degradation will also be discussed.

PARTICULAR CELLULAR STRUCTURES

Pathogenic fungi can produce a wide array of cellular structures specialized in cell wall penetration, such as appressoria, haustoria, or microhyphae. Alteration of plant surfaces results from mechanical pressure and enzymatic activites (332). Involvement of enzymes, i.e. cutinase and pectinase (274) and cellulase (281) in the penetration process has been suggested and partly demonstrated for appressorial fungi, although immunocytochemical evidence for the presence of these enzymes is lacking.

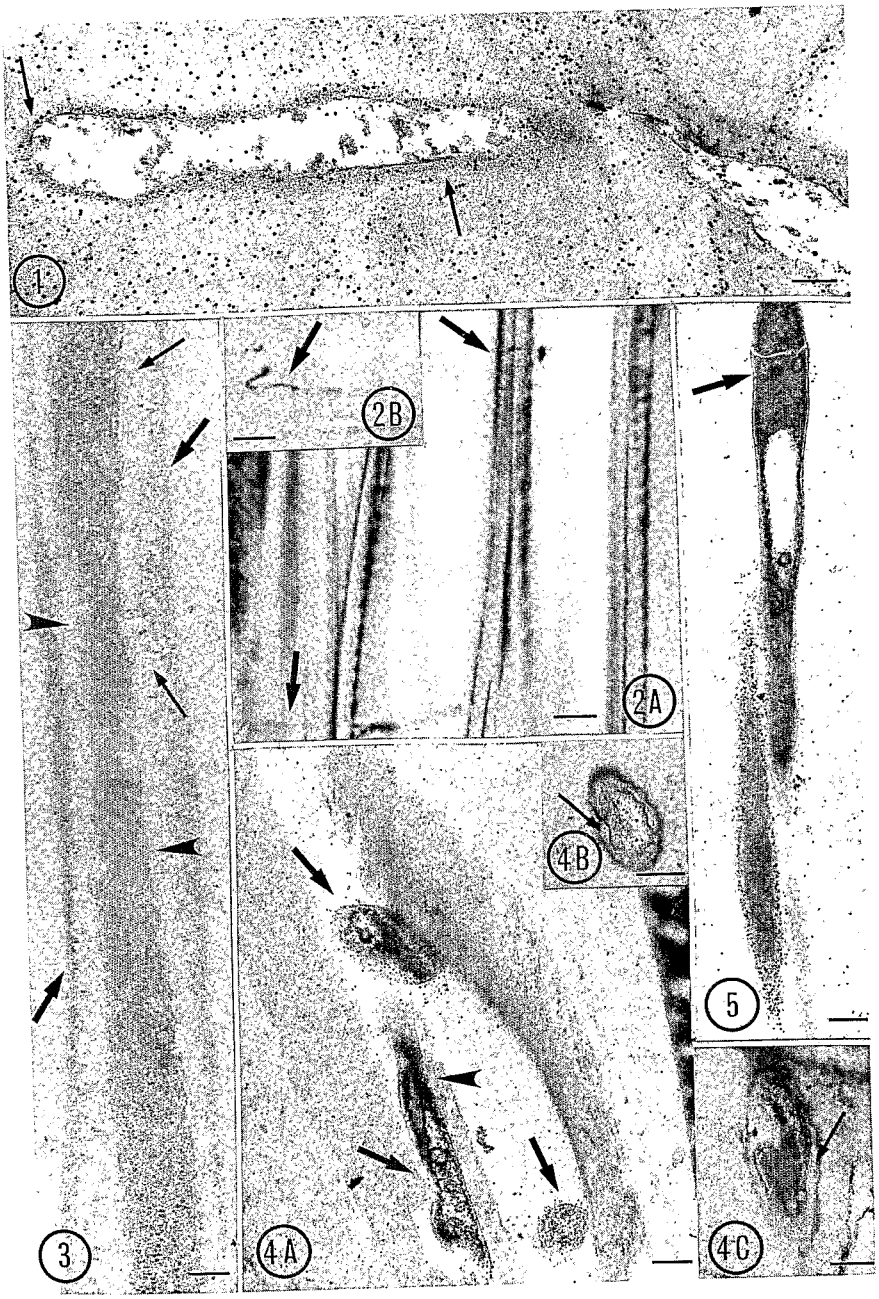
Appressoria

For several fungi such as rusts (228, 229), *Colletotrichum* species (332), endophytes (Stone et al., this volume) and vesicular-arbuscular mycorrhizae (Bonfante, this volume), the interaction with the host surface results in the formation of appressoria.

Appressoria were first described in 1883 as spore-like organs formed on germ tubes of *Colletotrichum* species (154). They are usually described as forms that provide anchorage for infection pegs or germ tubes, and are able to overcome the host's physical barriers such as the cuticle layer (154). Their formation starts when growth of germ tubes has stopped. The fungal cell wall, cytoskeletal microtubules, and apical vesicles are probably involved in differentiation of appressoria (229). Appressoria are often associated with stomatal penetration, but they have also been described in intact plant surface penetration (321, 327).

Initiation and maturation of appressoria are controlled by several factors, including temperature, plant exudates, and the fungal genotype (154). During appressorium formation, chitin of the fungal wall was not detected, likely resulting from glucan overlay (322). Penetration of host surfaces by appressoria requires signaling that may result in tight adherence to the substrate (229, 340). The extracellular matrix of these structures contains specific proteins involved in recognition (156, 322) and initiation of plant cell wall degradation (228, 317). Infection pegs, or appressorial cones, arise from the appressorium and subsequently penetrate the HCW (327).

