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Wood Decay in Living and Dead Trees: A Pictorial Overview

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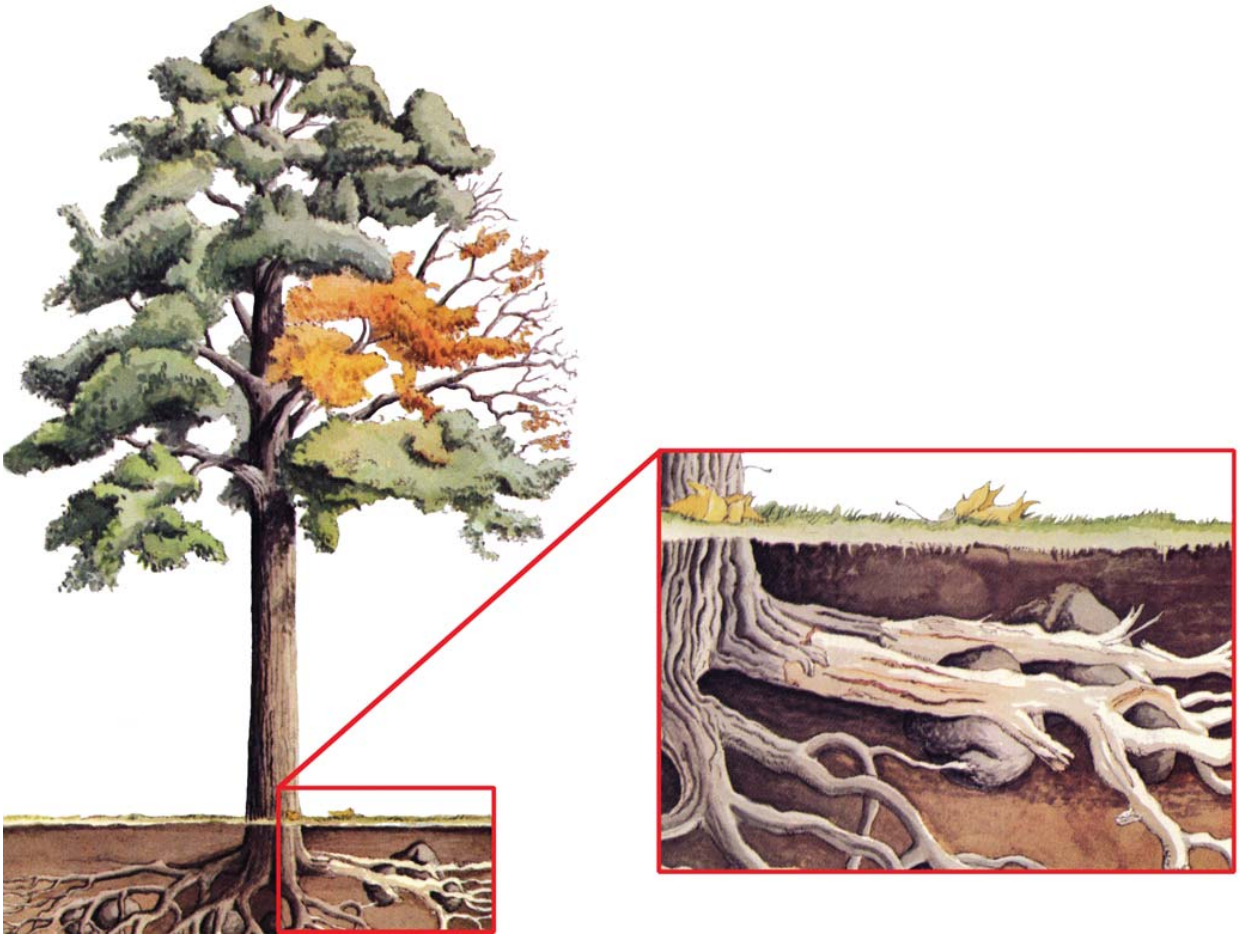
Kenneth R. Dudzik



Abstract

Pioneering research by Alex L. Shigo and his associates has produced a series of pictorial guidelines to provide a better understanding of how trees respond to wounding and subsequent microbial infections that lead to wood decay. The purpose of this paper is to visually summarize through the use of 96 color photographs and illustrations, the varied patterns of wood discoloration and decay observed in the dissection of thousands of trees. This information has served as a conceptual framework for understanding the biochemical processes that limit the spread of wood-destroying infections initiated by wounding during the maturation of all trees. This understanding has helped those who work with forests, trees, and wood to resolve practical problems and improve the health and productivity of trees and forests.

Cover

Watercolor illustration by David M. Carroll, Warner, New Hampshire.

Photos

All photos are by the U.S. Forest Service.

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INTRODUCTION

Pioneering research by the U.S. Forest Service has led to a series of pictorial guidelines for assessing the impact of wood decay in living trees. The series begins with a photo guide containing 100 color photographs (Shigo and Larson 1969) followed by three booklets using color illustrations to expand on concepts derived from years of tree dissections presented in the photo guide (Shigo 1976, 1979; Shigo and Marx 1977). The basic information presented in the booklets of how trees respond to wounding and subsequent microbial infections was applied to tree care and forestry and became the basis of much scientific research (Shigo 1984). Further expansion of Alex Shigo's concepts and their application to practical problems has improved the health and productivity of trees and forests, and these concepts have been published in several books (Shigo 1986a, 1986b, 1991). This basic approach of using color photographs and illustrations continues to promote a better understanding of tree response to wounding and wound-initiated infections causing wood to decay in living trees (Smith and Shortle 1998, Smith et al. 2001, Shortle et al. 2003).

