Mechanisms of Wood Decay and the Unique Features of Heartrots

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Despite the diversity of microbes associated with decayed hearts of living trees (71,89), the degradation of the cell wall components is still ascribable to Hymenomycetes. Our discussion here is limited to them, although we by no means want to discount their probable interactions with other microbes that might play key roles in the decay process (89). Of the several thousand wood decaying fungi, only a small number—a few hundred—can cause decay in the hearts of living trees (95). The term heartrot is used to include the overall process that culminates in degradation of the wood in the hearts of living trees; decay per se may be only a part of the total process. The interior, primarily nonliving portion of a tree is referred to here as the *heart*. The term heartwood has been avoided.

A great deal is known about the mechanisms by which fungi degrade the principal components of wood. Recent advances have been stimulated primarily by a desire to exploit cellulases and lignin-degrading enzymes. Another lesser incentive has been to understand and control decay in wood products. A desire to understand the mechanism of heartrots, with a view to their eventual control, has been of essentially no consequence in stimulating research on the mechanisms of wood decay. Nevertheless, there is no reason to suspect that the basic mechanisms of wood deterioration during heartrot differ from those in the same wood after the tree dies or has been converted into products. Indeed, two of the most studied wood decay fungi that destroy slash and products have been reported also to cause heartrots (*Coriolus* [*Polyporus*] *versicolor* [11,21] and *Poria placenta* [=*P. monticola*] [11]) although these do not appear to be very important heartrot-causing fungi. Despite a few exceptions like these, however, the same species of fungi usually are not involved in both heartrots and wood products decay (23).

This introduces three intriguing questions concerning the unique features of heartrots, which we shall discuss here:

What special capabilities enable some fungi to invade and decay the hearts of living trees?

Why do many, if not most, heartrot fungi cease to cause decay when the tree is cut or even when it dies (11,23)?

Why is the durability of wood in use not always related to its resistance to decay in living trees (11)?

In the following, decay mechanisms and factors affecting wood decay are reviewed with these questions in mind. The three questions are then addressed specifically in a final section.

WOOD AS A SUBSTRATE

Gross composition and variation. Although all wood basically is composed of the structural polymers cellulose, lignin, and hemicelluloses, there is considerable variation. Variation is particularly evident in the hearts of living trees in which a wide array of nonstructural extraneous materials (extractives) are deposited as the maturing cells die (41,80,85); this variation is primarily between species. Extractives probably are of considerable consequence in heartrot, as discussed later.

Of the structural components, cellulose is the most consistent, varying minimally between wood species. Lignin and hemicelluloses, however, vary both in composition and amounts—especially between hardwoods and conifers, but also among hardwoods; conifer wood is far more homogeneous. Composition of typical woods has been reported by Timell (94). The extents to which the variation in lignin and hemicellulose influences the relative susceptibilities of the hearts of trees to heartrot is not known. In wood products and in slash, there is a strong tendency for softwoods to be degraded primarily by brown-rot fungi and hardwoods by white-rot fungi (84). This generalization appears to extend to heartrots as well, although there are several exceptions, as there are in products decay. The basis for the host preference is not clear, but probably does not lie in the differences in hemicellulose components (38). More likely it is related to the fact that the lignin in hardwoods is easier to biodegrade than that in coniferous wood (101), and white-rot fungi, in apparent contrast to brown-rot fungi, must degrade lignin in order to decay wood.

Chemistry and structure of wood. It is beyond the scope of this article to review in any detail the chemistry of the major wood polymers and their structural arrangements in cell walls. Comprehensive reviews should be consulted (1,13,18,94). For clarity in discussing wood decay, the following brief picture should suffice.

The xylem cell walls that comprise wood derive their basic shape and structure from long cellulose microfibrils. These are arranged in many lamellar sheets (83), which collectively form one primary and three secondary cell wall layers. These layers differ in orientation of the cellulose microfibrils. Each microfibril is a bundle of individual molecules (81). Each molecule consists of 10,000–14,000 β-D-glucopyranose residues linked in straight chains by 1,4 glycosidic bonds. Numerous hydrogen bonds between adjacent cellulose molecules assure a high degree of association (crystallinity). This makes cellulose much less accessible to cellulolytic enzymes than is suggested by its chemical composition alone, which is similar to that of starch, a readily biodegradable polymer.

