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Review

Reactive oxygen species as agents of wood decay by fungi

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Abstract

Although many wood decay basidiomycetes secrete oxidative and hydrolytic enzymes that participate in lignocellulose biodedgradation, it is generally recognized now that these enzymes cannot penetrate sound wood, and that fungi must employ smaller agents to initiate decay. Reactive oxygen species (ROS) are likely candidates, and evidence is accumulating that some wood decay fungi produce these oxidants. For example, we have observed that, when they grow on wood, white rot and brown rot basidiomycetes degrade polyethylene glycol, a recalcitrant polymer that resists attack by species other than nonspecific, extracellular oxidants. By contrast, xylariaceous ascomycetes do not degrade polyethylene glylcol, and therefore do not produce these oxidants when they degrade wood. The ROS that basidiomycetes employ remain unidentified in most cases, but the hydroxyl radical (·OH) is a likely participant, because there is a well-established pathway for its production via the Fenton reaction ($H_2O_2 + Fe^{2+} + H^+ \rightarrow H_2O + Fe^{3+} + \cdot OH$). The two best documented fungal pathways for ·OH production involve the extracellular reduction of Fe^{3+} and O_2 by cellobiose dehydrogenase or by secreted hydroquinones. Peroxyl (ROO·) and hydroperoxyl (·OOH) radicals, although they are weaker oxidants, may also be involved in wood decay because they are produced via reactions of ·OH and by some fungal enzymes. © 2002 Elsevier Science Inc. All rights reserved,

Keywords: Wood decay fungi; White rot; Brown rot; Soft rot; Hydroxyl radical; Peroxyl radicals; Fenton chemistry

1. Introduction

The hypothesis that wood decay fungi use extracellular reactive oxygen species (ROS) to degrade lignocellulose dates from the middle of the last century [1,2]. This idea has been somewhat neglected in the last twenty years, probably because interesting fungal peroxidases with ligninolytic properties were discovered in the intervening time [3–5]. However, it is now clear that lignocellulose-degrading enzymes are too large to penetrate lignified cell walls in sound wood [6,7], and these results have refocused attention on ROS, which might be the agents that initiate decay within the secondary wood cell wall. Most of the work to date has focused on hydroxyl radicals, (.OH), but other ROS, such as peroxyl radicals (ROO) or hydroperoxyl radicals (·OOH), might also be produced by fungi to attack wood polymers. This review will focus on the abovementioned species, which are the ROS most often encountered in biologic systems, but it is important to note that some other biologic ROS have not been ruled out as agents of wood decay. These agents include singlet oxygen, hypochlorous acid, and peroxynitrite [8].

There are three major types of wood decay by fungi, and ROS have been implicated in all of them. White rot basidiomycetes degrade both wood polysaccharides and lignin, and in some cases delignify wood selectively [6,9]. There are reports that white rot fungi produce extracellular ·OH [10–12], but they also secrete peroxidases, laccases, and cellulases that undoubtedly participate in lignocellulose degradation [3–5]. Brown rot basidiomycetes degrade wood polysaccharides while leaving behind an oxidized, polymeric lignin residue [9]. Brown rotters lack most of the oxidative enzymes that white rot fungi have, and most of them lack complete cellulase systems, but many studies report that these fungi produce ·OH [13–21].Soft rot ascomycetes and deuteromycetes resemble brown rotters in that they degrade wood polysaccharides more readily than lignin [9]. Little is known about their decay mechanisms, but there is evidence that some produce ROS [22,23].

2. Hydroxyl radical

The hydroxyl radical (·OH) has received more attention than other ROS in studies of wood decay, in part because there is a well-recognized route for its production in bio-