The purpose of this paper is to create a visual summary (using color photographs and illustrations) of the varied patterns of wood discoloration and decay observed in the dissection of thousands of trees. This information has helped those who work with forests, trees, and wood interpret patterns of decay in living trees (Smith 2006). A better understanding of the CODIT (Compartmentalization Of Decay In Trees) model and the concept of compartmentalization (Shortle 2000) in all tree species worldwide provides a basis for better practices to improve tree survival, health, and productivity. For more detailed information and further illustration, consult the cited references.

The Authors

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WOOD DECAY

Wood decay is the biological process by which cellulose and lignin, the two most abundant organic compounds on Earth, are converted to carbon dioxide and water with a release of energy to maintain forest processes. Wood decay cycles essential nutrients needed for the regeneration of small trees to replace dead, decaying trees (Fig. 1a). Wood decay in living trees provides a substrate in which insects are active and available as food for wildlife, and it creates cavities to shelter wildlife (Fig. 1b, sapsucker nest in decayed aspen). Wood decay in living trees is a major disease causing loss of wood products and product quality (Fig. 1c). Wood decay damages wood in service (Fig. 1d, utility pole) and creates hazards for people and property (Fig. 1e). The causal agents of wood decay are macrofungi often seen fruiting on living trees in the forest that have basal logging scars (Fig. 1f), at home-sites (Fig. 1g), and in public places (Fig. 1h).

The starting point for wood decay in living trees is a wound. In this illustration of a generic hardwood, we see the two basic types of wounds, the stub (Fig. 1i1) and the scar (Fig. 1i2). Stubs occur when branches and tops are broken or branches die from growth suppression or disease (stubs also occur in roots for the same reasons). The stub protruding from the stem delays wound closure and allows the decay process to proceed for many years. Scars, particularly at the base of the tree, are caused by removal or death of bark. Exposed wood surfaces crack open as the wood dries and insects then bore into the exposed wood. The time needed to close the wound depends on the size of the scar and the growth rate of the tree.



Figure 1.

AGENTS OF WOUNDING

Birds (of the Picidae family) such as sapsuckers make many shallow wounds (Fig. 2a) that deform stems (Fig. 2b, birch) and cause ring shake (Fig. 2c, hemlock). Insects such as sugar maple borers (Fig. 2d) make deeper wounds that deform and weaken stems (Fig. 2e) and initiate internal discoloration and decay (Fig. 2f). Bark beetles attack weakened trees and initiate a layer of dead and discolored wood (Fig. 2g, arrow) that will not provide protection if the tree is subsequently wounded and infected by a wood-decay pathogen. Larger animals such as bear, deer, beaver, etc. remove bark and expose wood to infection (Fig. 2h, feeding by bears on western hemlock bark created a basal scar, arrow, from which a major column of decaying wood developed over the next few decades). Decay begins in both live and dying sapwood and spreads into dead heartwood. Wood decay always ends up in the heart of living trees, not because wood-rotting fungi prefer heartwood, but because the tree grows and adds new wood while live sapwood compartmentalizes infected wood by active protective processes (Fig. 2i). The old concept of heartrot, the decay of dead heartwood by heartwood-rotting fungi, has been replaced by a more dynamic concept of discoloration and decay in living trees based on research conducted over the past 40 years. The concept of compartmentalization of decay in trees has been developed in which the living tree and the wood-rot pathogen play an active part.



Figure 2.

Storm injury causing broken tops and branches leads to the slow progress of decay in exposed wood (Fig. 3a, arrow, broken top in sugar maple). Fire was a major cause of tree injury in the eastern U.S. in the late 19th and early 20th centuries and continues to be a major cause of injury in the West (Fig. 3b). Harvesting with heavy equipment became a major cause of injury in the 20th century (Fig. 3c). Trees differ in response to infection initiated by wounds of equal size due in part to genetic variability among trees (Fig. 3d, columns of discolored and decaying wood initiated by logging scars on two different sugar maples show the wide variation in response resulting in a large column, left; or a small one, right). Root damage resulting from breakage, compaction (cuts off oxygen), or change in water (too little or too much) is a common starting place for wood decay belowground, especially in urban areas (Fig. 3e). Dying (browning) or dead branches in the crown of a tree (Fig. 3e) often indicate the loss of some functional roots belowground. Trees are often wounded in high use areas (Fig. 3f, bark peeled without considering the adverse effects of wounding). This type of damage has been called People Pressure Disease. Many people do not realize that under the outer dead bark is living inner bark, living outer wood, and the cambial layer in between, which produces new wood and bark each year. When trees are wounded by human activity, microbial infection begins and tree health declines.