Filling the spaces between the microfibrils, and between the

lamellae that they comprise, is a matrix of hemicelluloses and lignin. The area between primary cell walls of contiguous cells, the middle lamella, is 40–85% lignin. Approximately 80% of the lignin, however, is within the secondary walls (29). Wood hemicelluloses (94) are also β-1,4-linked glycans. They are both linear and branched and are relatively short (containing perhaps 100–300 sugar resiudes) in comparison to cellulose. Substituents on these polymers include acetyl groups, monosaccharides, and uronic acids. Hardwood hemicelluloses are mainly substituted xylans (β-1,4-linked D-xylopyranose units), and the major hemicelluloses of conifers are glucomannans—copolymers of D-glucopyranose and D-mannopyranose units linked β-1,4.

Lignin is quite different from the hemicelluloses and cellulose, and is also the most resistant to biodegradation. It is a three-dimensional, amorphous, branched polymer of phenylpropane units joined by a variety of interunit linkages (1). Lignin is formed by a random free-radical oxidative copolymerization of three different oxy-cinnamyl alcohols which occur in varying ratios depending on the tree species. The greatest differences are between conifers and hardwoods and between normal and reaction woods; conifers contain only small amounts of sinapyl alcohol-derived units, which are common in hardwoods. Relative to wood decay, lignin has two noteworthy features: (a) it is largely nonhydrolyzable, differing in that respect from almost all other biopolymers, and (b) it forms a protective layer around the wood polysaccharides that limits cellulase accessibility within the cell walls.

Lignin and hemicelluloses within the cell wall are covalently linked but it is not clear how. Recent studies suggest various modes of such connections, but not their frequencies (72,100). Nothing is known about the importance of lignin-carbohydrate bonds in wood decay, but probably they are of minor consequence.

MECHANISMS OF WOOD DECAY

Heartrot fungi, as well as other wood decay fungi, can be classified as causing either white rots or brown rots. The two types of decay differ markedly in physical and chemical characteristics (19,51), as is described in the following. Table 1 lists some examples of both types of fungi, their hosts, and certain other characteristics which will be discussed later.

White rot. White-rot fungi degrade cellulose and the hemicelluloses at approximately the same rates relative to the original amounts present, whereas lignin is decomposed at a similar rate, or usually somewhat faster on a relative basis.

Wilcox (97) and Liese (66) reviewed the microscopical features of decay. Initially, hyphae of white-rot fungi are concentrated in the ray cells and vessels, although other cells are invaded very early in decay. The hyphae initially invade other cells from ray cells and vessels via pits, or directly by penetration of cell walls. Many different enzymes are secreted at hyphal tips and on lateral surfaces; these enzymes assist cell wall penetration and enlarge bore holes to perforations at the later stages of decay.

Hyphae growing inside the cell lumens degrade the layers of the secondary wall from the inside, progressing successively from the S_3 layer outwards. Along the young hyphae, lysis furrows are produced, which deepen and coalesce with time toward the highly lignified middle lamella, which is relatively resistant to attack. The degradation products of the various wall layers seem to be completely absorbed by the hyphae. Cowling (19) showed by chemical analysis that white-rot fungi successively depolymerize cell wall substances only to the extent that the products can be utilized consecutively for metabolism. The action of the enzyme system of white-rot fungi is restricted to the cell wall layers in the immediate vicinity of the hyphae, in contrast to that of brown-rot fungal enzymes, which apparently diffuse into the inner layers of the cell wall.

Eriksson (24) recently reviewed the mechanism of cellulose hydrolysis by a white-rot slash-degrading fungus, *Phanerochaete chrysosporium* (the teliomorph of *Sporotrichum pulverulentum*). His extensive studies, as well as those by other workers with imperfect fungi (5,9,76,99), support the hypothesis that one or more endo-1,4-β-D-glucanases act randomly over the exposed surface of crystalline cellulose. The exposed nonreducing termini are then hydrolyzed by an exo-1,4-β-glucanase, with production of cellobiose. Cellobiose may be cleaved by a β-glucosidase, yielding glucose. Exo- and certain of the endoglucanases thus act synergistically—perhaps as a loose complex (99). Certain of the endoglucanases could then be considered to be C_1 components of the cellulose complex. Excellent discussions of the C_1-C_x hypothesis in light of very recent findings are provided by Reese (76) and by Wood and McCrea (99). In *P. chrysosporium,* five endoglucanases and one exo-glucanase were found. The total weight of the endoglucanase proteins was approximately equal to that of the exo-glucanase protein.

