An Expanded Concept of Tree Decay

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CLASSICAL CONCEPT OF TREE DECAY

More than a century ago the concept of spontaneous generation of life, and with it the autogenetic theory of plant disease, was abandoned by most scientists. Anton DeBary established the germ theory of disease. Shortly thereafter, Robert Hartig ushered in the field of forest pathology by developing a concept of tree decay centered about three major points: fresh wounds, the infection of the freshly exposed heartwood by Hymenomycetes, and the subsequent decay of the infected wood. This concept of tree decay has persisted in textbooks virtually unchanged for nearly a century. Emphasis is upon the physical and chemical characteristics of the decayed wood and on the taxonomy of the associated Hymenomycetes. Until the 1950's, all attention was focused on the Hymenomycetes, in spite of the publication of scattered research papers indicating that not only were other fungi associated with the Hymenomycetes, but also that some of those alone could cause decay in (or at least alter) wood.

In living trees, the decayed wood has been considered to be primarily heartwood, and heartwood has been considered to be the dead, nonresponsive central core of the living tree. Heartwood has been considered to be any wood darker in color than the sapwood. Many variations of heartwood have been described: wound heartwood, false heartwood, precocious heartwood, and so forth. The decay of heartwood in living trees has been termed "heart-rot," and completing the circle, "heart-rot" in turn has been defined as the decay of the heartwood, the dead, nonresponsive central core of the tree.

Many pathologists have not considered tree decay to be a disease. Because such decay was considered to be confined to the dead, nonresponsive central core, they considered the fungi involved to be saprophytes rather than parasites. They had overlooked the essential point that parasitism is defined on the basis of interactions at the organism level and, further, that there are living cells in "heartwood." Tree decay is a disease and the associated fungi are both pathogenic and parasitic.

Cavities within trees were described as the final result of complete digestion of the heartwood. The progression of decay in a longitudinal or vertical direction was considered to be similar to processes occurring in a horizontal or cross-sectional direction. Initial infection was described as germination of spores of Hymenomycetes on freshly exposed heartwood even though spores of many such fungi never have been observed to germinate on any substrate. Details on many of these points are in Chapter 16 of the textbook, Forest Pathology, by Boyce (1).

DEFICIENCIES OF THE CONCEPT

The results of studies published during the past two decades have revealed several obvious deficiencies in the classical concept of tree decay. The classical concept does not consider: the response of the living tree to wounding and to invasion by microorganisms, the bacteria and nonhymenomycetous fungi that infect tree wounds, that many tree species lack a true heartwood, and that even in true heartwood injured and infected tissue often is walled off. Thus, the classical concept must be expanded and modified.

First, it is necessary to consider how a tree is constructed and how it is pre-set chemically and anatomically to survive or react after injury to the xylem. Trees do not heal wounds that extend into the xylem; the word *heal* denotes to repair, to replace, or to restore injured and infected tissues to a previous healthy state. Trees cannot do this. Instead, a tree is a highly compartmented organism. These compartments are delimited by the last cells which form in the annual rings and the rays. After injury, the tree's defense system reacts anatomically and chemically to isolate, wall off, or compartmentalize the infecting microorganisms and damaged tissue so that the fewest compartments are affected. Compartmentalization has great survival value for a long-lived tree.

Many types of microorganisms are involved in the processes that lead to discolored and decayed wood. In some cases, the Hymenomycetes are the first organisms to interact with the tree. But in most cases the freshly-exposed injured tissues are rapidly colonized by bacteria, yeasts, and many types of nonhymenomycetous fungi. The first microorganisms to penetrate into the tree alter both the sapwood and heartwood.

After a tree is wounded it reacts by forming inhibitory compounds in the tissues surrounding the wound. These inhibitory compounds limit the infection caused by most microorganisms. Nevertheless, if the microorganisms are aggressive enough, or if environmental conditions favor the microorganisms, infection will occur. There is intense competition among microorganisms for the available simple nutrients in the tree. Some pioneer microorganisms alter the wood in such a way that they stimulate spore germination of and infection by the Hymenomycetes. Other microorganisms alter the wood in such a way that they slow the rate of infection by Hymenomycetes. Regardless of the nature of the processes, succession does occur.