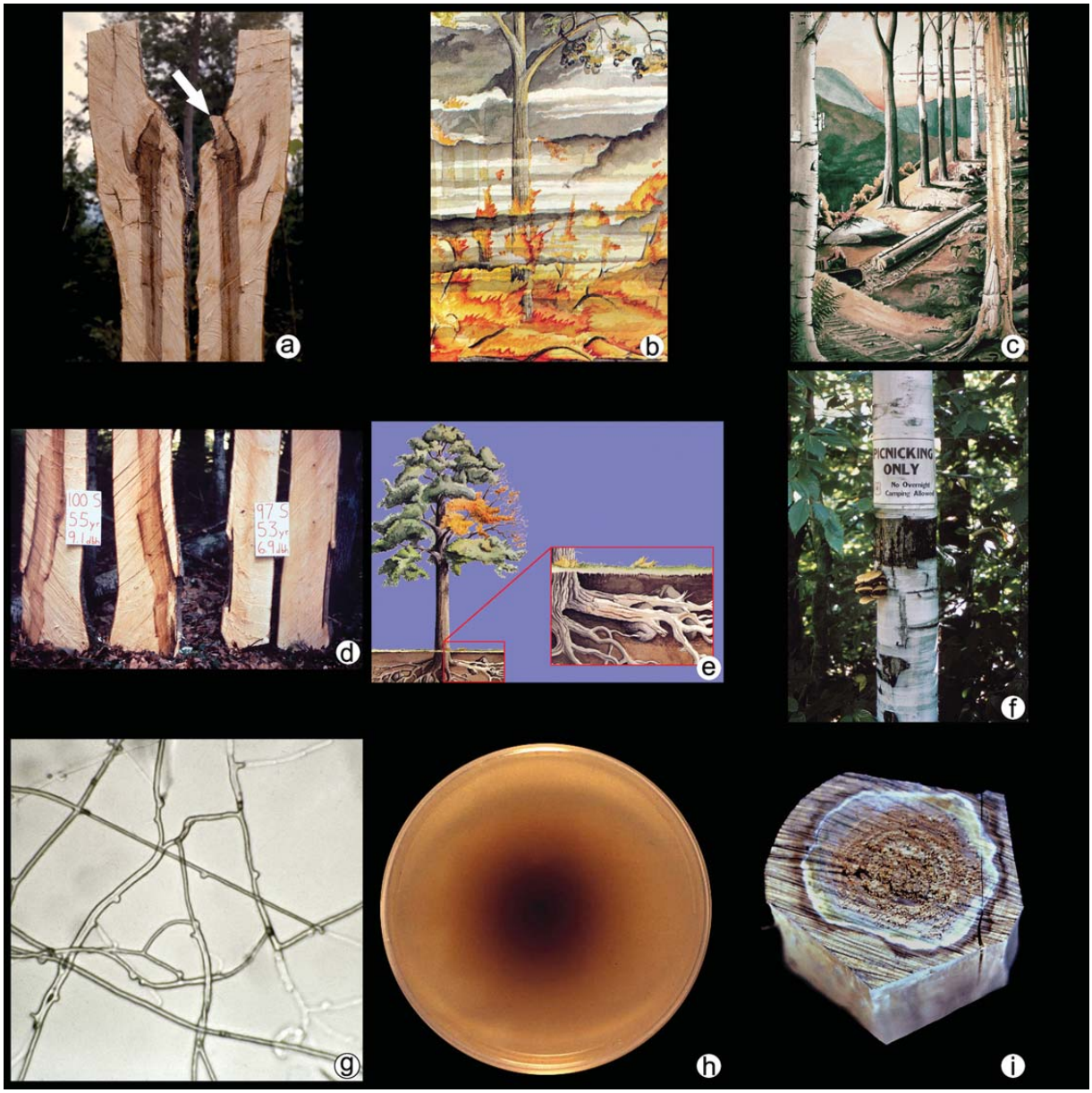

Figure 3.

AGENTS OF INFECTION

Wood-decay fungi spread through wood as microscopic strands called hyphae, which begin as either germinating spores or bits of hyphae (Fig. 3g) carried to the wound by insects. These strands of wood-decay fungi often secrete oxidizing enzymes that discolor wood and reduce its resistance to decay (Fig. 3h, plate of gallic acid medium inoculated with a wood-rot pathogen growing in the dark area in the center of the plate and secreting oxidizing enzymes that discolor the substrate). As a wood-decay fungus grows and develops in wood, its microscopic strands come together to form a mycelium, visible as a white ring on the surface of an infected wood disk after several days of incubation within a plastic bag (Fig. 3i). In nature, given the right conditions of temperature, humidity, and light, the fruiting bodies we see on trees and dead wood arise from this mycelium.

Looking at a cross-section of red maple 2 years after inoculation with several fungi - some pathogenic, some not - we see differences in the response of live sapwood and in the formation of decayed wood (Fig. 4a). One of the inoculated wood-decay fungi (see arrow) is a maple pathogen and is now decaying wood after passing quickly through the initial discoloration phase in which pioneer fungi and bacteria developed along with the wood-decay pathogen. Other wood-decay fungi that are less pathogenic may eventually produce visibly decayed wood if the wound remains open, allowing aeration to occur (to be active, wood-decay fungi need higher oxygen levels than associated pioneer fungi and bacteria). As the wound closes, there will eventually be insufficient aeration for the wood-decay fungi, potentially allowing the pioneer fungi and bacteria to remain active in the discolored wood.

Pioneer fungi associated with the wood-decay fungi during the initial discoloration phase have small spores. They may be present in healthy sapwood, but not active until wood is exposed by wounding (called “latent infection”). They may also be carried to the wound by insects, or they may grow into the wood from the bark. Some are dark and stain the wood; some are not (Fig. 4b). Some are pathogenic and spread into living sapwood; others are not and become active only after living cells of wood have died.