Figs. 1–5. Microhypha development in HCW. Double fixation with glutaraldehyde and OsO_4; Fig. 1: glutaraldehyde alone. **1**. *Phellinus noxius* in sterilized wood sections. Labeling for cellulose with gold-labeled exoglucanase. The fungal cell plasmalemma is in close contact with HCW, and from or through it radiate arrays of fibrillar strands extend into HCW (small arrows). Bar: 0.2 μm. **2A**, **2B**. LM observation of microhyphae (arrows) grown through or between cell walls, at times continuous with similar structures in cell lumina. Bars: 5 μm. **3**. *Phialophora mutabilis* in copper-chromium-arsenic treated birch. Fungal wall lacking at times (arrowhead) or unperceptible. The cavity margin is marked by a halo of opaque matter (arrows); fibrillar strands or tubular-like structures extend from the fungal cell wall or contents through the cavity to the halo (small arrows). Bar: 0.2 μm (Courtesy of Drs. Hale and Eaton). **4-5**. *Ophiostoma ulmi* in sterilized elm wood sections. HCW degradation around microhypha (arrows) in gelatinous fiber wall layer (Fig. **4A**) and in secondary walls of vessel elements or of parenchyma cells (Figs. **4B**, **4C**). HCW portion between the opaque halos and the fungal cell (small arrows) has apparently not yet been degraded in 4C, whereas its alteration is almost complete in 4B. In both, apparent absence of wall around the fungal cells. Septate microhypha in a fiber cell lumen. **4A**, **5**. Labeling with polyclonal antibodies to fimbriae, predominating over the area corresponding to the fungus plasmalemma (or its invagination) or inner portion of the wall; rows of gold particles in close contact with HCW indicate lack of a wall or presence of a very thin one (Fig. **4A**, arrowhead). Scale bars: 4A, 0.5 μm. 4B, 0.3 μm. 4C, 0.4 μm. In Fig. 5 note much less pronounced labeling in the septate microhypha (arrow). Bar: 0.5 μm.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Haustoria

After contact with the host is established, the pathogen develops penetration pegs, vesicles and inter- or intracellular mycelium, as well as haustoria, which start colonisation of host tissues. Haustoria are produced by fungi such as rusts, powdery mildews, downy mildews, and the Exobasidiales. Previous ultrastructural work (149) distinguished four types of haustoria based mainly on the morphological association between the haustorium and the host cell. For instance, in *Phytophthora* species they range from simple, button-shaped to digit-like structures. Haustoria of *Exobasidium* spp. are short and lobed while those of *Cercosporidium* are highly branched (327).

Haustoria arise either directly from penetration pegs or from intercellular hyphae initiated by substomatal vesicles (210, 296). Elongation of the haustorium neck results in the emergence of an haustorium surrounded by extrahaustorial electron-dense matrix and membrane (7, 89). HCW penetration is associated with the formation of an electron-dense penetration matrix which is thought to become the extrahaustorial matrix. High fungal metabolic activities that have been characterized during haustorium formation are probably related to the digestion of HCW at penetration sites.

During penetration, the host plasma membrane is invaginated (149). The extrahaustorial membrane is continuous with the host plasmalemma and strongly adheres to the haustorium. Although the extrahaustorial membrane is presumed to serve as a boundary between the host cytoplasm and the fungus, it may be involved in active exchanges (322), mainly to provide nutrients to the pathogen.

Haustorial wall components are probably involved in recognition mechanisms between avirulent races of stem rust and resistant wheat

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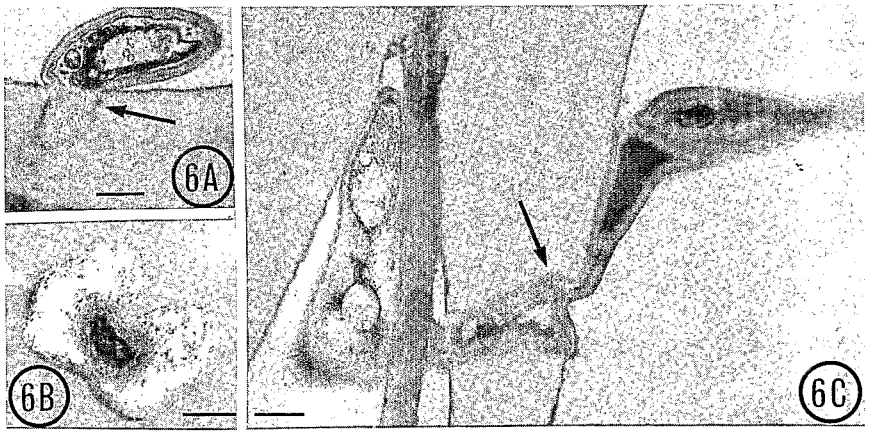
Fig. 6. *Ophiostoma ulmi* in xylem tissues of naturally diseased trees (1978) sampled the following spring, when disease recurrence was noted. **6A, 6C.** Opaque halos in HCW in apparent continuity with outside wall layer of the fungal cell (small arrow). **6B.** Large cavity formed in unmodified finer wall layer with only a portion of the halo remaining (lower part). Scale bars: 6A, 0.4 µm; 6B, 0.2 µm; 6C, 0.3 µm.

Figs. 7-8. Microhyphae (arrows) in walls of proto- and metaxylem vessel elements in a susceptible carnation infected by *Fusarium oxysporum* f. sp. *dianthi*. Labeling with a colloidal gold-complexed WGA lectin for chitin revealed a very thin, but apparently discontinuous wall around these hyphae (Fig. **7**, small arrows). Note strong labeling of the secondary thickenings (ST). The irregular microhyphae in Fig. **8** (small arrow) are about 0.1 µm in diameter. Scale bars: 0.5 µm.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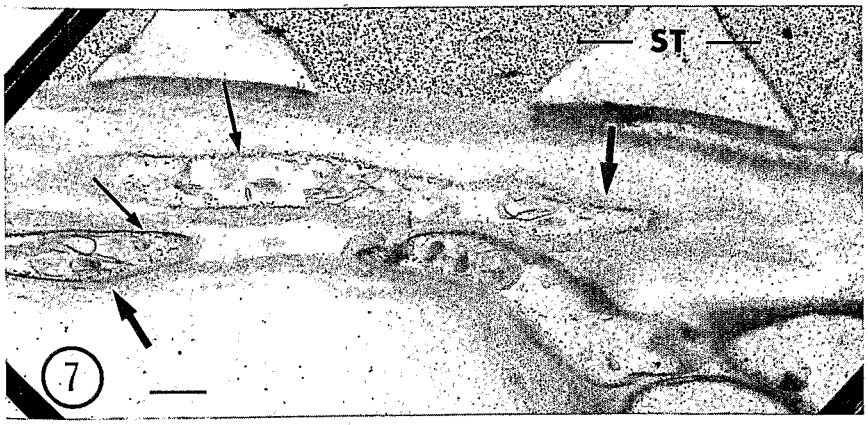
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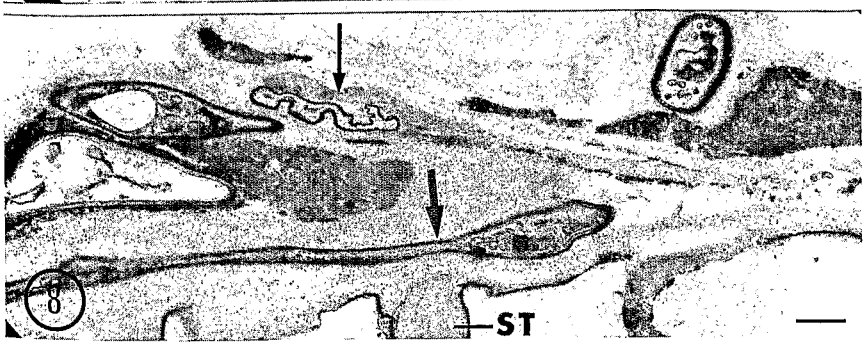
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ST

