SYMPOSIUM

on Wood Decay in Living Trees. Mechanisms of Tree Defense and Wood Decay

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Stem Decay Perspectives—An Introduction to the Mechanisms of Tree Defense and Decay Patterns

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Forest pathology originated in the study of wood decay and this symposium reflects a continuing concern with the devastating stem decay losses in forest, park, and urban areas.

A century ago, Robert Hartig laid the foundation for forest pathology with classic studies of rust, foliage, stem, and root-rot diseases caused by fungi. Perhaps his greatest contribution was clarification of the role of hyphae in wood decay. Hartig's 1874 paper, recently translated by Merrill et al (14), is available as a Phytopathology Classic.

Early studies on decay emphasized etiology, the development of culling criteria and related volume losses, and the determination of pathologic rotations. A few highlights among these are: Wagener and Davidson's comprehensive monograph (28) on heartrot in living trees, Boyce's classic forest pathology textbook (2) that comprehensively covers stem decays, Roth and Sleeth's studies (19) on decay in sprout stands, and Hepting's research on fire scars and associated decay (8), and detailed review of stem decays grouped by major timber species (9).

An extensive literature on wood decay has accumulated in the past 100 yr. The 1956 Timber Resources Review conducted by the U.S. Forest Service (10) ranked heartrot the major loss factor in saw timber. With the shift from old growth to second generation stands for timber production, it was assumed that heartrot would become less important in the shorter rotations. Heartrot losses differ but are not necessarily reduced under shorter rotation management. In urban, park, and recreation sites, heartrot decay appears to be a major problem. Recently we studied a case of serious windthrow and decay that followed root rot caused by *Phaeolus* (*Polyporus*) *schweinitzii* in a stand of 30-yr-old Japanese larch in New York. A recent survey of street trees in Syracuse and Rochester, NY, revealed that approximately 25% of those >13-cm in diameter had fungal fruiting bodies or other external evidences of heartrot (27). Increased wounding during selective logging and coppice reproduction, particularly of hardwoods apparently also add to decay problems. Heartrot is, therefore, a continuing problem despite earlier optimistic predictions. The Review of Plant Pathology for 1977 includes abstracts of at least 65 papers on some aspect of heartrot.

Advances in enzymology, analytical detection of chemical compounds, isotope labeling, computers, data recorders, sensing devices, and electron microscopy provide new capabilities for the fundamental study of tree stem decay. Rapid progress has been made in the related field of the chemistry of wood products decay. Recent research includes biological delignification of pulp chips (5) and saccharification of wood wastes for alcohol production (16).

This symposium is a critical review of the development of decay in stems and the related host-defense reactions revealed by these new developments.

To put the topic in perspective, we will first highlight the heartrot disease cycle and, to minimize possible confusion, some terminology of the subject will be introduced. Finally, we will outline the presentations of this symposium.

Heartrot disease cycle. Concepts of wood decay are still being formulated and tested. Hartig's (14) original idea that infection occurs only through branch stubs and wounds that expose the heartwood is no longer fully accepted. Haddow (6) and Etheridge and Craig (4) showed that infection can occur through twigs no more than a few millimeters in diameter. Shigo and Marx also showed that many centrally located stem decays can be traced to earlier outer sapwood wounds. At the present time, infection courts in sapwood wounds are considered to be the most common points of entry.

Merrill (13) provides a good summary of the literature on spore germination and infection initiation by heartrot fungi and identifies some of the conflicting interpretation of the factors affecting spore germination and heartwood infection by fungi. This critical initial step in the disease cycle of stem decays still is poorly understood.

Much of the extensive research on invasion of stem tissues by decay fungi in recent years was stimulated by Shigo's (22) concepts of organism succession and compartmentalization developed to explain the patterns of decay observed in living trees.