Hydrolytic enzymes are not the only ones involved in cellulose degradation by white-rot fungi, however. The results of very recent research has disclosed a hemoprotein, cellobiose oxidase, which oxidizes cellobiose to cellobiono-δ-lactone, with O_2 serving as electron acceptor. The enzyme is responsible for the much more rapid hydrolysis of cellulose in O_2 than in N_2 (24). Similar activity is

TABLE 1. Some characteristics of selected heartrot and wood slash and wood products decaying fungi

Fungi	Hosts	Type of decay	References
Primarily causing heartrot			
Phellinus robineae	Black locust	White	8, 11
Tyromyces amarus	Incense cedar	Brown	8, 11, 98
Inonotus rheades	Hardwoods	White	8
Causing heartrots and slash/products decay			
Coriolus versicolor	Hardwoods	White	11
Poria placenta	Conifers	Brown	11
Haematostereum sanguinolentum	Balsam fir	White	28
Causing primarily slash/products decay			
Phanerochaete chrysosporium	Hardwoods	White	67, 75
Gloeophyllum saepiarium	Conifers	Brown	8, 11, 23
Phlebia phlebiodes	Conifers	White	68, 69

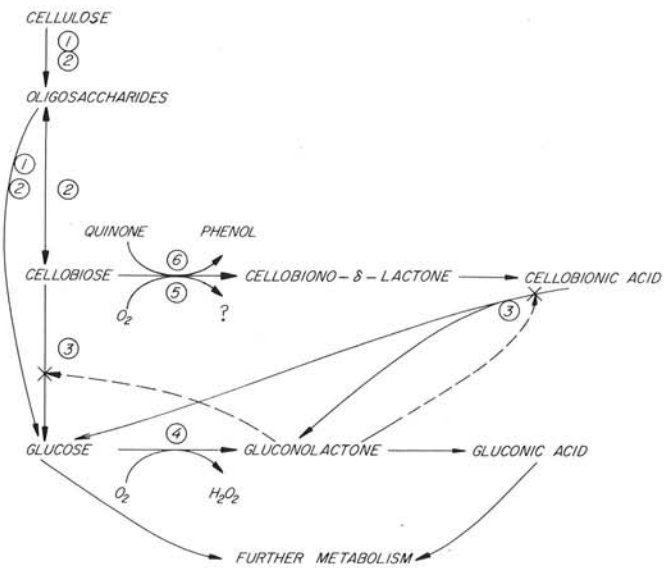

Fig. 1. Enzyme mechanism for cellulose degradation by *Phanerochaete chrysosporium* (adapted from Eriksson [24]). Enzymes: (1) endo-glucanase; (2) exo-glucanase; (3) β-glucosidase; (4) glucose oxidase; (5) cellobiose oxidase; and (6) cellobiose: quinone oxidoreductase.

not found in the imperfect fungi examined (99). Cellobiose is also oxidized to cellobiono-δ-lactone by cellobiose-quinone oxidoreductase; quinones serve as electron acceptors for this activity (96). A glucose oxidase also has been implicated in the overall process; it oxidizes glucose to gluconolactone (24). These various oxidizing activities seem to regulate the levels of glucose and cellobiose, and ultimately coordinate the rates of cellulose hydrolysis and metabolism of end products. Fig. 1 summarizes the interconversions and regulatory interactions in cellulose hydrolysis as proposed by Eriksson (24).

One of the differences between heartrot fungi and other wood decay fungi might be in their responses to the very low oxygen concentrations (see "Environmental Factors . . .") in the hearts of living trees. This should be studied further. O_2 concentration also is of central importance in lignin metabolism, as discussed below.

Degradation of hemicelluloses proceeds in a manner roughly analogous to that of cellulose, but the mechanism of attack has been studied in much less detail (22). The hemicellulose chains are attacked first by endo-enzymes ("mannanases," "xylanases") which produce progressively shorter chains, which are hydrolyzed to simple sugars by β-glycosidases ("mannosidases," "xylosidases," "glucosidases"). Whether exo-enzymes are involved has not been determined (22). The enzymes involved in the removal of side chain substituents (arabinose, uronic acids, acetyls) apparently have not been studied.