(113). Recent work indicates that polysaccharides and glycoproteins are present in the haustorium wall and extrahaustorial matrix. A linked-glucose or mannose was found within the *Puccinia graminis* extracellular sheath (ES) (113) while β-1,3 glucans and β-1,6 linkages were identified in ES of *P. sorghi* (106). Haustorium-like structures found in endophytes (Stone et al., this volume) and root-rot fungi (347) do not display the same specialized organization found in true haustoria.

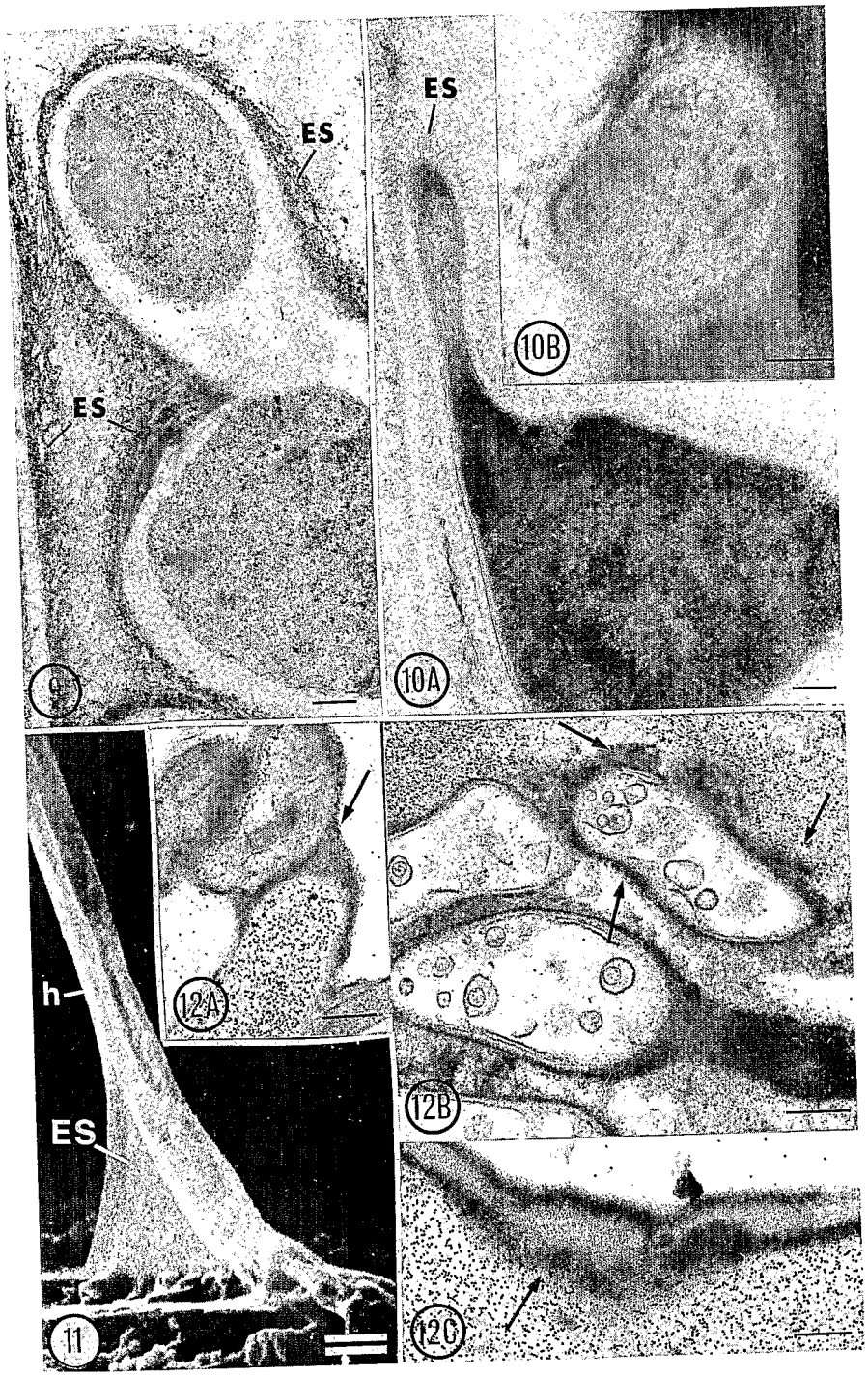
Microhyphae

Fungi may differentiate microhyphae (MH), also called fine hyphae, that can extend for appreciable distances into host walls and cause their breakdown and/or rupture. Microhyphae were first reported in soft rot and blue stain fungi (292), and *Ophiostoma ulmi* (359), and then in Ascomycotina (including Deuteromycotina) and Basidiomycotina (123, 290, 336, 369, 370, 528). *Phellinus noxius*, a decay fungus of tropical trees, has also been shown to produce microhyphae in its attack of wood cell walls (Fig. 1).

The association of MH with cavity formation by soft rot fungi in the S2 cell wall layers can be seen in the light microscope (96); the presence of MH between or within cell walls is generally not evident in LM (Figs. 2A, 2B). Transmission electron microscope (TEM) studies have confirmed the formation of MH and give a better insight into their structural organization and mode of action.