If the oxygen content of wood is relatively high, these fungi grow as mycelium and produce spores in the infected wood (Figs. 4c, d). If the oxygen content is low, the small spores bud to form colonies that look like bacterial colonies, but mycelium is not produced. Under low oxygen conditions, bacteria (Fig. 4e) flourish in wood producing a condition called wetwood. This condition may persist for a long time and inhibit the decay process, but once the wood dries and becomes oxygenated, the wood-decay fungi take over and the wood decomposes. It is possible for wood to become anaerobic (no oxygen), and only specialized anaerobic bacteria survive (Fig. 4f, pustule of *Clostridium* in oak).

Experiments in which a wood-decay pathogen is inoculated onto living or dead sweetgum sapwood (since repeated using other tree species) demonstrate the interaction of live tree cells with spreading mycelium (Fig. 4g). On the left, the network of living cells in sapwood has been killed and the mycelium is well developed after 21 days. On the right, living sapwood cells are interacting with the pathogen by producing protective substances and enzymes while the pathogen produces its own substances and enzymes causing the wood to discolor. Wood

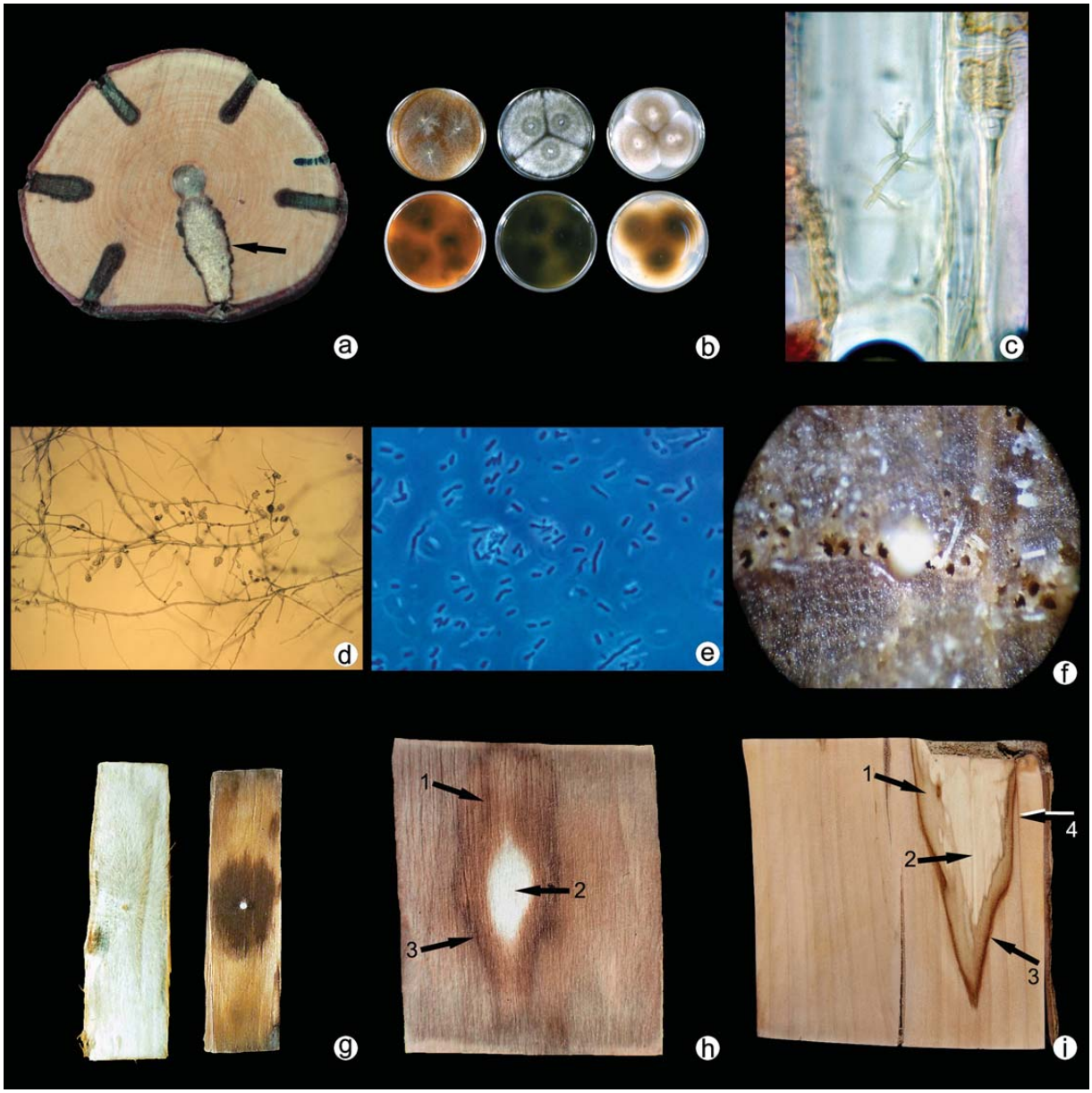

Figure 4.

discolors before decay occurs. The discoloration process slows the decay process, giving the tree's vascular cambium time to grow and add new wood. At the boundary between infected discolored wood and sapwood, a dark margin appears (Fig. 4h3). This reaction zone retards the spread of the decay-causing pathogen. Using live sapwood in the lab, we were able to reproduce all the stages of tree decay: (1) wood discoloration (Fig. 4h1), (2) decolorized decaying wood in center surrounded by discolored infected wood (Fig. 4h2), and (3) a darkly colored reaction zone at the margin between infected and sound sapwood (Fig. 4h3). The production of this marginal zone resulting from the interaction of the wood-decay pathogen and live cells of sapwood is called compartmentalization. If this protective layer that resists the spread of infection occurs in sapwood present at the time of wounding, it is called a reaction zone. If the protective layer forms in altered sapwood formed after wounding, it is called a barrier zone. Chemically the reaction zone and the barrier zone are the same, but the anatomical structure of the barrier zone is different, which makes it more resistant to the spread of infection. Columns that developed after a boring wound in sweetgum (Fig. 4i) show all the same stages of decay (Figs. 4i1, 4i2, 4i3). Reaction zones formed in sapwood present at the time of wounding and barrier zones formed in new wood after wounding to localize the stem disease. The barrier zone (Fig. 4i4) connects to the wound-wood ribs as trees continue to grow and close the wound. Closure stops the aeration process needed for active decay.