As with cellulases, simple sugars repress the production of most hemicellulose-degrading enzymes by white-rot fungi (25,38). Cellulose apparently is the only carbon source necessary to induce the formation of hemicellulose-degrading enzymes by white-rot fungi (25,38). Eriksson and Goodell (25) proposed that a single regulatory protein governs the induction of cellulase, mannanase, and xylanase in *Bjerkandera adusta* (=*Polyporus adustus*). They further indicated that controlling the induction of these enzymes by means of a single regulator protein would be a simple, effective way of adjusting the rate at which wood is degraded.

Ahlgren and Eriksson (2) studied some of the properties of hemicellulose-degrading enzymes of *Haematostereum* (*Stereum*) *sanguinolentum* and *Heterobasidion annosum* (=*Fomes annosus*). Mannanase was found to have a relatively large molecular size compared to xylanase and cellulase, but all the endoenzyme molecules were smaller than their corresponding β-glycosidases. Isoelectric separation showed that most of the hemicellulose-degrading enzymes, like the cellulases, are proteins of acidic character. The pH optima of the hemicellulose-degrading enzymes also are in the acid range (pH 4–5).

Lignin degradation by white-rot fungi has been reviewed very recently (4,55,57). The specific degradative enzyme activities involved in lignin metabolism have not yet been fully described. Nevertheless, it seems apparent that the polymer is attacked first by oxygenases, with formation of low-molecular-weight aliphatic and aromatic products which are taken up by the hyphae and further metabolized. Rapid progress is currently being made toward understanding the ligninolytic system.

It was found recently that lignin is not degraded at a significant rate by *Phanerochaete chrysosporium* or by *Coriolus versicolor* under an atmosphere of 5% O_2, although growth was unaffected by the low O_2 atmosphere (58). It would be of interest to determine whether heartrot fungi causing white rots also exhibit a strong dependence on an O_2-rich atmosphere to degrade lignin.

Brown rot. Brown-rot fungi utilize the hemicelluloses and cellulose of the cell wall, leaving the lignin essentially undigested. They do modify the lignin, however, as indicated by demethylation and the accumulation of oxidized polymeric lignin degradation products (52,53). Quite recently, isolated lignins unexpectedly were shown to be degraded to CO_2 to an appreciable extent by brown-rot fungi in liquid cultures (34). Evidently the differences between the conditions in culture and in decaying wood profoundly affect the lignin-degrading ability of brown-rot fungi. This should be studied further.

Hyphae of brown-rot fungi, like those of white-rot fungi, grow inside the lumina in contact with the tertiary wall, into the capillaries of which the secreted enzymes are able to diffuse (6,66,97). Microscopic observation of brown-rotted wood indicates that enzymatic attack is not localized near the hyphae. Apparently, there is widespread and deep diffusion of the degrading catalysts. As decay proceeds, the cellulose fragments and the hemicelluloses are gradually destroyed at approximately the same relative rates.

Brown-rotted wood tends to shrink abnormally when dried, giving rise to a characteristic cubical pattern of checking. At comparable stages of decay, as measured by weight loss, brown-rot fungi reduce the strength of wood much more than do white-rot fungi. The alkali solubility of brown-rotted wood also increases substantially. These changes occur because brown-rot fungi drastically reduce the degree of polymerization of cellulose even at very low weight loss; the decomposition products are readily soluble in alkali. The degradation products are produced faster than they are utilized (19).

Thus, the brown-rot fungi must produce an agent which penetrates the microfibrillar structure of cellulose and breaks down the glucosidic bonds in cellulose very early during decay. Polysaccharidases cannot penetrate the lignin barrier to get to polysaccharides unless the lignin itself is degraded and, as mentioned above, brown-rot fungi do not cause much damage to lignin in wood. Therefore, to cause this rapid initial depolymerization of cellulose, an agent with molecules smaller than any known cellulase would be required, as recognized first by Cowling (19). No isolated cellulase has been able to duplicate the effect of brown-rot fungi on wood (20,39). In fact, cellulase preparations from most brown-rot fungi exhibit very little activity toward crystalline-insoluble celluloses (37,40). The fungi do, however, possess an enzyme that degrades soluble celluloses (carboxymethylcelluloses). Therefore, they evidently employ a different mechanism for attacking the cellulose in wood (20). Recent studies have suggested that this is the case (39,40,59–62).