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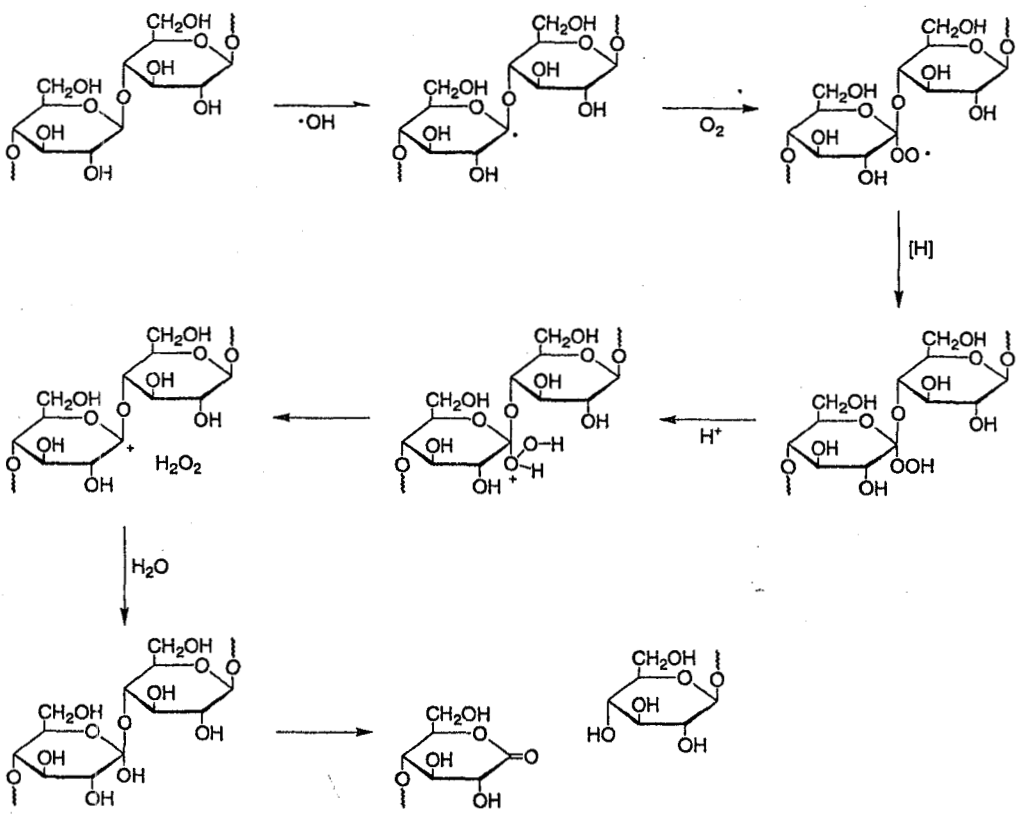

Fig. 1. One of many possible routes for the oxidation of cellulose by 'OH. This route cleaves the glycosidic linkage and generates a gluconic acid residue (as the lactone), which is a reaction also accomplished by brown rot fungi [21].

logic systems via the Fenton reaction ($H_2O_2 + Fe^{2+} + H^+ \rightarrow H_2O + Fe^{3+} + \cdot OH$). Although iron in most biologic systems is generally sequestered in redox-inactive complexes to prevent oxidative damage via Fenton chemistry [8], this is not the case in wood, which contains enough iron to make ·OH generation feasible [2], provided chelators or reductants are available to solubilize the metal. In addition, ·OH is a reasonable candidate for a wood decay agent because it is the strongest oxidant that can occur in aqueous systems. It reacts rapidly with virtually all organic molecules, either by abstracting hydrogens from aliphatic structures or by adding as an electrophile to aromatic ones [8].

2.1. Attack of ·OH on polysaccharides

·OH abstracts hydrogen atoms from the sugar subunits of polysaccharides such as cellulose with high rate constants around 10^9 M^{-1} sec^{-1} [24]. These reactions produce transient carbon-centered radicals that react rapidly with 0, to give ROO. species (Fig. 1). If the peroxyl radical already carries a hydroxyl group on the same carbon, it eliminates ·OOH [8]. If it carries no a -hydroxyl group, it undergoes a variety of oxidoreductions, some of which result in cleavage of the polysaccharide chain. Because these reactions lead to complex product mixtures, and because cellulose is difficult to solubilize for analysis, it has not been rigorously proven that any decay fungus uses ·OH to cleave this polysaccharide. However, cellulose degraded by one brown rot fungus, *Postia placenta*, was shown to contain small quantities of oxidized residues that suggested attack by ·OH [21].

2.2. Attack of ·OH on lignin

Studies with lignin model compounds indicate that ·OH attacks the subunits of lignin with rate constants around 10^{10} M^{-1} sec^{-1} [24], both by abstracting aliphatic C_a-hydrogens and by adding to aromatic rings (Fig. 2). When hydrogen abstraction occurs, the product is a benzylic ketone, whereas addition to the ring gives a hydroxylated cyclohexadienyl radical. Under acidic conditions, this ring-oxidized intermediate can combine with a proton and eliminate water, thus yielding an aryl cation radical that undergoes carbon-carbon bond cleavage and other degradative reactions [25]. However, published data indicate that the cyclohexadienyl radical is more likely to decay to other products in which the aromatic ring becomes hydroxylated [25,26]. For example, when ·OH addition occurs at unsubstituted ring positions, the radical is further oxidized and loses a proton to introduce a new phenolic hydroxyl group. On the other hand, when a position with a methoxyl sub-