The fundamental thesis of the successional concept should be kept in mind. Nonhymenomycetous pioneer organisms are postulated to precede decay fungi because they are isolated consistently from the discolored margins of decay columns, and wood decay fungi consistently are recovered only from decayed wood. Recently, Shortle and Cowling (26) demonstrated that the initial wood discoloration is induced by decay fungi in some timber species. What then is the fate of the decay fungus that induces the initial discoloration? Do the "pioneers" have an initial detoxifying role, and then after such a precondition period, does a dormant or new infection by other decay fungi become active? Further study of the successional concept of decay initiation is needed. The decay process may differ with tree and pathogen species. Some fungi may initiate decay directly and it will be particularly important to differentiate "true" heartrot decay systems of living trees from those of dying and dead trees.

The sporulation of various decay fungi has been investigated, but caution is advised in interpreting the results. Usually, sporulation studies are limited to monitoring a few fungal fruiting bodies at a given time. The senior author monitored the sporulation of three *Phellinus tremulae* (*Fomes igniarius* var. *populinus*) fruit bodies in the same stand for 2 yr. It became obvious that not all responded in the same way to the same environmental conditions. None of the fruit bodies studied in New York or Minnesota sporulated in the evening in response to decreasing temperature and increasing relative humidty as reported from Ontario, Canada, by Riley (18). Collections of stem sections with attached fruit bodies also seemed to respond differently to identical environmental conditions. The only possible conclusion is that the primary factors affecting sporulation still are not known.

The assumption has been that heartrot fungi are disseminated primarily by wind-dispersed basidiospores. A recent paper by Castello et al (3) implicated the bark beetle, *Dendroctonus pseudotsugae,* in dissemination of *Cryptoporus volvatus* and *Fomitopsis* (*Fomes*) *pinicola.* Also the possible importance of imperfect spores (eg conidia, oidia, and chlamydospores, which are common in Hymenomycetes) should not be overlooked in studies of dissemination.

The epidemiology of most stem-decay diseases is unknown. Recent work with *Armillariella* (*Armillaria*) *mellea* in ponderosa pine points to progressive vegetative spread continuing for centuries (21). In addition, data from that study show that the current stand originated in the presence of *A. mellea.* It has been proposed that *A. mellea* infections in roots may predispose them to subsequent infections by *Phaeolus* (*Polyporus*) *schweinitzii* (1).

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What factors influence decay intensification in stands? Hartig struggled with the same question and could only suggest the association of decay and intensive exposure to wounding.

Consideration of the role of genetic resistance of trees to heartrot is shifting from speculation to experimentation (12,24), and biological control of some stem decays by treating wounds with spore suspensions of *Trichoderma harzianum* has been demonstrated (17).

Terminology of stem decay. To minimize confusion that might develop from the use of different terms for similar phenomena, some of these should be explained.

The terms "heartwood" and "heartrot fungi" should be defined. Heartwood is formed by the aging and death of parenchymal cells in centrally located xylem. It develops centripetially and may or may not involve a pigmentation change. Accumulation of gas emboli in adjacent conductive cells and isolation of parenchyma cells has been proposed as the cause of death (29). Some trees such as oaks and pines develop visible heartwood early; in others, such as birch and maple, the process is delayed. Biochemical changes during cell death may allow heartwood to become impregnated with colored compounds, which may be fungistatic. Stem decay in the living tree is not limited to the dead heartwood. The heartwood of cypress, cedars, and black locust is very resistant to decay by saprobic fungi; in living trees of those species, however, heartwood is decayed readily by at least one heartrot fungus.

Heartrot fungi normally develop only in living trees. When the tree dies, they are replaced by decay fungi, which are better adapted to a saprobic system.

The terms "pathological heartwood" and "wound-initiated discoloration" are used to describe sapwood xylem tissues that superficially resemble heartwood. Tree wounds produce a series of responses leading to death of cells and infiltration with compounds similar to those found in true heartwood. Activities of microorganisms and the oxidative reactions associated with the exposed wound influence wound-initiated discoloration. The role of microorganisms in true heartwood formation appears to be minimal. In general, heartwood and wound-initiated heartwood may appear similar, but there is no reason to expect that specific decay-causing fungi recognize these two tissues as similar.