DEVELOPMENT OF COLUMNS OF DISCOLORED AND DECAYING WOOD

[Note on tree species illustrated: Sweetgum (Figs. 5a-c), oak (Figs. 5d-g), maple (Figs. 5h-6b), and birch (Fig. 6c). Common features include (1) discoloration expands before decay, (2) the spreading infection is compartmentalized by live sapwood, and (3) the decay process ends in a hollow tree because compartmentalization allowed the tree to continue to produce sound wood and bark on the outside while old wood decayed on the inside.]

Figure 5a shows a young sweetgum, all sapwood, with a 2-year-old scar wound and a thin layer of discolored wood in which we see the inward projections of discolored wood due to drying cracks and insect bore-holes. After 10-15 years, a sweetgum with a similar wound shows about half of the exposed section of trunk decayed within the limits of protective layers, the reaction zone (Fig. 5b3) derived from normal sapwood and the barrier zone (Fig. 5b4) derived from newly formed anomalous sapwood. Figure 5c shows a sweetgum 30-50 years after wounding in which a hollow has developed as all sapwood exposed by wounding has decomposed and several inches of sound, healthy wood has formed beyond the protective barrier zone allowing the tree to survive.

Figure 5d shows an oak 10-15 years after wounding with a column of discolored and decayed wood well developed on the wounded face. Figure 5e shows an oak 30-50 years after wounding. The tree became hollow after all the exposed sapwood and heartwood decomposed and several inches of sound healthy sapwood and heartwood formed beyond the barrier zone. The outer limit of decay is the position of the cambium at the time of wounding (Fig. 5e, arrow). The cambium formed a barrier zone to compartmentalize the infection and wound-wood to close the wound.