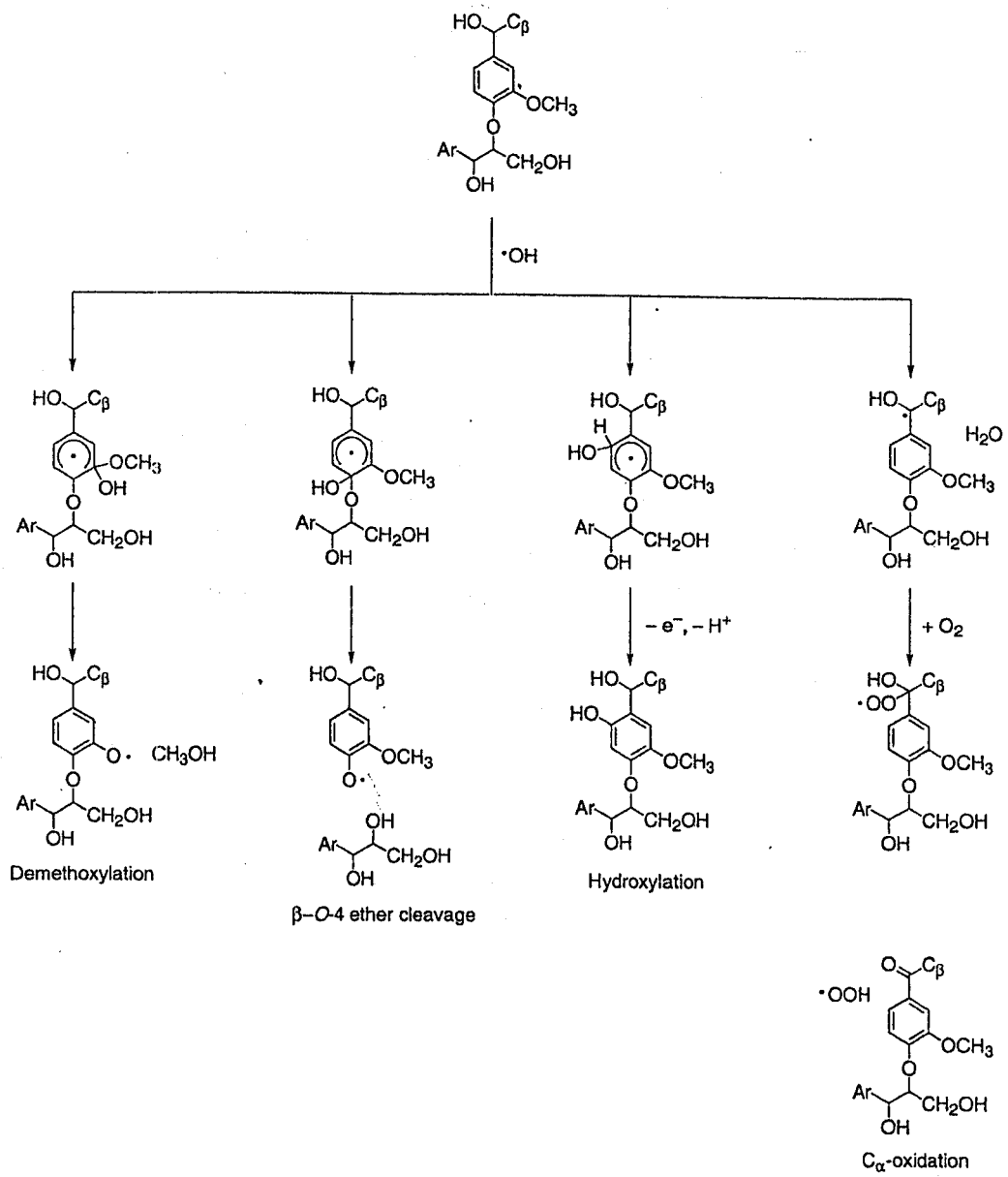

Fig. 2. Typical reactions of ·OH with the major arylglycerol-b-aryl ether structure of lignin.

stituent is attacked, methanol is eliminated and a lignin phenoxy radical is produced. Reduction of this intermediate, if it does not undergo polymerization first, results in a phenolic hydroxyl at the position of the original methoxyl [25]. An analogous reaction occurs when a ring position that carries the major b-*O*-4 ether linkage of lignin is attacked, but in this case the polymer is cleaved [26].