Many conditions affect the nature of succession: type of wound, position of wound, time of year the wound occurred, and the size and depth of the wound. Successions are complex events, and it will take time and patience to unravel their intricacies.

CURRENT RESEARCH

The papers in this symposium focused on four major points: the unique internal environment of the wood in the living tree, the dynamic response of a tree to infection, the inhibitory materials that affect resistance to infection by the Hymenomycetes, and the metabolic shunts that occur after wounding and infection.

Hart and Shrimpton (4) touched upon three critical points. First, that in studies published to date seldom have the rules of proof been followed that are necessary to establish a cause and effect relationship between a particular chemical extractive and the decay resistance of wood. Second, that we must know more about where wood extractives are localized in the cell walls and lumina, and how these extractives are bound to other wood constituents. Evidence suggests that these chemicals are not bound to cellulose but may be bound to lignin. Are they bound to hemicelluloses or other extractives? Third, that decay resistance probably is multifunctional. Most studies to date have concentrated on a single compound or class of compounds, and have ignored the multitude of other extractives present in the wood. Interactions among

extractives are highly probable and this will be a difficult subject to unravel.

In the sequence of events leading to discoloration and decay of living trees, the initial reaction is an autonomous response of the tree. An understanding of these plant responses is prerequisite to understanding the rest of the discoloration-decay sequence. These initial responses may condition the rest of the entire sequence. Mullick (7) recently postulated that blockage or cessation of xylem transport is one of the first reactions of wounded xylem tissues. Perhaps it is this response which leads to the formation of transition and reaction zones and the changes in moisture levels as discussed by Shain (8). Mullick (7) stressed the lack of conclusive proof at the present time. This is a subject worthy of critical study.

There are seven other points which we wish to raise. First, most of the research to date, as summarized in three of the four previous papers of this symposium, has been done in the laboratory. There is grave danger in extrapolating the results of a limited petri dish study to the complex sequences of events involved in the processes of discoloration and decay of wood in nature. Such in vitro studies must be done; nevertheless, it must always be foremost in our minds that the results of such studies may *never* relate to nature and that they may be only laboratory artifacts!

Second, decay fungi *never* occur alone, either in living trees, in slash, or in wood in use. Wood decay is the final result of a sequence of events. Studying those organisms or processes at the *end* of this sequence will not lead to an understanding of the total process.

In living trees, the microorganisms associated with the decay fungi affect pH; the zones in which they occur may be quite alkaline. They may be associated with an accumulation of minerals; some of these minerals may affect the metabolism of the organisms appearing later in the succession. The metabolic activities of the associated microorganisms undoubtedly affect CO_2-O_2 relations in the living tree; this may not occur or may occur to a lesser extent in dead trees or cut lumber. The associated microorganisms may secrete antibiotics or stimulants, they may detoxify or consume extractives, or may modify the wood constituents.

Third, successions are a complex series of events. We may have to do thousands of studies and examine thousands of trees to detect the *patterns* that exist. An understanding of the general nature of these patterns may be as close as we ever come to unraveling the intricacies involved. It may never be possible to duplicate these patterns in vitro, or even to create them at will in vivo.

Fourth, in most studies to date, including those of Hart and Shrimpton (4) and Highley and Kirk (5), the decay organisms used have been the slash-rotting fungi—those which decay litter and woody debris. Although such studies may relate to wood in use, they probably do not relate at all to decay in living trees. The true heartrotting fungi have been little used in such studies, probably because they cause very small amounts of decay in vitro. A further point of concern is the extrapolation from information about a fungus which is primarily a root-rotting fungus to those fungi which rarely if ever occur in the roots. Such extrapolations would be more acceptable if it could be demonstrated that *Fomes pini* elicits the same responses as *F. annosus* under similar conditions.