Leightley and Eaton (290) and Hale and Eaton (201, 202, 203),

Figs. 9-12. Extracellular sheaths (ES) in four fungi. TEM observations except Fig. 11. **9**. Thick ES around or between *Gremmeniella abietina* cells, strongly labeled for RNA with gold-labeled RNAse. Scale bar: 0.2 μm. Courtesy of Dr. Nicole Benhamou. **10A**. Loose ES around cells of *Ophiostoma ulmi* in sterilized American elm wood fixed with high pressure freezing. **10B**. Apparent fibrils or strands radiating from or through fungal walls into HCW (small arrows). Scale bars: 0.2 μm. **11**. SEM observations of ES coating hyphae (h) of *Phellinus noxius* developing in sterilized birch sections. Note close adherence of ES to host wall surface. Scale bar: 20 μm. **12A–12C**. *Fusarium oxysporum* f. sp. *dianthi* in infected susceptible carnation, labeled for chitin (**12A**) or cellulose (**12B, 12C**). **12A**. ES-like layer around fungal cells, containing tubular-like structures (small arrow) that extend within the host wall of the vessel element. **12B**. Masses or bands of opaque matter, similar to and continuous with the outside fungal wall layer (small arrows), that extend within HCW (h: hyphae). **12C**. A band of similar matter along a vessel wall, comprising stacks of fibrillar strands, that extend into HCW (small arrow). Scale bars: A, B, 0.4 μm; C, 0.3 μm.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using time-lapse cinematography and material fixed sequentially after inoculation for TEM observations, described the most salient features of MH. Approaching 0.2 µm in diameter, developing MH were devoid, at least in part, of a detectable wall (Fig. 3), and contained a few ribosomes, odd strands of endoplasmic reticulum, occasional microtubules, and membranous networks (157). Host wall penetration by MH is accompanied by the formation of a halo of electron opaque material that chisels through the wall some distance from the microhypha. This halo constitutes the ensuing margin of the cavity (Fig. 3). Fibrillar strands, membranous, and tubular-like structures often extend from the fungal cell into the host wall or in the formed cavity. Layers of extracellular membranes have also been observed surrounding cells of basidiomycetes associated with degradation of wood cell walls (169).

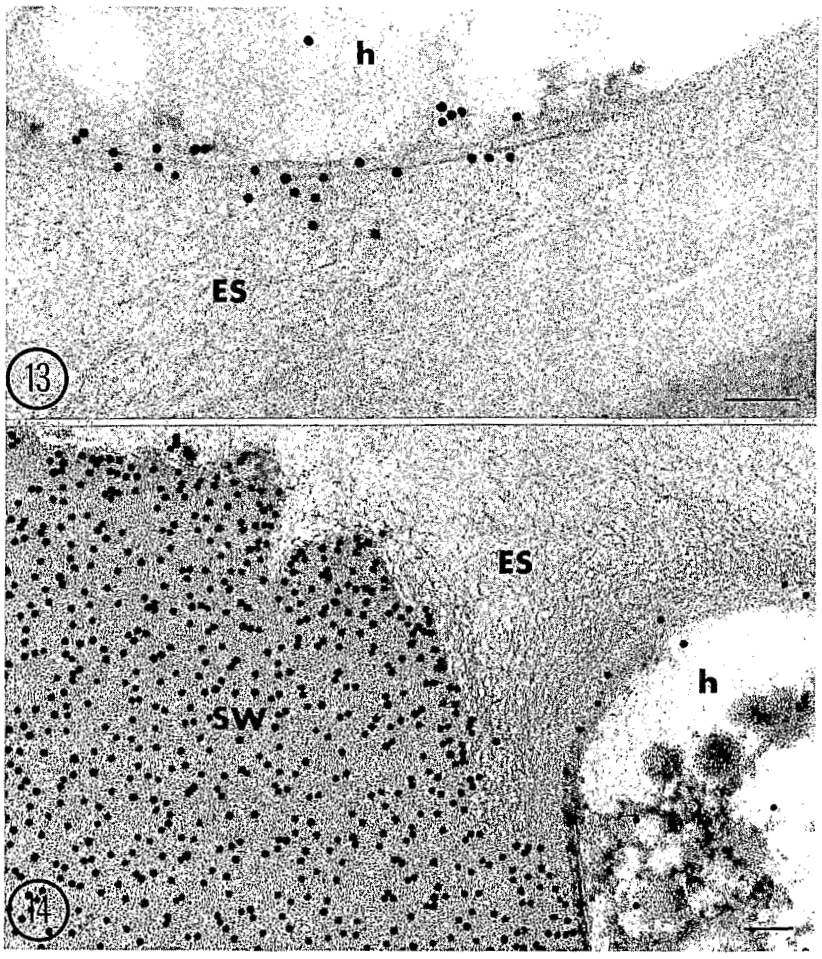
TEM observations of *O. ulmi* development in living elm branches or in sterilized wood sections and of *P. noxius* in wood sections (Figs. 4, 5, 6) have shown that the MH of these fungi have essentially the same characteristics as those of soft rot fungi (201). The use of colloidal gold-labeled lectins, enzymes, and antibodies has led to a better knowledge of the fine structure of MH and of their association with host wall alterations. For example, probes for host wall and fungal constituents, including the plasmalemma and wall layers (47, 360), confirmed the absence of a delimiting fungal wall layer (Figs. 4, 5).

Formation of MH is mainly known in woody plant hosts, and predominantly in secondary cell wall layers of secondary xylem. Recently, however, Ouellette and Baayen (unpublished) have studied the formation of MH by *Fusarium oxysporum* f. sp. *dianthi* developing in a susceptible carnation cultivar. This wilt-causing organism breeches and spreads through and within cell walls of vessel elements (primary and secondary xylem) and adjacent parenchyma cells, by means of long MH (Figs. 7, 8) that are also very small in diameter and often devoid of a detectable wall. A very thin wall, however, can be present (Fig. 7). Profuse wall colonization (Fig. 8) appears to be directly related to symptom expression resulting from extensive tissue disruption; living cells are not appreciably affected until their walls rupture.