These studies show that the oxidation of lignin by ·OH results in diverse reactions, some of which are expected to degrade the polymer. However, it remains unclear whether any wood decay fungus uses ·OH to oxidize lignin. White rot fungi cleave lignin to products that are indicative of cation radical intermediates, but these oxidations are relatively selective, and lignin peroxidases or other oxidants rather than ·OH are probably responsible for them [9,27]. Studies of brown rotted lignin have shown that it has been demethoxylated, but there is no general agreement as to whether it has been cleaved or hydroxylated at unsubstituted positions [20,28,29]. As is the case with cellulose, the difficulties encountered with the analysis of brown rotted lignin leave it unclear whether ·OH is the species that oxidizes the polymer.

3. Peroxyl and hydroperoxyl radicals

If wood decay fungi produce ·OH, then it is also necessary to consider the effects that ROO· and ·OOH have on

lignocellulose, because both of these ROS are expected as secondary radicals when ·OH oxidizes wood polymers [30]. In addition, the manganese-dependent peroxidases of white rot fungi peroxidize unsaturated fatty acids, which results in the formation of ROS that include ROO· [31]. Recent work has clarified the mechanism of these reactions: The Mn^{3+} chelates produced by manganese peroxidase do not initiate lipid peroxidation by abstracting allylic hydrogens as some oxidants do, but rather abstract a methylene hydrogen from the carbon adjacent to the fatty acid's carboxyl group. The resulting resonance-stabilized acyl radicals then propagate lipid peroxidation and ROO· formation by abstracting allylic hydrogens from unsaturated fatty acids [32]. Further work is needed to determine whether this Mn-dependent mechanism contributes significantly to wood decay, but at present it seems reasonable because white rot fungi produce extracellular lipids that could serve as a source of peroxyl radicals [33,34].

Although ROO· arid ·OOH are less reactive than ·OH, they are strong oxidants [35], and may consequently attack lignocellulose. They abstract benzylic hydrogens, such as those at C_a of the lignin side chain, because resonance-stabilized carbon-centered radicals are formed as products [27]. It is likely that they can also abstract hydrogens from polysaccharides, because an adjacent hydroxyl or ether oxygen will stabilize the carbon-centered radical that is formed [36]. However, ROO· and ·OOH will not abstract hydrogens from unactivated carbons, and thus are more selective oxidants than ·OH is. Unlike ·OH, peroxyl radicals do not add readily to aromatic rings, although there is some evidence that they can abstract electrons from highly conjugated, polyaromatic hydrocarbons [37]. Peroxyl radicals can also abstract electrons from aromatic ethers such as those found in lignin, and this reaction results in ligninolytic reactions similar to those that lignin peroxidase catalyzes, However, these electron transfer reactions are slow unless the attacking peroxyl radical contains electron-withdrawing substituents [27].

4. Detection of ROS in fungal cultures

It is difficult to identify ·OH or other ROS unequivocally in any biologic sample, and fungi growing on wood present a particularly difficult problem. Because wood degradation is slow, it is seldom feasible to detect free radicals in fungal cultures by electron spin resonance spectroscopy, which would otherwise be the most diagnostic method [8]. A more straightforward approach is to add a molecular probe that undergoes characteristic reactions with ROS over an extended time, and then to look for the accumulation of diagnostic products in the cultures. However, although this approach is frequently used in many systems, it suffers two principal pitfalls. First, these probes are usually not very specific. For example, many "·OH detectors" are oxidized by species other than ·OH, sometimes to the same products that ·OH gives [8]. Second, most of the probes that have been used are small compounds that could enter cells, and consequently they cannot establish whether ROS production is extracellular as required for attack on lignocellulose [18].

4.1. Previous work arid research needs

These problems notwithstanding, there is evidence that decay fungi produce ROS when they grow on wood. Both brown rotters and white rotters oxidize dimethyl sulfoxide to methanesulfinic acid [12,38], which is fairly good evidence for ·OH production [39], although it is not clear whether the radicals are intracellular or extracellular. Wood decay basidiomycetes also hydroxylate phthalic hydrazide to give a chemiluminescent product that is characteristic of ·OH addition [10,13]. Here again, it is not certain that the responsible oxidants are extracellular, and further work is also needed to rule out the possibility that intracellular enzymes such as cytochrome P-450 might oxidize phthalic hydrazide. It has also been reported that products characteristic of ·OH attack are produced when brown rot fungi oxidize a variety of other low molecular weight organic chemicals, including chlorophenols [40], fluoroquinoline antibiotics [41,42], and the spin trap 5,5-dimethylpyrroline-*N*-oxide [43].