Koenigs (59–62) discovered evidence that the mechanism of cellulose degradation by brown-rot fungi is nonenzymatic, and that the active agent might be an H_2O_2/Fe^{++} system. He found that brown-rot fungi produce sufficient H_2O_2 to degrade cellulose with the amounts of Fe^{++} present in woods. In addition, the effects of H_2O_2/Fe^{++} on cellulose mimic those of brown-rot fungi (61,62). The hydrogen peroxide molecule is small enough to penetrate the microstructure of the secondary cell wall, attack the cellulose, or perhaps predispose the cellulose to degradation by conventional cellulases. This kind of mechanism would be oxidative. Highley (39) recently, and others (7,12) many years ago, presented evidence that brown-rot fungi do oxidize cellulose. Additional study is needed, however, to establish the exact mechanism of cellulose degradation by brown-rot fungi.

Hemicelluloses also are utilized by brown-rot fungi. Chemical analysis of brown-rotted coniferous wood showed that the major hemicellulose, glucomannan, apparently is removed initially at a higher rate than is cellulose, which suggests that further degradation and removal of depolymerized (oxidized) cellulose may depend on prior removal of this major hemicellulose component (56). In accord with this finding, Lyr (70) reported that the hemicellulase activities of several wood-destroying fungi in culture reached a maximum rapidly and before cellulase activity reached a maximum. Utilization of noncellulosic carbohydrates may result in the production of metabolic products such as H_2O_2, which are necessary for cellulose degradation. The brown-rot fungus, *Poria placenta,* could not utilize isolated cellulose, but cellulose in the presence of hemicelluloses was utilized rapidly (39). The mechanism of hemicellulose breakdown by brown-rot fungi appears similar to that of white-rot fungi (38,49,50). However, little definitive work has been done on the hemicellulases of brown-rot fungi with respect to specificities, molecular size, and other properties of the enzymes such as active sites. Like white-rot hemicellulose-degrading enzymes, the brown-rot enzymes have acidic pH optima (38). The molecular sizes of two hemicellulases from the brown-rot (heartrot) fungus, *Phaeolus* (*Polyporus*) *schweinitzii* were found to be about the same as that of the cellulase from the same fungus, 35,000 to 37,000 daltons (49). Thus, these enzymes also would be too large to diffuse into the transient capillaries of wood.

ENVIRONMENTAL FACTORS INFLUENCING WOOD DECAY

In addition to the chemical and physical constraints imposed by the structure and chemistry of wood, several other factors greatly influence decay rates.

Nutrients. The basic nutritional requirements of wood-rotting fungi are satisfied by the structural carbohydrates and certain extraneous materials in wood. Degradation of lignin does not occur in the absence of the metabolism of wood carbohydrates, and lignin is probably of limited importance as a carbon source (54).

Among nonstructural nutrients in wood, nitrogen plays the most important role. The requirements evidently are not great; wood usually contains about 0.03–0.1% nitrogen (73). Wood decay fungi have an efficient mechanism for its metabolism and reuse (65). Studies of growth and nutrition of 42 wood-rotting Basidiomycetes in synthetic media (45) revealed that none required nitrogen in organic form, but that growth was usually greater with organic nitrogen than with ammonium salts. Supplemental nitrate did not support the growth of 41 of these fungi.

The role of nitrogen in wood decay has been extensively studied by Cowling and coworkers (65,73). Decay rates were highly correlated with the nitrogen content of the individual annual increments (73). Recent field evidence (64) and results of in vitro culture studies support the theory that in wood-decaying fungi, the autolysis and reuse of the nitrogen in their own mycelium, or the lysis of other fungi in wood during decay, together with an extremely economical use of nitrogen in metabolism, could contribute to the ability of wood-destroying Basidiomycetes to conserve and function with the small amount of nitrogen available in wood. However, for sporophore formation, other sources of N, such as bacterial fixation (64) may be required in some cases.

Recently, data from several studies (3,64) have demonstrated nitrogenase activity in decaying heart tissue of live standing coniferous trees, and several groups of bacteria have been reported (86) that apparently are capable of fixing dinitrogen anaerobically. Furthermore, the data from these experiments support the contention, stated elsewhere in this paper, that heartrot fungi are capable of growing at extremely low O_2 levels.

Thiamine is the only vitamin essential for the growth of most wood-decay fungi, and it is provided by wood (45).