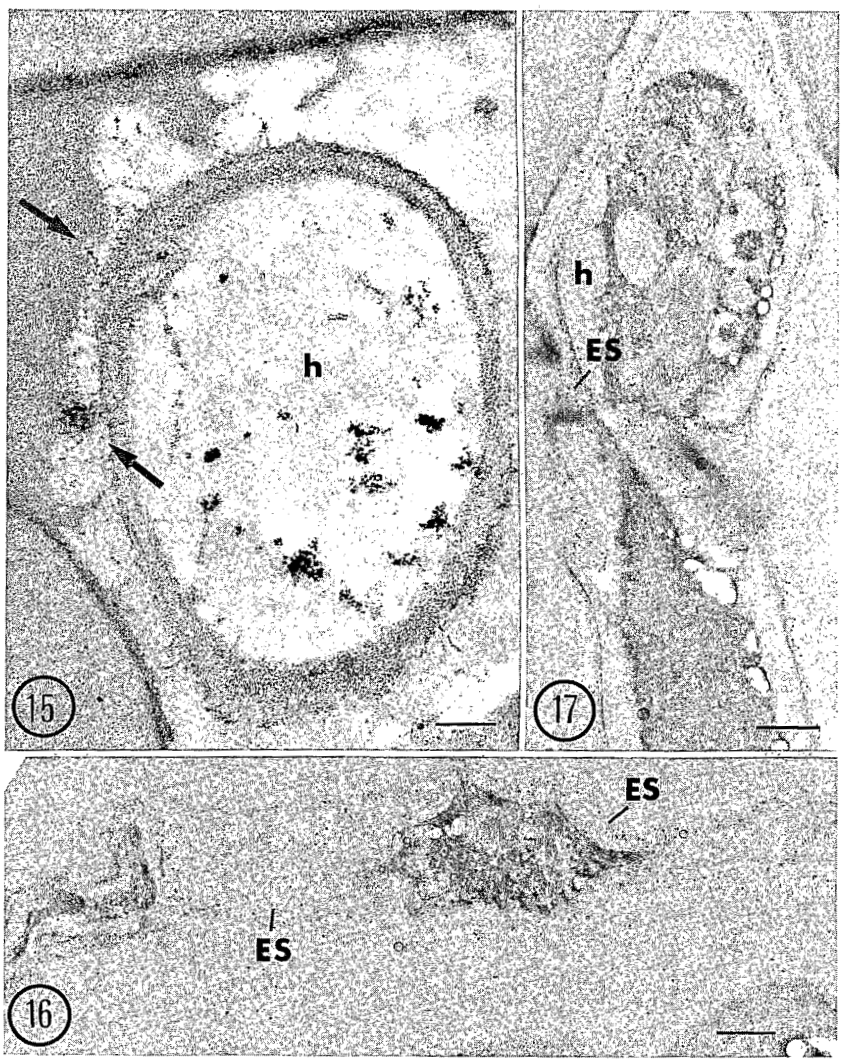
EXTRACELLULAR SHEATHS

Extracellular sheaths (ES) associated with fungal cells were detected as early as 1963 (291). Recently, numerous investigations have been devoted to ES produced by various fungi, including

pathogenic (113, 346), saprophytic (421), endophytic (506), and mycorrhyzal fungi (183). Electron microscopy and recent cytochemical techniques have provided direct information on the roles ES may play in host-fungi interactions.



Figs. 13–14. TEM observations of *Rigidoporus lignosus* inoculated onto sterilized birch (*Betula papyrifera*) sections. **13**. Immunolocalization of laccase L1 over hyphal (h) cell wall and adjacent ES fibrils. Bar: 0.1 µm. **14**. Close association of ES fibrils with host secondary wall (SW) degradation. SW labeled for cellulose. Bar: 0.1 µm.



Figs. 15–17. 15. *Phanerochaete chrysosporium* in birch wood; P.A.T.Ag staining. ES of the hypha (h) is in close association with degraded parts of HCW (arrows). Scale bar: 0.2 μm. **16.** *Phlebia radiata* in birch wood. Immunocytochemical labeling with anti-lignin peroxidase (LiP). The hyphal cells shown are surrounded (and bridged) by ES, which shows a strong positive reaction for LiP. Fixed with 2% glutar-and paraformaldehyde. Scale bar: 0.3 μm. **17.** *Phanerochaete chrysosporium* degrading *Populus* wood. Immunolabeling with anti-xylanase. A positive reaction for xylanases is evident over the wall of the hypha (h) and portions of its ES located in close contact with the degraded HCW. Scale bar: 0.4 μm.

Occurrence of ES in fungi

A wide range of fungi produce a conspicuous extracellular layer of material. ES occur within many groups of fungi, from protoctista, i.e. *Peronospora* and *Sclerospora*, to higher groups, including obligate parasites (*Cronartium* and *Puccinia*), wood-rotting fungi (*Phanerochaete*, *Rigidoporus*), saprobes, and mycorrhyzal fungi. Generally, ES formation seem to occur during a certain period of the fungal life cycle or in response to particular physiological conditions, independently of the type of disease they cause.

Ultrastructure

Extracellular sheaths coat several fungal structures such as appressoria, haustoria, conidia, and penetrating hyphae (117, 162, 327), thus displaying variations in structure and morphology. In most cases, ES appear as a fibrillar network, with dense or loosened fibrils arranged around the hyphal cell, either located at tips or along the length of hyphae (Figs. 9-12C). In most cases, ES adhere to the fungal structure and can establish a close junction between the mycelium and HCW (363, 421) (Figs. 9, 13-14).

Chemical Constitution

Chemical and Biochemical data

Most information concerning the chemical nature of ES has been gained from studies on lignolytic Basidiomycetes. These fungi are able to produce large ES when grown in starvation conditions (471). Formation of ES is dependent on growth conditions that influence the secondary metabolism of the fungus (51, 421).

Fungal ES have been shown to contain a β-1,3-linked glucan main chain, more or less substituted with single glucose units or short glucosyl side chains attached by β-1,6 linkages (51, 87, 144, 421). Slight structural differences between the cell wall and the ES glucans have been demonstrated by β-1,3 glucanase degradation. The enzymatic digestion produced different ratios of glucose and gentiobiose according to the substitution degree of the β-1,3-linked backbone with β-1,6-glucosyl residues. The adherent glucan was less substituted by 1,6 glucose units than the free glucan in the culture medium (388). Similar analyses have indicated that the ES produced by *P. chrysosporium* and liberated into the medium (87) has a highly branched structure.

Surface proteinaceous fibrils, called "fimbriae" because of their similarity with bacterial fimbriae, have also been described in several fungi (130, 393). These fimbriae consist of glycoproteins characteristic of yeast-like fungi (179). Other non-enzymatic proteins were found extracellularly (156), some of them containing a proline-rich fraction (341). Enzymatic proteins were also associated with ES of *Sphacelia sorghi* (136). Binding of proteins to the glucan was non-covalent. Recently, immunocytochemical techniques have confirmed these findings and allowed identification of several ES-associated enzymes.

Cytochemical Data

(Poly)saccharides, enzymes, and non enzymatic proteins have all been detected in ES. Using the P.A.T.Ag. stain, β-1,6 glycosyl residues were observed in several Basidiomycetes (211, 346, 421) (Fig. 15). Immunogold (421) and fluorescence labeling (88) have been used to detect β-1,3-glucans within ES of Ascomycetes and Basidiomycetes. This compound was also detected in ES of *Ophiostoma ulmi* (Chamberland, this volume). Chitin was never localized in ES of rust fungi (106, 113) or of rotting fungi (343), although it is abundant in their cell walls.