To make further progress in this area, it would be advantageous to have a high molecular weight target compound that cannot penetrate fungal hyphae, and that is oxidized to diagnostic products by ·OH. For example, it may be possible to design a simple polymeric aromatic compound whose rings are hydroxylated by ·OH. If the pattern of hydroxylation is unique to ·OH (as opposed to that caused by singlet oxygen or other oxidants), then the polymer could possibly be used as a specific detector of extracellular ·OH. Similar approaches that use salicylic acid, tryptophan, or other small aromatic probes have been used successfully to detect ·OH generation in intracellular systems [8,44].

4.2. Polyethylene glycol as a general probe for extracellular ROS

Although no specific probe for extracellular ·OH currently exists, we have been able to get some evidence that wood decay basidiomycetes produce extracellular ROS. Our approach was to monitor the degradation of another polymer, polyethylene glycol (PEG), by fungi growing on wood. PEG has several properties that make it a good detector of nonspecific, extracellular oxidants: First, ·OH oxidizes and cleaves PEG rapidly by abstracting its methylene hydrogens [18,45,46]. Second, PEG with a molecular weight greater than 1000 is unable to penetrate fungal cell walls [47,48]. Finally, PEG is nonhydrolyzable

Table 1
Mineralization of [^{14}C]PEG by decay fungi on wood

Fungus	Wood in culture	Temp (°C)	PEG mineralized (%)[a]			Wt loss in wood (%)[a]
			4 wk	8 wk	12 wk	12 wk
White rotters						
Ceriporiopsis subvermispora (FP-90031)	Red maple	30	20.8 ± 2.0	31.2 ± 3.0	42.2 ± 3.0	45.3 ± 3.1
Phanerochaete chrysosporium (ATCC 24725)	Red maple	30	9.5 ± 0.4	15.5 ± 0.7	22.6 ± 1.7	51.9 ± 7.0
Brown rotters						
Coniophora puteana (ATCC 12675)	Southern yellow pine	26	40.2 ± 7.0	45.7 ± 7.0	50.6 ± 8.8	10.3 ± 1.0
Gloeophyllum trabeum (ATCC 11539)	White spruce	28	76.8 ± 4.0	82.0 ± 3.4	83.8 ± 2.7	48.9 ± 22.5
Postia placenta (ATCC 44394)	Southern yellow pine	26	41.8 ± 12.5	54.9 ± 14.4	60.1 ± 15.0	24.2 ± 1.9
Soft rotters						
Daldinia concentrica (FP-140074-sp)	Red maple	26	0.0 ± 0.0	0.0 ± 0.0	0.0 ± 0.0	30.4 ± 3.4
Xylaria polymorpha (HHB-12598)	Red maple	26	0.1 ± 0.0	0.1 ± 0.0	0.1 ± 0.1	13.2 ± 0.8

[a] Means for six replicates ± SD. Each wood block contained 0.5 mg of *hydroxyethyl*-[^{14}C]PEG (Amersham, 1.0 × 10^4 dpm mg^{-1}) that had an initial molecular weight of 4000. No mineralization of PEG occurred in uninoculated cultures.

under physiological conditions and, with the possible exception of a few bacteria, no organisms produce extracellular enzymes to oxidize it [49]. However, a disadvantage of PEG must also be noted: it is not a specific probe for ·OH, because some weaker nonspecific oxidants also cleave it. For example, we have observed that PEG is cleaved rapidly by peroxyl radicals obtained from the thermolysis of 4,4´-azobis(4-cyanovaleric acid), and that it is cleaved slowly by peroxidizing linoleic acid (data not shown). Therefore, PEG degradation by a fungus serves only as a general indication that extracellular ROS are produced.

Table 1 shows how PEG can be used to detect extracellular, nonspecific oxidants that fungi produce when they grow on natural substrates. We monitored the mineralization of [^{14}C]PEG by three brown rot basidiomycetes, two white rot basidiomycetes, and two xylariaceous soft rot ascomycetes. The fungi were grown on sterile blocks of wood that had been infused beforehand with the radiolabeled polymer, using published methods [50]. $^{14}CO_2$ evolved from the PEG was trapped at 2-day intervals for 12 weeks [50], and the blocks were checked for dry weight loss at the end of the experiment to assess whether each fungus expressed its full degradative system.