As previously indicated, the heartwood of some species is highly resistant to decay by saprobic decay fungi. This type of wood is described as "durable". Durability is a property of a wood product, not of a tree. No correlation exists between the durability of a wood product and resistance to decay in a living tree. When used as a wood product, the sapwood of most tree species is nondurable even though it was very resistant to decay in the living tree.

Two symposium participants use different terms to identify similar zones of the decay column. Shigo and Marx (23) proposed a useful system of numbers to identify the walls or barriers that limit the spread of decay microorganisms colonizing wounds in trees. Wall one is the upper and lower zone of the injured area in which wounding and invasion by microorganisms stimulates plugging of vascular elements. Wall two is an inner zone composed of summerwood cells with reduced pitting, a higher lignin content, and small lumens. Wall three is formed by the response reaction of the living radial cells. Walls one, two, and three restrict or slow the invasion of wood by fungi. Wall four is formed after wounding and is a barrier zone to outward expansion of the decay. Cells produced by the cambium in the vicinity of a wound are morphologically and chemically modified to prevent invasion by decay fungi. The total process of limiting pathogen invasion in tree stems was termed "compartmentalization."

Although these walls are described as four distinct planes of pathogen restraint, this is a conceptual model. At the functional level other groupings may develop.

Walls one, two, and three will be considered under a common mechanism by our speakers. The terms "transition zone" and "reaction zone" will be used by Shain (20); and "marginal zone" and "discoloration zone" will be used by Shortle (25).

The transition zone and marginal zone are light-colored tissues bordering the "normal" wood tissues that are responding chemically but are not in direct contact with the microorganisms of the discoloration and decay zone. Invading microorganisms and host chemical responses interact in the reaction or discoloration zone. Shortle (25) introduces the term "dark edge" to describe the zone of interaction between the barrier (wall four) and the discoloration zones.

Overview of the program. Hart and Shrimpton (7) will discuss stilbenes as inhibitors of wood decay. Natural durability of some woods is associated with this group of extractable compounds. It is logical to consider stilbenes among the many types of preformed chemicals that affect resistance to decay in trees even though they do not satisfy the criteria for an effective decay resistant compound when tested in wood.

Resistance to decay as a chemical response of the injured sapwood in living trees will be developed by Shain (20). Pinosylvin will be presented as a dynamic response compound. Ethylene production, moisture loss, starch degradation, and mineral accumulation all are parts of a necrotic, hypersensitive response of the tree to wounding and invasion by decay microorganisms.

Shortle (25) will develop the concept that wound-initiated wood discoloration is induced by the decay fungi, and that the activities of both decay- and nondecay-producing fungi are dependent upon the discoloration process during the transition from a living to a nonliving substrate. Successful invasion by both "pioneers" and decay fungi appear to be affected by factors associated with the defense levels of the tree, namely, vitality, preservative, and solubility factors. The vitality factor is associated with parenchyma cell responses, the preservatives factor is caused by phenolic impregnation of the discolored tissues surrounding the decay column, and the plugging of vascular elements and limited solubility of carbon and nitrogen sources reduces nutrient availability to invading microorganisms.

Whereas the first three presentations emphasize the tree and its response, Highley and Kirk (11) will describe certain unique characteristics of heartrot decay fungi. Many fungi utilize cellulose, fewer utilize wood composed of cellulose and lignin, and very few fungi decay wood in living trees. Their presentation does not minimize the role of other fungi and bacteria, sometimes referred to as non-Hymenomycetes or pioneer organisms, but rather focuses on the organisms responsible for degradation of the cell wall component. The roles of oxygen in cellulose degradation, simple sugar repressor effects on cellulase enzymes, nitrogen availability, temperature responses, acidity, carbon dioxide tolerance, and moisture on the unique aspects of heartrot decay fungi will be developed.

Merrill and Shigo (15) summarize the symposium with the topic "An Expanded Concept of Tree Decay." Future research may provide the foundation for significant reduction of decay losses. Previously forest pathologists' major practical contribution to resource management was loss assessment. By expanding tree-decay concepts, we may set the stage for exciting new approaches for minimizing decay losses in trees.

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