Numerous oligosaccharides have been identified by means of fluorescent- or gold-lectin probes. ConA-binding sites, indicating

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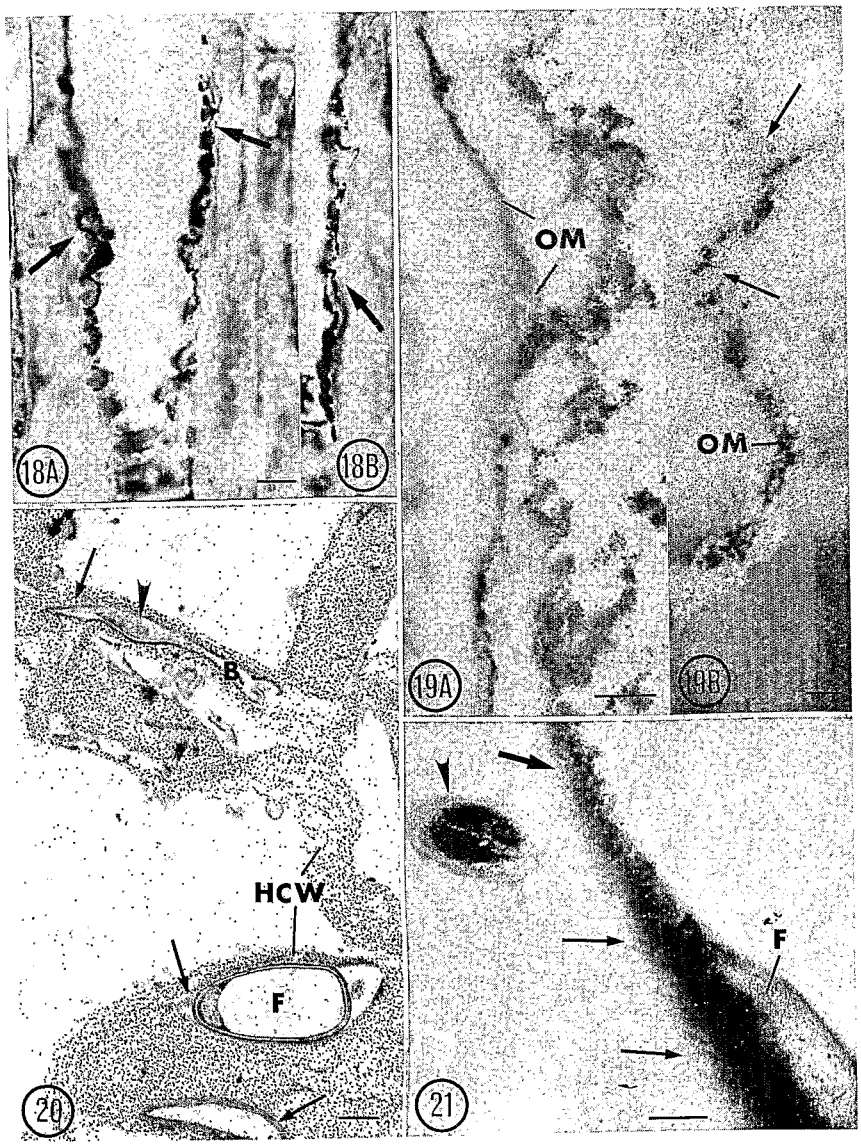
Figs. 18–21. HCW pervasion and degradation associated with bands and masses of electron opaque material (OM)(arrows). **18A, 18B**. Metaxylem vessel elements invaded by *Ophiostoma ulmi*, at the tip of developing streaks. Scale bar: 7 μm. **19A**. Corresponding view in TEM of OM chiseling through and eroding the wall of vessel element. Scale bar: 0.4 μm. **19B**. High magnification of OM associated with HCW (middle lamella and secondary layer) alteration. Note integrity of HCW where OM is discontinuous (small arrows) and composition of the latter, opaque bodies associated with a homogenous fibrillar matrix. Scale bar: 0.1 μm. **20**. Margins of several cavities in proto- and metaxylem cell walls of susceptible carnation infected by *Fusarium oxysporum* f. sp. *dianthi*, demarcated by bands of OM (small arrows). A typical fungal cell (F), with two distinct wall layers, abutting on OM in portions close to HCW. Another cavity (B) contains what appears to be an apparent fungal cell portion (arrow head). HCW labeled for cellulose. Scale bar: 0.4 μm. **21**. Strands of opaque material (small arrows) extending into HCW (vessel element) from a layer of similar material coating the vessel wall. On the opposite side of the vessel lumen a fungal cell present in continuity with a small or collapsed hypha that was similar in appearance to the structure shown at F. A microhypha is present (arrow head) in the vessel wall. Scale bar: 0.3 μm.