The data (Table 1) show that all of the fungi degraded wood significantly under our culture conditions, and that all of the basidiomycetes degraded PEG when they were competent to degrade wood. 'This result is significant because it shows that the ability to produce extracellular ROS is widespread among wood decay basidiomycetes, irrespective of the decay type they cause. The data also illustrate the pitfalls of working with fungi in defined culture media, which may fail to elicit their full biodegradative systems. For example, *P. chrysosporium* clearly produces extracellular ROS on wood (Table 1), but is unable to degrade PEG in the standard glucose-based, low nitrogen medium that is widely used for physiological investigations of white-rot fungi [51, 52]. Finally, this experiment shows that the two soft rot ascomycetes we tested do not use ·OH to degrade wood polymers — tlfey did, the PEG we infiltrated into the wood would have been mineralized because its ethylene glycol subunit reacts with ·OH at approximately the same rate that the sugar subunits of polysaccharides react [24,45].

'5. Fungal mechanisms for production of Fenton reagent

Because ·OH is a likely participant in wood decay by fungi, it is useful to outline some of the properties that a fungal Fenton system would require to contribute optimally to this process. .OH production should occur at a distance from the fungal hyphae, and ideally within the lignified secondary cell wall rather than on its surface. To accomplish this, ·OH needs to be generated in situ by the reaction of low molecular weight fungal reductants that are stable enough to diffuse into the wood cell wall before they react. The function of these small fungal metabolites is to reduce Fe^{3+} to Fe^{2+}, or to reduce O_2 to H_2O_2.

5.1. ·OH from cellobiose dehydrogenase turnover

The cellobiose dehydrogenase-catalyzed reduction of Fe^{3+} provides a route for extracellular ·OH production that

meets most of the above requirements. Cellobiose dehydrogenase (CDH) is an extracellular enzyme produced by many white rot basidiomycetes, by brown rot basidiomycetes in the family *Coniophoraceae*, and by some soft rot ascomycetes [53]. This flavin- and heme-containing enzyme can act as a cellobiose oxidase, thus reducing O_2 and producing H_2O_2, but Fe^{3+} is a better electron acceptor than O_2, and as a result, CDHs can act as Fe^{3+} reductases [53,54]. In the presence of Fe^{3+} and O_2, the CDH of the white rotter *Phanerochaete chrysosporium* supports Fenton chemistry, as shown by the hydroxylation of salicylic acid [11]. This system also cleaves polysaccharides and synthetic lignin in reactions that do not require, but are stimulated by, exogenous H_2O_2 [55].

P.M. Wood and coworkers have pointed out additional features of the CDH system that may make it a good generator of Fenton reagent in wood. Many brown rot and white rot fungi secrete oxalic acid, which is a strong chelator of Fe^{3+} and Fe^{2+} [9]. If oxalate is the predominant chelator of iron in fungus-colonized wood, which seems probable (see below), then the redox properties of Fe-oxalate complexes will have a large influence on the Fenton Chemistry that occurs [16,56]. CDH can reduce Fe^{3+}-oxalate effectively only at pH values below approximately 2.5, because the reduction potential of the Fe^{3+}-trioxalate complex that predominates above this pH is too negative. However, if the region immediately adjacent to the fungal hyphae is sufficiently acidic, the more reducible Fe^{3+}-dioxalate complex will also be present. CDH reduces this complex to uncomplexed Fe^{2+} or to the Fe^{2+}-monooxalate complex. These Fe^{2+} complexes are relatively stable at pH 2.5, but as they diffuse away from the hyphae into a region with a lower oxalate concentration, they will encounter a higher pH, which will result in formation of the Fe^{2+}-dioxalate complex. Around pH 4, Fe^{2+}-dioxalate complexes autooxidize rapidly, owing to the electron-donating properties of the oxalate dianion, and as a result ·OOH is produced. This ·OOH is reduced by Fe^{2+} or dismutates, thus generating H_2O_2, the second ingredient needed for Fenton chemistry [16,56].

The attractive feature of this model is that the forination of a complete Fenton system is delayed until Fe^{2+} has diffused some distance from the fungal hyphae. In this way, Fenton reagent might be produced within the secondary wood cell wall where it is needed to initiate decay. In addition, this mechanism would protect the fungus from the oxidative damage that would occur if ·OH were produced immediately outside the hyphae. One criticism of the model is that it assumes H_2O_2 is produced only via Fe^{2+} autooxidation. If CDH also uses O_2 as an electron acceptor, which seems possible given that Fe^{3+}-oxalate complexes are relatively difficult to reduce, then H_2O_2 will be produced at the same site that Fe^{2+} is. In addition, in fungi such as *P. chrysosporium* that secrete H_2O_2-producing enzymes such as glyoxal oxidase [57], it appears unlikely that H_2O_2 production could depend entirely upon Fe^{2+} autooxidation. Another potential problem with this model is that it requires a pH below approximately 2.5 near the hyphae, even at early stages of decay. It is not clear whether these conditions generally occur, and if they do not, CDH will not reduce Fe^{3+}.