Extraneous compounds. The hearts of many trees are particularly rich in extractives; ie, materials extractable with neutral solvents (41,80). These nonstructural constituents include many secondary metabolites of trees, among them alkaloids, terpenes, and a variety of phenolic compounds, which powerfully influence the susceptibility of host woods to decay.

Many are highly toxic to decay fungi (82,85). Some extractives, however, have been shown to stimulate growth of wood decay fungi (32,77,78,79).

Some of the extraneous components in the hearts of living trees are volatile. Fries (31) has reviewed the effects of such materials on growth and development of fungi. He believes that many exert a powerful influence, acting perhaps as regulators of intermediary metabolism. Many markedly stimulate metabolic activities. Production of such compounds by the living tree, and their diffusion into the dead heart tissues conceivably could exert a selective pressure.

Temperature. Temperature is one of the many ecological parameters affecting the activities of wood-decaying fungi. Most of these fungi are mesophillic, generally unable to grow above 40 C, and with temperature optima of 25–30 C (15,16,30,45). Even most tropical wood decay fungi have temperature optima of 30 C or less (87). Some wood decay fungi, however, have special tolerance to higher temperatures, with optima above 40 C and being unable to grow below 20 C.

Jensen (47) studied the effect of fluctuating temperatures on growth of four heart-rot fungi and concluded that if temperature fluctuations were small, growth of the organisms was stimulated, whereas if the fluctuations were large, the growth of the organisms was suppressed. He also determined the internal tree temperature of red oak and found, as expected, that as the air temperature increased, the internal tree temperature also increased, but at a slower rate, and did not rise as high as the air temperature unless that remained high for several hours. The same pattern was noted with a decrease in temperature. Thus, in a tree, temperature fluctuations probably have only minimal effect on fungal activities. In contrast, in decaying slash there are rapid and substantial temperature fluctuations so that the activities of decay fungi frequently are affected.

Loman (68,69) investigated the influence of high temperatures on fungi decaying lodgepole pine slash in Alberta, Canada. *Phlebia* (*Peniophora*) *phlebioides* and *Gloeophyllum saepiarium* (=*Lenzites sepiaria*), which have high temperature optima and wide temperature tolerances, were the dominant decay fungi in the upper 5 cm of exposed slash. *Haematostereum sanguinolentum* and *Coniophora puteana,* which have lower temperature optima and narrower temperature tolerances were active at greater depths in the slash. Temperatures lethal to *Coniophora* and *Stereum,* but not lethal to *P. phlebioides* and *G. saepiarium,* were measured in the upper and central parts of slash during periods of hot weather. Occasionally, low-temperature fungi were found in the upper parts of slash, suggesting that they were able to invade these areas during cool periods.

pH. The acidity of wood depends to a considerable degree on the presence of volatile acids, the most important of which are acetic and formic acids; the content of acetic acid in the wood of living trees may exceed 0.4% per unit of dry weight (43). Because of the free acids, the pH of fresh wood often may be quite low. According to the data of Gray (33), however, the acidity of hardwoods varies between 2.8 and 6.8, while the pH of softwoods varies between 2.7 and 8.8.

The pH of sapwood differs from that of the nonliving central wood. J. C. Ward (*personal communication*) found the pH of functional sapwood to be in a relatively narrow range around 6.0 for both conifers and hardwoods. The pH of heartwood can vary from less than 3 to greater than 7. Also the pH of sapwood can be altered with drying conditions.

Jennison (45) found the growth optimum of 42 species of decay fungi to vary between pH 3.5 and 5.5; he did not test below pH 3.5. Butcher (14) found wood-inhabiting fungi to have a comparatively wide pH range, at least 4.0–9.0, and a growth optimum between pH 5.0 and 6.0. Brown-rot fungi can withstand more acidic conditions than can white-rot fungi and they grow more poorly at higher pH's than do white-rot fungi (35). Highley (38) found the cellulases and hemicellulases of brown-rot fungi to be more tolerant of low pH and more sensitive to high pH than those of white-rot fungi. Medium acidity was shown recently to exert a strong influence on the rate of lignin degradation by the white-rot fungus *P. chrysosporium*; little degradation occurred below pH 3 or above pH 5 (58). While decaying wood, brown-rot fungi create a low pH, primarily by oxalic acid production (93); however, unlike white-rot fungi, which also produce oxalic acid, the brown-rot fungi are unable to metabolize this product (91,93).