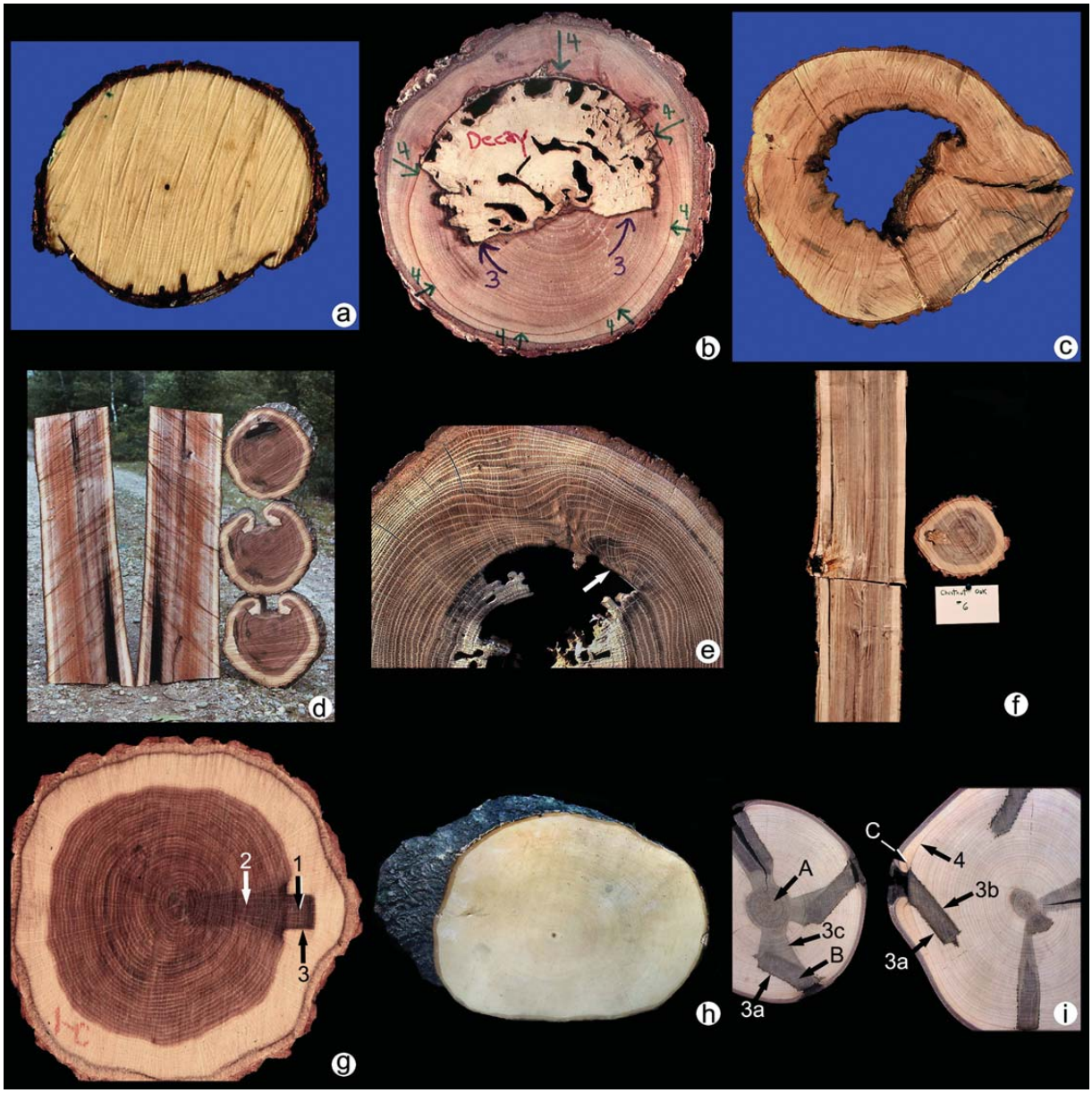

Figure 5.

Five years after an experimental wound in oak was inoculated with a wood-decay fungus, the sapwood became discolored, the discolored wood became infected and decayed before decay developed in discolored heartwood (Fig. 5f). The spread of the wood-decay fungus from discolored sapwood (Fig. 5g1) into live healthy sapwood is limited by the formation of a reaction zone (Fig. 5g3) in response to activity of the wood pathogen. This response is compartmentalization. The advance of the wood pathogen from discolored sapwood into discolored heartwood (Fig. 5g2) is limited by wood-preserving substances and the supply of oxygen coming from the open wound. This is decay resistance, not compartmentalization. Rapid closure of the wound stops infection at the discoloration stage. Under low oxygen conditions, bacterial wetwood may occur or possibly a slow type of decay called "soft-rot."

Sugar maples can grow for 100 years or more forming only sapwood, although the symplast gradually dies in aging wood (Fig. 5h). Wounding initiates wood discoloration (Fig. 5i, red maple). The central column of discolored wood (Fig. 5iA) is related to loss of branches and is in the pre-infection stage in which cells die and discolor, but the wood is not yet infected. Columns of discolored wood initiated by bore-hole wounds (Fig. 5iB) are infected with wood-decay fungi and their pioneer associates. Note the dark reaction zones that form between the infected wood and live sapwood (Fig. 5i3a). This protective response is strongest where wood rays connect to phloem (live inner bark) on the outer, bark-facing side of the columns (Fig. 5i3a, outer reaction zone). The response is weaker on the inner side of the column (Fig. 5i3b) or may not occur at all (Fig.5i3c). The live cells of the inner side have only stored food for the production of protective substances. On the outer side they have this stored food plus additional food supplied by the phloem. Fungi spread more easily through the weak inner layer than the strong outer layers. Look carefully inside the wound-wood rib (Fig. 5iC) to see the layer of wood formed after wounding by the cambium between rings 2 and 3 (Fig. 5i4). This layer has more live cells than normal sapwood present at the time of wounding and lacks long vessels for sap conduction. This layer will become the barrier zone when infection reaches it and triggers the formation of a zone even stronger than the reaction zone that forms in normal sapwood.

Figure 6a shows a well compartmentalized column of discolored and decaying wood 5 years after a bore-hole wound to a live healthy sugar maple. Wound closure is not complete and decay is taking place but is localized to one side of the tree. Figure 6b shows an over-tapped sugar maple with columns from many wounds coalescing into a central column of discolored wood, which will decay over many years as long as some of the wounds remain open to provide oxygen.

A yellow birch becomes hollow 44 years after receiving a logging wound (Fig. 6c). All wood exposed by the wound has been completely decayed for several feet. Above the hollow, infected, discolored wood continues to decay. This part of the column merges into discolored wood that is generally associated with branch loss (stub outside, knot inside) and that may be infected. Four decades after the injury was inflicted and the exposed wood became infected, the wood has passed through progressive stages of deterioration and decomposition until a void is formed or the tree dies. The formation of a hollow tree is the result of the protective response of living cells of sapwood to wall-off (compartmentalize) fungal infection initiated by wounding and the continued formation of wood by the vascular cambium.

Once a tree is dead or made into wood products, no active life processes provide protection. The dead sapwood will quickly decay unless kept dry or wood-preserving substances are added as in this treated utility pole (Fig. 6d). However, once decay fungi become active beyond the outer preserved wood, they will spread and decompose wood as in the living tree. Brown-rot (which looks like burnt wood) in this utility pole is evident and it is progressing. Brown-rot is the most dangerous type of decay because wood quickly loses strength before any outward symptoms develop. Wood beyond the symptomatic stage of decay (where the finger is pointing) may look sound, but may have lost up to 70 percent of its strength. Electrical measurements can be made to distinguish between sound and infected wood to help assess risk of failure. In a living tree wound closure, compartmentalization, and cambial growth determine tree strength. Two to four inches of sound wood without cracks in the outer stem provide sufficient strength for trees to survive.

White-rot of wood causes strength loss much more slowly than brown-rot. We see new wood being formed in an oak with a basal wound, while old wood is being lost to decay in the center (Fig. 6e). If compartmentalization is strong (remember, compartmentalization varies genetically) and growth is good, this tree could survive a very long time; if compartmentalization is weak and growth is poor, the tree may not survive. The decay was progressing vertically and was detected in its early stages by electrical measurements taken well beyond the advanced, symptomatic stage using water extracts of wood drilled from bands of different colors (see drill holes, Fig. 6e).