5.2. ·OH from quinone redox cycling

Quinone redox cycling has long been known as a source of ·OH [8], and could provide a mechanism for extracellular Fenton chemistry in wood decay fungi. The principle of this mechanism is that the fungus reduces an extracellular quinone to its hydroquinone, which then reacts with Fe^{3+} to give Fe^{2+} and a semiquinone radical. The semiquinone then reduces O_2 to give ·OOH and the original quinone. Because ·OOH is a source of H_2O_2, this cycle will generate a complete Fenton system [19].

This mechanism requires that the fungus have a mechanism to reduce extracellular quinones to their hydroquinone forms. Enzymes potentially capable of this reaction include intracellular benzoquinone reductases such as the enzyme from *P. chrysosporium* [58], provided the fungus can export the hydroquinones that are formed. Other possible quinone-reducing enzymes include extracellular sugar dehydrogenases such as CDH, which have been shown to use quinones as alternate electon acceptors [53]. The enzymes responsible have not been conclusively identified in any fungus, but it has been shown that the brown rotter *Gloeophyllum trabeum* and the white rotter *Pleurotus eryngii* both reduce extracellular quinones to give extracellular hydroquinones [19,59,60].

This model also assumes an adequate source of quinones. To some extent, these might be supplied by wood extractives or products of ligninolysis, as F. Guillen and coworkers have proposed for the P. eryngii system [59], but it must be considered that the ·OH generated by the quinone redox cycle will attack the quinones and hydroquinones in addition to wood polymers and other targets. Consequently, this system is most likely to operate effectively if the fungus provides its own source of extracellular quinones. Brown rot fungi in the genus *Gloeophyllum* fulfill this requirement: they secrete and reduce both 2,5-dimethoxy-1,4-benzoquinone and 4,5-dimethoxy- 1,2-benzoquinone [17,19,60, 61]. This system drives Fenton chemistry in *G. trabeum* cultures that are grown on defined glucose medium [17, 19].

An additional requirement is that the hydroquinones need to have sufficiently negative reduction potentials to reduce the chelates of' Fe^{3+} that predominate in fungus-colonized wood. Most brown rot fungi secrete oxalic acid, which binds Fe^{3+} strongly even at the acidic pH values found in sound and decaying wood [17,62–64].Therefore, it is a reasonable working hypothesis that fungal hydroqui-

nones need to be able to reduce Fe^{3+}-oxalate complexes if Fenton chemistry via quinone redox cycling is to occur. It has also been proposed that the catechols produced by some brown rot fungi act not only as Fe^{3+} reductants, but also as Fe^{3+} chelators [15]. At the low pH values associated with brown rotted wood, catechols are much weaker chelators of Fe^{3+} than oxalate is, but they may chelate it during the early stages of decay, when the wood is not highly acidic. It remains unclear how important the iron-chelating ability of catechols is during brown rot, because one fungal metabolite of this type, 4,5-dimethoxycatechol, is able to reduce Fe^{3+} rapidly even in the presence of oxalate at pH 4, under which conditions it is not expected to chelate Fe^{3+} significantly. Moreover, another fungal metabolite, 2,5-dimethoxyhydroquinone, also reduces Fe^{3+} effectively, even though it lacks catecholic functional groups [17].

A hydroquinone/Fe^{3+}-oxalate mechanism, like the CDH mechanism, provides a potential way for ·OH generation to be delayed until the reagents have diffused into the wood cell wall. Methoxyhydroquinones are not expected to have reduction potentials that are negative enough to reduce the Fe^{3+}-trioxalate complex rapidly, and it has been shown that high oxalate concentrations inhibit Fe^{3+} reduction by these species at pH 4. However, methoxyhydroquinones do reduce Fe^{3+} rapidly in dilute (approx. 1 mM) oxalate at pH 4, probably because the more reducible Fe^{3+}-dioxalate complex is present at this low oxalate concentration [17]. Because *Gloeophyllum* species accumulate only low levels of oxalate [62,64], this property of the methoxyhydroquinones may ensure that the quinone redox cycle occurs at a distance from the fungal hyphae.