The tolerance of 125 Basidiomycetes to acidity was compared by Hintikka (43). Tolerance of acidity was found to be greater in wood-inhabiting than in litter-decomposing Basidiomycetes. Wood is more dense in structure than is soil and is not as well ventilated. Thus, Hintikka (43) suggested that acetic acid accumulates in wood in greater quantities than in soil, and may, like other chemical factors, exert a selective effect on the invading microbial population.

O_2 and CO_2. A heartrot fungus attacking a tree, often must grow in a near-anaerobic atmosphere with high CO_2 concentrations (44,48,63,102). Oxygen concentrations of less than 1% of the volume of gases in tree trunks is apparently common (48) and CO_2 concentrations as high as 11% and 20% were reported by Hintikka and Korhonen (44) and Jensen (48), respectively.

Wood-degrading fungi evidently vary in capacity to grow in atmospheres high in CO_2. Hintikka and Korhonen (44) found that several wood-inhabiting fungi continued to grow in 70% CO_2, whereas many litter-decomposing fungi were totally inhibited by 30% CO_2. Jensen (46) found growth of four wood-decay fungi to be inhibited by subatmospheric O_2 concentrations and CO_2

concentrations of 10% or more. No measurable growth was observed without O_2, but growth occurred at 1% O_2, and increased as O_2 increased. In nature, low O_2 is invariably associated with high CO_2. Inhibition of growth by CO_2 appears only at low O_2 tensions.

CO_2 also affects spore germination. Morton and French (74) found that germination of basidiospores *Inonotus rheades* (=*Polyporus dryophilus*) was stimulated by CO_2. Removal of CO_2 prevented germination.

Moisture. The moisture content of tree hearts will differ depending on species and site (27). Generally, the heart contains less moisture than sapwood (89). *Picea glauca* sapwood contains 138–162% moisture while the heartwood contains only 47–48% (17). Similar differences have been reported in pine (41). Some hardwood hearts, however, have higher moisture contents than the sapwood (42). Wounded standing trees will have a higher moisture content than wind-thrown or insect-killed trees. These differences in moisture content in trees and the subsequent changes in moisture content have a profound effect on decay activities.

Etheridge (27) studied the relationship between infection of subalpine spruce and moisture content. His data showed that even though a difference in moisture content of only 3% of saturation existed between the hearts of trees on moist sites versus dry sites, the lower moisture content of the hearts of dry-site trees was critical in preventing infection by the fungi. He further showed in laboratory tests that small changes in moisture content have a significant effect on decay; a difference in moisture content of only 3–4% of saturation had a critical effect on the rate of decay by *Coniophora puteana.*

Nondecay fungi and bacteria. Ample evidence now has been obtained to suggest that decay fungi often, if not invariably, occur after or together with other microorganisms (10,90). We do not propose to discuss the many implications of such microbial interactions in heartrot; they have been covered adequately by Shigo and Hillis (89). It suffices to say that microbial interactions obviously can be of direct or indirect influence, enhancing or hindering establishment of decay fungi, their decay rates, and their survival in the hearts of trees.

UNIQUE FEATURES OF HEARTROT

There is no reason to suspect that heartrot fungi differ from other decay fungi in the basic chemical mechanisms of decay. Thus, the answers to the three questions relating to heartrots, posed in the introduction, will not be found in a consideration of the decay mechanisms per se. Rather, it appears much more probable to us that certain as-yet-undetermined physiological characteristics separate heartrot fungi as a group from the wood-decaying fungi that do not attack hearts of living trees. Some fungi, such as *Phellinus nigricans* (=*Fomes igniarius*), *Coriolus versicolor,* and *Poria placenta,* apparently possess physiological traits of both groups. (In the following discussion of the three questions concerning heartrots we are only considering the initial phases in which the solid heart of the living tree is being decayed. Once decay has become advanced and the tree is hollowed out, invaded by insects, etc., tree hearts are not likely to present unique environments.)

What enables some decay fungi to cause heartrots of living trees? Many heartrot fungi invade trees through dead branch stubs (11). It seems unlikely that fungi decaying slash of a given wood species would be unable to colonize dead branch stubs of the same tree species. The essential difference between slash and heartrot fungi, therefore, must be in ability to penetrate into, survive in, and cause decay in the living tree's interior. Since no active resistance mechanism is involved in such entries through dead tissue, it follows that as successful colonizers, heartrot fungi can tolerate the chemical and physical constraints within the tree trunk. Constraints which are obviously suspect in this regard are levels of O_2, levels of CO_2, and the concentration and nature of volatile organic compounds. (Interaction with other microbes, either through these factors or more directly, also must be considered [89,92)]. Also, a very important determining factor can be host response when entry is via wounds (88). We are limiting the speculative discussion here to a consideration of decay once the fungi have gained entry to the tree interiors, and without involving interactions with other microorganisms.