18A
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HCW
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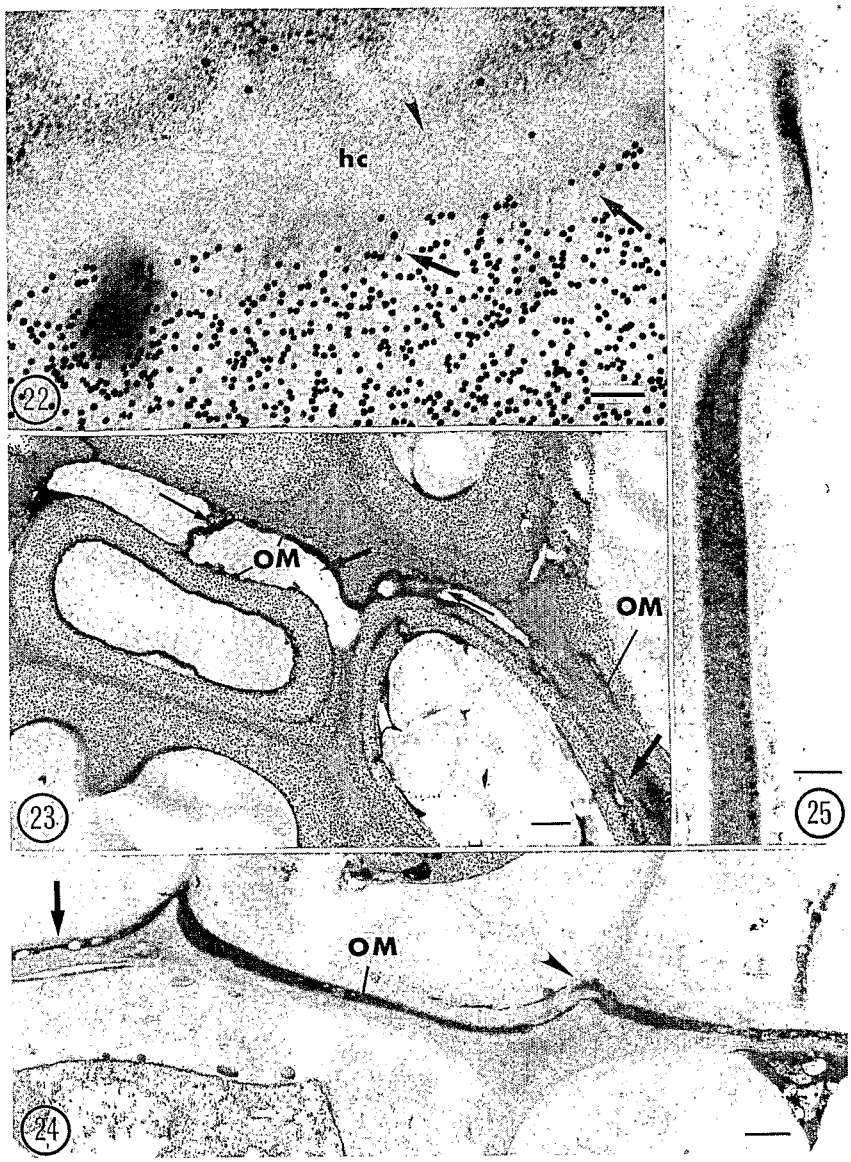
the presence of α-manno- or glucopyranosyl residues appear to be common within ES of rust fungi (113), and fungal symbionts (75). Other sugar residues such as galactose (306, 346), N-acetyl-D-galactosamine (306) and sialic acid (46) were also detected in various fungal ES. These sugar residues are probably present in complex forms as glycoproteins, thought to be involved in cell-cell adhesion and recognition. Antibodies raised against fimbrial proteins of *Ustilago* sp. were used to characterize such molecules in ES (48, 403). Localization of RNA in ES of *Ascocalyx abietina* (=*Gremmeniella abietina*) indicates that cytoplasmic constituents from fungal origin may be present in the sheath (46).

A strong acid phosphatase activity was observed in ES of endomycorrhyzal fungi during HCW penetration (183). Three major types of wall-degrading proteins were localized in ES wood-rotting fungi: (i) peroxidases, such as lignin and manganese peroxidases (65, 127, 421) (Fig. 16), (ii) laccase (344) (Fig. 13), and (iii) glycohydrolases, such as glucosidase (175), endo- and exoglucanases (421) and β-1,4-xylanase (193, 422) (Fig. 17). Microscopic observations revealed that these enzymes can be located over fibrils close to the fungal cell wall or over fibrils that were seen inside HCW (343, 346, 421). In addition, P.A.T.Ag staining performed after gold colloidal labeling strongly suggests a close association of lignin peroxidases with the glucan fibrils (421).

Figs. 22–25. 22. A *Fusarium oxysporum* f. sp. *vasinfectum* cell in close contact with a wall of a cotton xylem cell labeled for cellulose. Portions of fungal cell wall (hc) seem to penetrate HCW (arrows) by means of tubular-like structures, some of which appear to extend from within the fungal cell (arrow heads). Scale bar: 0.1 μm. **23.** *Ophiostoma ulmi* in sterilized *Ulmus campestris* wood sections. Fixation by high pressure freezing. Several small to large cavities (arrows) in HCW associated with OM, the latter encompassing portions of HCW (small arrows). Presence of gold particles indicate positive labeling for cellulose. Scale bar: 0.4μm. **24.** Bands of OM, extending intercellularly, at one point through the membrane of a half-bordered pit (arrow); OM bifurcating towards a secondary wall and possibly in continuity at one point with the smaller OM band present in this wall layer (arrowhead). This band extended between several more cells. Artificially inoculated American elm. Scale bar: 0.5 μm. **25.** A microhypha of *O. ulmi* growing in sterilized *U. campestris* wood sections, fixed with high pressure freezing. This hypha contains solely fibrillar material and opaque bodies, the size of those observed in OM described above. Scale bar: 0.3 μm.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Origin of extracellular sheaths

The origin of fungal ES is poorly documented as compared with their chemical constitution. Bullock et al. (88) suggested that ES of *Sclerotinia minor* result from secretion of fibrillar material through the fungal wall. In contrast, work on ES of wood-rotting fungi (346, 421) revealed close connections between ES and external portions of the fungal cell wall, suggesting that ES could originate from fungal wall components. This observation is consistent with the fact that β-1,3-glucans are constitutive components of both the fungal cell wall and ES (421). Likewise, it has been suggested that extracellular mycofibrils of *Postia placenta* originate from the hyphal surface (285). The extracellular matrix of some rust fungi also appears connected to the haustorial wall but not to the extrahaustorial membrane. In *F. oxysporum* f. sp. *dianthi*, ES is formed as an apparent extension from the outside cell wall layer, with indications that it may also be linked with cell contents (Figs. 12A–C). This ES can also pervade HCW. Proteinaceous fimbriae are thought to be assembled in the fungal cytoplasm and extruded through the wall (393). This was confirmed by immunocytochemical observations (47, 179). On the other hand, Gardiner and Day (179) showed that fimbriae could appear either as extruded long fibrils or as a short fringe embedded in the wall.

Roles of extracellular sheaths

The discovery of extracellular proteins and glycoproteins within rust ES (113, 156, 228, 322) has suggested that they may be involved in binding fungal structures to plant surfaces. Sugars, such as glucose, mannose, fucose, and galactose, characterized in ES and cell wall of several fungi (46, 113, 306, 346) may also play an important role in specific attachment in plant-fungi systems, being bound by plant surface lectins.

Microscopic observations of fungal ES when hyphae were localized in the vicinity of HCW revealed that the ES network plays an essential role in wall degradation. During colonization of living trees or inert wood (336, 343, 421), ES preceded wall penetration by hyphae. Attack of xylem cells was associated with fungal fibrils that extended from hyphae into HCW (Fig. 14) (346). In advanced decay, sheath fibrils were closely associated with detached portions of HCWs, even surrounding and penetrating them (336, 343, 421). At times, ES alone was attached to degraded HCWs (421).