Decay in living trees takes many forms. A white-pocket-rot in white pine (Fig. 6f) differs from the white-rot in oak (Fig. 6e). This pattern of decay in pine is called “ring-rot” because decay progresses quickly in discolored sapwood exposed by branch stubs, slowly in heartwood internal to the exposed sapwood, and is walled off by the protective layer formed after the infection. Over the next few decades, new sapwood and heartwood are added normally, as long as the tree remains healthy. This pattern of decay may then repeat itself several times in trees hundreds of years old. What would happen if growth after wounding was suppressed? Without several inches of sound wood outside the advancing decay, the tree would likely fail.



Figure 6.

CONCEPTS OF TREE DECAY

There are three basic types of host-pathogen interactions in trees: (1) obligate pathogens that interact only with live tree cells, as in the case of tree rust diseases; (2) facultative pathogens that interact both with live and dead tissues in trees, and (3) obligate saprophytes that act only on dead tissue in trees. Strong facultative wood-decay pathogens are fungi that attack both wood and bark and cause root-rots belowground and canker-rots aboveground. Weak facultative pathogens attack living, dying, and dead sapwood exposed by wounding. These are the most common fungi in live trees and vary in the degree of damage they cause depending on the tree's strength of compartmentalization and growth. Obligate saprophytes are the heartwood-decaying fungi that are highly specialized to attack strongly preserved wood and are often the same fungi decaying wood products and dead wood on the forest floor. All the interactions are regulated to varying degrees by environmental conditions (heat, cold, wet, dry, available nutrients, etc.).

Pioneer bacteria thrive in discolored wood that is not well aerated, and a wet condition can develop that is called "wetwood." Bacteria growing in live trees can cause a fluxing and odor problem. Pioneer fungi generally have small spores that can be transported in sap. Some of these fungi that happen to develop shortly after wounding and during the growing season may be fatal (sapstreak disease, Verticillium wilt, etc.). These fungi are short lived, whereas many pioneer fungi are long lived and may persist with bacteria in wetwood or may cause a soft-rot of well-preserved wood, making it more easily decayed once the wood partly dries and becomes aerated.

Root-rot fungi are strong facultative pathogens that infect the living wood and bark of roots and the butt section of tree trunks, e.g., *Annosum* root rot in red pine (Fig. 6g). This pathogen kills and decays wood and bark, which limits the effectiveness of compartmentalization. We see a resin-soaked reaction zone on the cut surface of the stump (Fig. 6h, arrow), which limits the spread of infection in wood, but the death of bark and cambium limits the effectiveness of barrier zone formation and wound closure. Essentially, the pathogen continually enlarges the wound to prevent closure and barrier zone formation. The fruit bodies of some root-rot fungi (Fig. 6i) are short lived and others seldom fruit at all. Thus, detecting the activity of root-rot fungi depends on observing indicators found at the base of trees.

Basal cracks between the buttress roots are useful indicators of root rot disease (Fig. 7a, balsam fir). The open cracks let in oxygen and accelerate the butt-rot process (Fig. 7b). Cracks and decay at the base of the tree create a hazard to people and property. As the root-rot pathogen spreads along the roots below ground, it moves into the stem through the slow-growing, thin sapwood zone between the buttress roots (Fig. 7c). This zone has a weaker response to compartmentalize the root-rot pathogen than the buttress roots.

We see the same pattern in hardwoods as in softwoods – cracks between the buttress roots near ground-line (Fig. 7d). Infection enters the stem between the buttress roots in which compartmentalization is more effective in healthy trees than the slow-growing region between the roots (Fig. 7e). Cracks develop in the weakened zone (Fig. 7f, collar-crack in yellow birch). However, crown loss due to ice storm injury or insect defoliation may reduce tree vitality, increasing the chances of root infection (mycelium of an *Armillaria* root-rot fungus spreading into the stem of birch after crown loss due to an ice storm, Fig. 7g). A central column of decayed wood (Fig. 7h, arrow) at the base of dead birch trees that lost more than half their crown by ice storm injury indicated that root-rot disease was well advanced before storm injury and tree morality.

Basal wounds of young fast-growing trees not only expose wood to decay but may cause seams to develop on the wounded face and internal cracks to develop (Fig. 7i). These seams are often called “frost cracks” but are initiated by basal wounds. When such trees become larger, they are subject to breakage and create a hazard.