A criticism of this model is that, if the pH in the immediate vicinity of the hyphae is much below a value of about 3, the more reducible: Fe^{3+}-dioxalate complex will be present, and quinone redox cycling could occur too near the hyphae. However, it is important to note that this reaction is thermodynamically disfavored at low pH, because protons are products of the reaction (hydroquinone $+Fe^{3+}$ ® semiquinone + Fe^{2+} +. H^{+}). Since a typical methoxyhydroquinone is expected to have a reduction potential around +200 mV [8], and since the reduction potential of the Fe^{3+}-dioxalate complex is similar at + 181 mV [16], acidic conditions are expected to inhibit redox cycling.

5.3. Other ·OH-generating systems

The two systems just outlined are not the only possible ones that wood decay fungi might use to produce ·OH. B. Goodell and coworkers support a model for *G. trabeum* that is similar to the quinone redox cycle described above, but they also propose that some of the extracellular phenolic compounds secreted by the fungus carry enough reducing equivalents that each of them can reduce multiple molecules of Fe^{3+} [15]. A. Enoki's group has obtained evidence that a wide variety of wood decay fungi produce extracellular Fe^{2+}-binding glycopeptides that could play a role in Fenton chemistry [12,23,38]. Our picture of fungal free radical decay mechanisms is by no means complete, and more work is needed to assess these possibilities.

Acknowledgments

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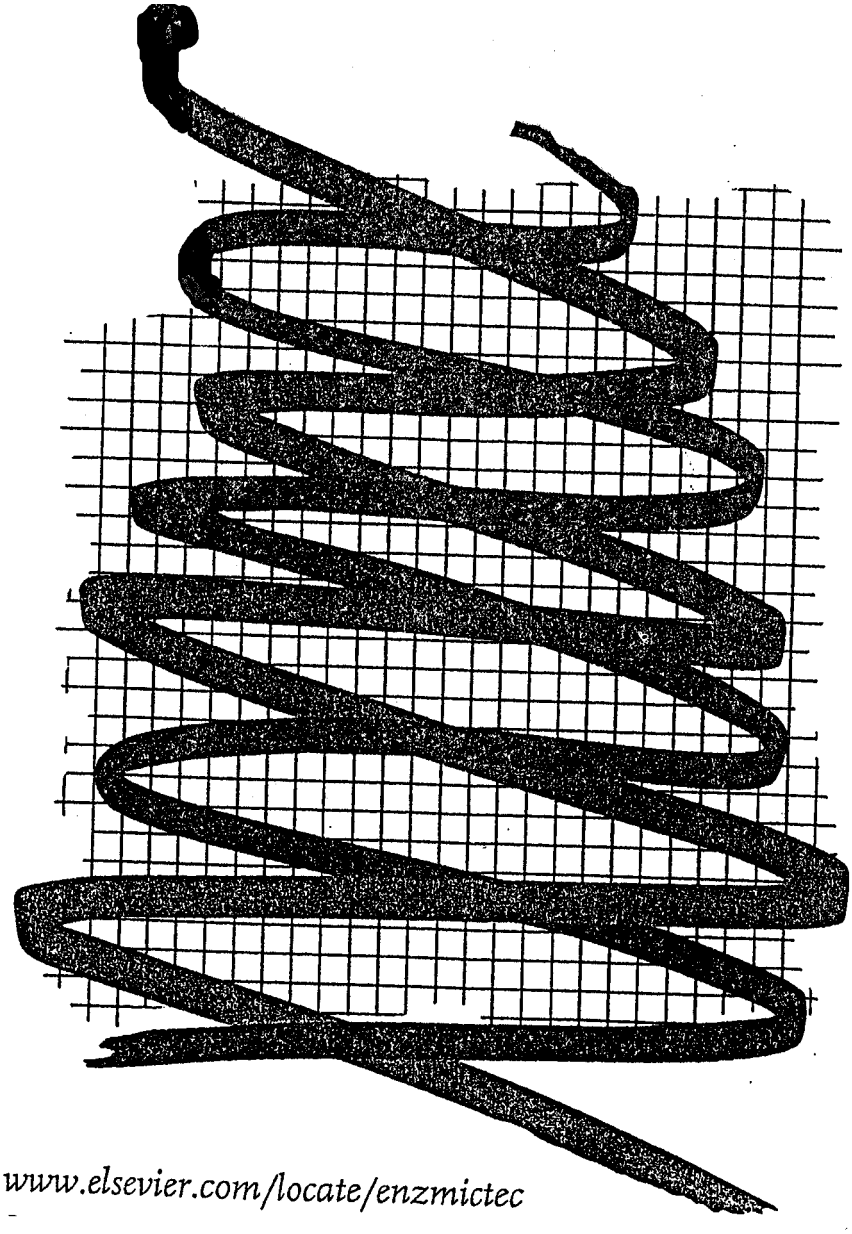

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