Fifth, most existing data on tree decay must be examined with great skepticism. The problem lies in the characterization of the tissues being studied. Prior to the relatively recent studies by Shigo and his co-workers, all dark-colored wood was considered to be heartwood. Much of this wood actually was discolored due to the autonomous responses of the tree to wounding and the invasion and subsequent activities of various pioneer microorganisms. We now know that heartwood and discolored wood are not the same, and do not react the same. Thus, there is no way to interpret the results of earlier studies.

Sixth, it has long been stated that the true heartrotting fungi invade through dead branch stubs, and this seems to be true in hardwoods. In conifers, however, stubs of branches which have died naturally often are the most decay resistant portions of the wood. Further, Etheridge and Craig (3) showed that *Echinodontium tinctorium,* a true heartrotting fungus, invaded only through small living branches. Other heartrotting fungi in conifers may behave similarly, and it also may be true for certain fungi occurring in hardwoods. For example, Etheridge (2) was unable to find *Fomes igniarius* growing inwards through dead branches of aspen. If the fungus occurred in a dead branch, it always occurred as an outward growth from an internal column of decayed wood.

Even if some fungi do enter living trees through branch stubs, at the present time it seems unwarranted to assume that all of the cells in the branch stub are dead and that the tissues are incapable of reacting to this invasion. For example, Shigo and Shortle (9) recently showed that even so-called dead, unresponsive true heartwood of oak can, and does, react to invasion by the fungi involved in the discoloration-decay syndrome.

Seventh, many techniques (particularly inoculations) used in in vivo studies have been inadequate. In retrospect, some of these are ludicrous.

The most commonly used technique has been to insert a large wooden dowel overgrown by a test fungus into a deep hole in a tree. Such an inoculum load is probably a billion-fold greater than any occurring in nature, even if one ignores the fact that the natural inoculum probably consists of basidiospores! Further, the mycelium was placed in direct contact with freshly injured xylem tissues. To add insult to injury, in many studies oat seeds or similar nutrients were added to the wound to give the inoculum an even greater boost. The only information obtainable from such studies is a description of what happens when trees are inoculated in such a manner. All such data are artifacts, which have no relationship to the events that occur in nature. Further, when isolations were made from such trees, all nonhymenomycetous organisms were considered to be merely contaminants. Over a century after Robert Hartig showed that Hymenomycetes were involved in tree decay, still virtually nothing is known about the germination of hymenomycetous spores in various infection courts, the initial penetration into the suscept plant, or the subsequent colonization of the host (6).

A LOOK TO THE FUTURE

Clarification of the decay processes to include compartmentalization and succession provides new opportunities to regulate tree decay. A tree is constructed in an orderly manner, and when it is injured and infected, it responds in an orderly way (compartmentalization). When microorganisms infect trees, they do so in an orderly manner (succession). The more completely the nature of compartmentalization and succession are understood, the better are our chances for regulating the processes of tree decay.

We can now select individual trees within a species that compartmentalize discolored and decayed wood to very small volumes. Viewed in this way it is not so important whether the tree can be infected, but that the infection has been walled-off or compartmentalized to a very small volume within the tree. In this sense, it is now possible to select decay-resistant trees.

This compartmentalization effect also appears to function in tree roots. This may explain why some trees live for many years following multiple root infections, whereas some trees die very quickly after becoming infected.

The anatomy of trees in some species may favor a higher degree of compartmentalization than that of trees of other species. This may help to explain why some trees, such as species of *Populus* and *Betula,* have very long columnar defects, whereas species of *Acer,* especially *A. saccharum,* have very short columnar defects. With detailed anatomical information it may become possible to develop schemes that will outline the limits for defect potential in different tree species. It also would aid geneticists selecting tree breeding stock for reduced defect potential.

By understanding the successional patterns of colonizing microorganisms, it may be possible to utilize biological control agents that could stall or stimulate the decay process.

In summary, we must now consider the living tree, and how it compartmentalizes injuries and infections. We also must consider the impact of many microorganisms operating in successional patterns in living trees. Better regulation of the decay process depends upon a clearer understanding of this expanded concept of tree decay.

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