Lignin peroxidases (65, 127, 422, 466), xylanase (193), laccase

(344), and β-glucosidase (175) have been localized within ES of various rotting fungi, as have esterases, cellulases, pectinases, invertase (317), and cutinase (265) in *Colletotrichum* sp. The non covalent loose association between ES and enzymes (193, 421) may support a translocation by ES. The presence of associated β-1,3-glucanases and oxidases (122, 128, 246, 344) that can generate hydrogen peroxide necessary for the function of lignin and manganese peroxidases indicates that ES can be considered as a part of the lignin-degrading system of fungi. Uneven cytodetection of such enzymes in fungal ES, however, suggests their localization in ES may depend upon (i) the enzyme substrate accessibility, (ii) the pathogen environment, and (iii) the physiological condition of fungi.

Several additional roles have been suggested for fungal ES. Oligosaccharides of fungal glucans possibly linked with ES are known to be potent elicitors of phytoalexins (11, 107, 441). In wood-rotting fungi, polysaccharides and proteinaceous compounds of ES may provide nutrition for growing hyphae (471). Fungal ES may also protect fungal cells from unfavorable environmental conditions. For example, the fungal laccase found within *Rigidoporus lignosus* ES (344) polymerizes fungitoxic phenols released from lignin degradation (182).

OTHER SITUATIONS OF HCW ATTACK

Extensive host wall invasion and degradation have also been asssociated with masses and bands of electron opaque material (OM) (361, 362). Pronounced alterations of vessel walls, particularly of the primary and early secondary xylem, are noticeable in infection by *Ophiostoma ulmi* in elm as in nonhost trees (see Fig. 8 in Rioux and Biggs, this volume). Such alterations can be linked to the presence of variously configured OM (Figs. 18A, 18B). Ultrastructurally, such material is constituted of a fine fibrillar matrix and small particles the size of ribosomes (Figs. 19A, 19B). Cell wall breakdown and cavity formation in susceptible carnation can also be linked with bands of OM that seem analogous to the outside wall layer of the *Fusarium* pathogen cells (Fig. 20). Arrays of parallel tubular-like structures that penetrate directly into the host wall extend from these OM bands and masses.

In elm, xylem tissues adjacent to vessel elements of the secondary xylem may also be penetrated by bands of OM of various thickness and configurations with fine to larger strands projecting at angles from the main band (Figs. 23, 24, 25). These bands may extend between or within wall layers, with some portions often

ending within cells (360, 361). Murmanis and colleagues (336) also hypothesize that much of the OM material originates from the fungus.

CONCLUSION

Pathogenic fungi utilise various strategies to penetrate and degrade host cell walls. Appressoria and haustoria are structures involved both in wall adhesion and penetration while fungal sheaths have been shown to play an active role in cell-cell recognition and HCW degradation. However, recent cytological studies have revealed that uncommon fungal components, such as microphyphae, tubular-like structures, or opaque material, may also contribute to HCW alteration. This constitutes a new and exciting research avenue in plant pathology.

CELL WALL CHANGES IN HOST AND NONHOST SYSTEMS: MICROSCOPIC ASPECTS

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When plants are challenged by pathogens that penetrate cell walls directly, through natural openings, or wounds, the plant defense system carries out numerous metabolic modifications, some of which induce cell wall changes visible in light microscopy. These changes may appear in a few isolated cells or be present in many contiguous cells, thus forming a specialized defensive tissue.

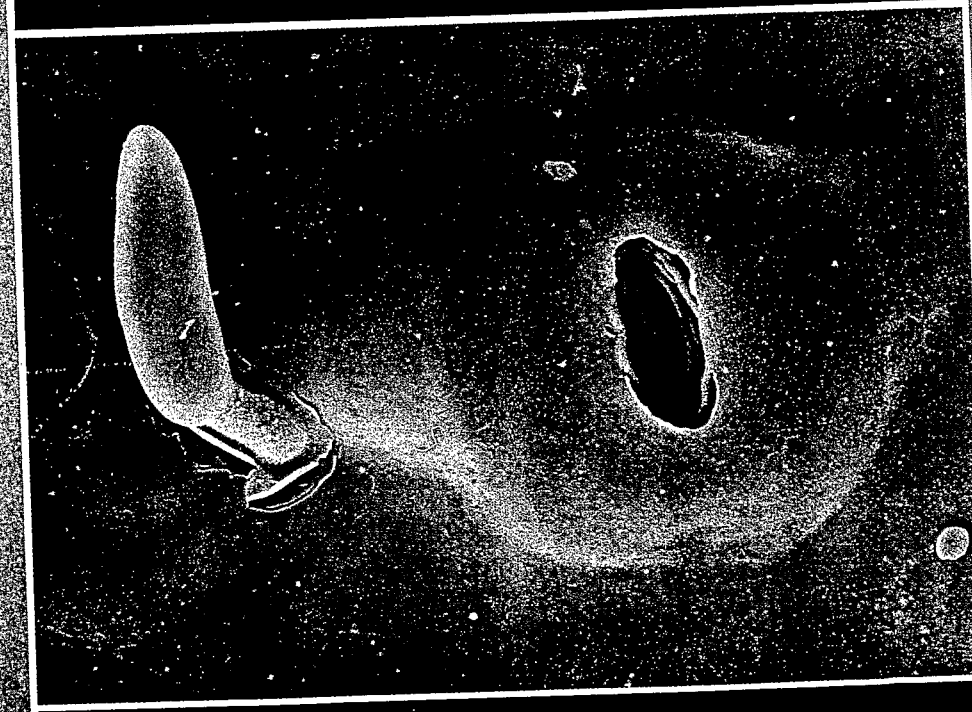
A better understanding of host-pathogen interactions has been obtained in the last two decades by comparing host and nonhost reactions to pathogens (217, 218, 406). Here we present a brief survey of cell wall changes in herbaceous and woody hosts and nonhosts and we discuss the effect these changes may have on the colonization rate of fungal intruders.

NONHOST CONCEPT

A nonhost species is exempt of a particular disease mainly because of resistance factors, but also because of disease escape (324). Thus, each plant species is a nonhost for most known pathogens. Nonhost resistance is mainly based on nonspecific active defense mechanisms that are under multigenic control, and hence shares many similarities with horizontal resistance. Consequently, the pathogen must inhibit or avoid triggering resistance mechanisms in its host or tolerate the effects of the defense reactions (220).

It is possible in some cases to compare cell wall changes that occur in wounded plants with those seen in infected nonhosts. Similarities between these two types of nonspecific defense reactions are more obvious in woody plants. Before discussing such active defense mechanisms, the following section will deal with constitutive characteristics of the nonhosts that appear to be implicated in their resistance to infection.

Host Wall Alterations by Parasitic Fungi



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