We already have suggested that heartrot fungi might have unusual tolerances for low O_2 with respect to cellulose and lignin degradation. This should be studied. Hintikka and Korhonen (44) have noted that lack of oxygen is invariably associated with high CO_2, and they have suggested that CO_2 plays an important role in the ecology of higher basidiomycetes. They found that wood decay fungi were much more tolerant of high CO_2 than were litter-decomposing species. Examination of their data, however, suggests that among the decay fungi that they studied, heartrot fungi did not exhibit a uniquely high tolerance to CO_2.

Response to common volatile organic materials (eg, acetic acid) within living trees conceivably could be involved in selecting heartrot fungi over other decay fungi; this could be expressed via stimulation of the former fungi, suppression of the latter, or both. Tolerance to extraneous materials other than volatiles is probably unlikely to be involved in selecting for heartrot fungi as a group. Once removed from the living tree, wood may be readily degraded by many fungi which do not cause heartrots.

Related to the question of why some fungi cause heartrots is the question of host specificity of heartrot fungi. Tree species vary greatly in moisture content and pH of the heart tissue, and undoubtedly in O_2 and CO_2 contents of the internal gases. These differences probably are involved in determining host specificity. Extraneous materials probably are major determinants of host specificity. Relatively few cases have been studied, but Wilcox (98) showed that the incense cedar heartrot fungus *Tyromyces* (*Polyporus*) *amarus* is peculiarly tolerant of the fungitoxic extractives of its host, and similar results with *Haematostereum sanguinolentum* and balsam fir were reported by Etheridge (28). The possible involvement of extraneous volatile materials in host specificity apparently has not been studied.

Why do many heartrot fungi cease to cause decay when the host tree dies or is converted into products? As has been mentioned, slash-rotting fungi apparently are adapted to the extremes of temperature fluctuations encountered in slash and in wood products. It seems likely that evolution has resulted in a loss of such tolerance in heartrot fungi. The same probably is true of moisture relationships. It also seems probable that the advantage conferred by the physiological characteristics that enable a heartrot fungus to invade and decay the hearts of living trees could be lost when the tree is converted.

Why is the durability of a wood species in use not always related to its resistance to decay in the living trees? Incense cedar and black locust are decay resistant in service, but are frequently severely decayed by the heartrot fungi *T. amarus* and *Phellinus robineae* (=*Fomes rimosus*), respectively, in the living tree. One can ask what makes such woods become resistant when the trees are cut. In most cases there is actually no increase in resistance on cutting, and that under "favorable" environmental conditions wood will decay at least as rapidly after being cut as it does in the living tree. Eslyn (26) noted that heartrot fungi evidently continue to decay cut tree boles—in the form of utility poles—for as long as they remained in a green or semi-green condition. However, poles that had been dried prior to installation were not invaded by heartrot fungi.

In contrast to incense cedar, living ponderosa pine is relatively free from heartrot (11), but the wood is quite nondurable when cut. In such cases, the living tissues of the tree obviously protect the central, dead wood. This protection could be active, as in the prevention of entrance of the fungus by a vigorous response to wounding, or passive, as in maintenance of an internal environment unsuitable for decay development (eg, near anaerobicity).

OBSERVATIONS

Speculation on the unique features of heartrot fungi has been presented here to stimulate research. It would appear that comparative studies of carefully selected heartrot fungi and slash or wood products-degrading fungi (avoiding those with dual activity) should be made. This could lead not only to answers to the

questions posed here, but also could suggest means for screening trees for resistance to heartrots.

As has long been recognized (11,36), it is common to find two trees which are of the same age and size, both nonwounded or wounded in identical manners, and growing under the same conditions within a few feet of each other, with one being sound and the other being seriously decayed by heartrot. These observations suggest the possibility that genetic differences in heartrot susceptibility exist which might be exploited to select and develop resistant lines. Other than silvicultural practices, this may be the only practical approach to controlling these most damaging of all forest fungi.

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