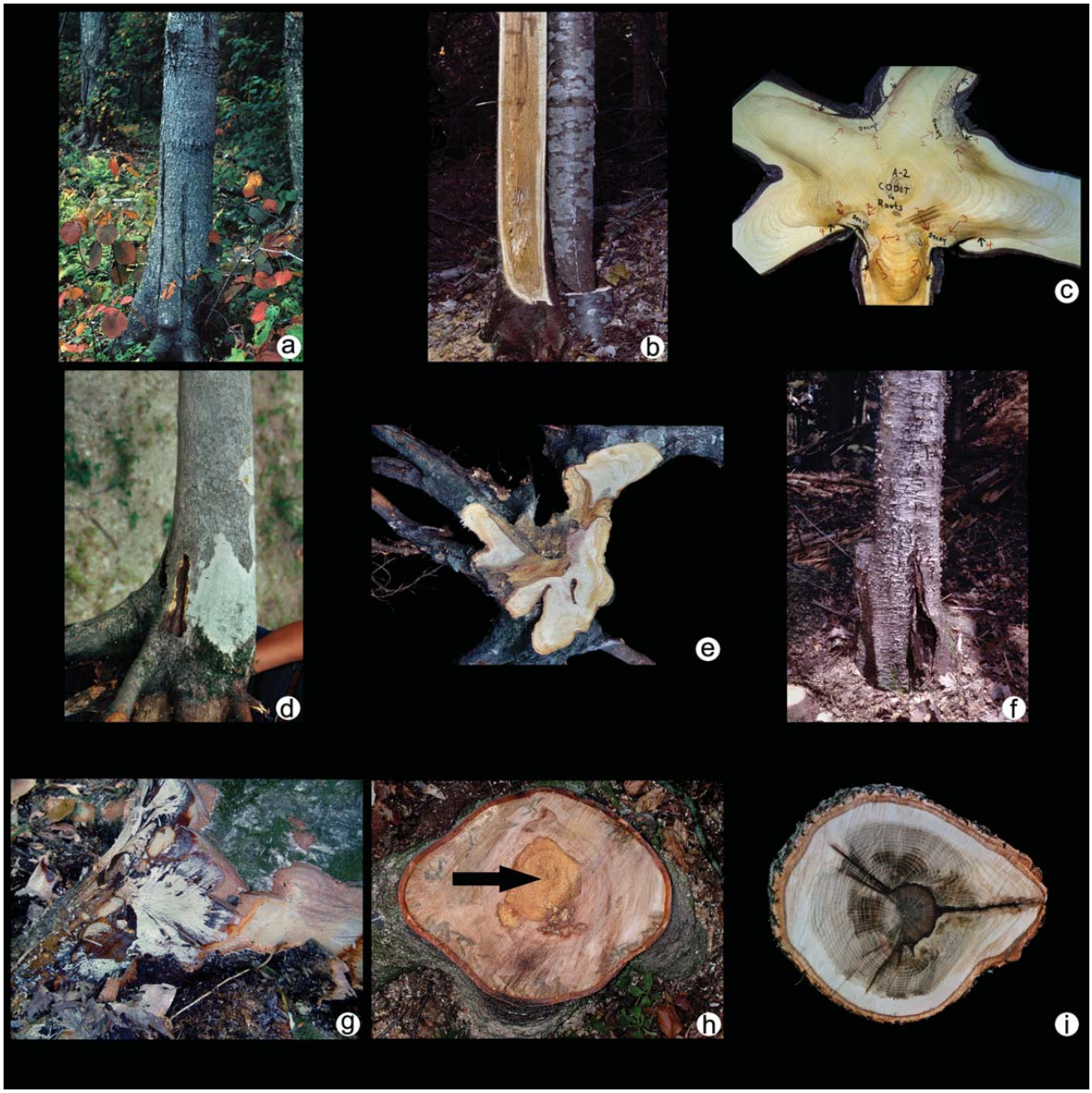

Figure 7.

Canker-rot fungi are strong facultative pathogens that attack the living wood and bark of tree trunks. This is a fruit body of *Phellinus pini,* a canker-rot pathogen of white pine and other softwoods (Fig. 8a). The fungus enters the tree through branch stubs ¾ to 1 ½ inches in diameter usually 6 to 15 feet aboveground. Pitch can often be seen coming from the base of such stubs before the fungus is developed enough to form a fruit body. Once established, the pathogen kills living wood and bark in the same manner as the root-rot fungi (Fig. 8b). The tree is partly successful in compartmentalizing the decay pathogen, but the fungus persists within the living tree. Advanced decay may cause the stem to snap under wind loading. Fungal infection of new stubs produces a ring-rot type of decay (Fig. 8c).

A canker-rot of birch is caused by *Inonotus obliquus*. This strong facultative pathogen kills bark and wood, decays wood, and produces a large black structure called a "sterile conk" made up of tree and fungus tissue (Fig. 8d). The actual fruit bodies of this fungus form only after the tree has fallen due to loss of strength from the open wound face and advanced decay. Another canker-rot of maple and beech is caused by *Inonotus glomeratus*. As in the case of obliquus canker, a sterile mass keeps the wound face open to accelerate decay and the formation of fruit bodies occurs only after the tree breaks off and falls to the ground (Fig. 8e).

Decay fungi that infect living, dying, and dead sapwood of open scars and stubs, but do not infect bark, are weak facultative pathogens. These are the most common decay fungi in living trees. A weak facultative sapwood pathogen infecting the stemwood of sugar maples exposed by logging scars gives highly varied results, due in part to genetic interaction (Fig. 8f). A strong compartmentalization response on the left yields a tree with mostly sound white wood after 15 years; a weak response on the right yields a tree with progressing decay.

Drying cracks on the wound surface and insect bore-holes accelerate the inward spread of infection in scar type wounds (Fig. 8g, red maple). Note how reaction zones form protective layers progressively deeper into sapwood (see arrows) until there is no longer enough stored energy to make them.

Cambial dieback (Fig. 8hd) following wounding reduces the effectiveness of compartmentalization because phloem is not available to bring needed sugars to be converted to protective substances in reaction zones in sapwood. Strong closure produced by the cambium promotes a strong compartmentalization response and markedly limits the spread of infection (Fig. 8hc). In an experiment (Fig. 8i) in red maple, a scar with bark removed (lower left) and a bore-hole wound with an intact cambium and bark (lower right) strongly resist the spread of a weak facultative decay pathogen. However, combining a scar and a bore-hole (upper center) promotes rapid spread of infection, which produces a much larger column of discolored and decayed wood.

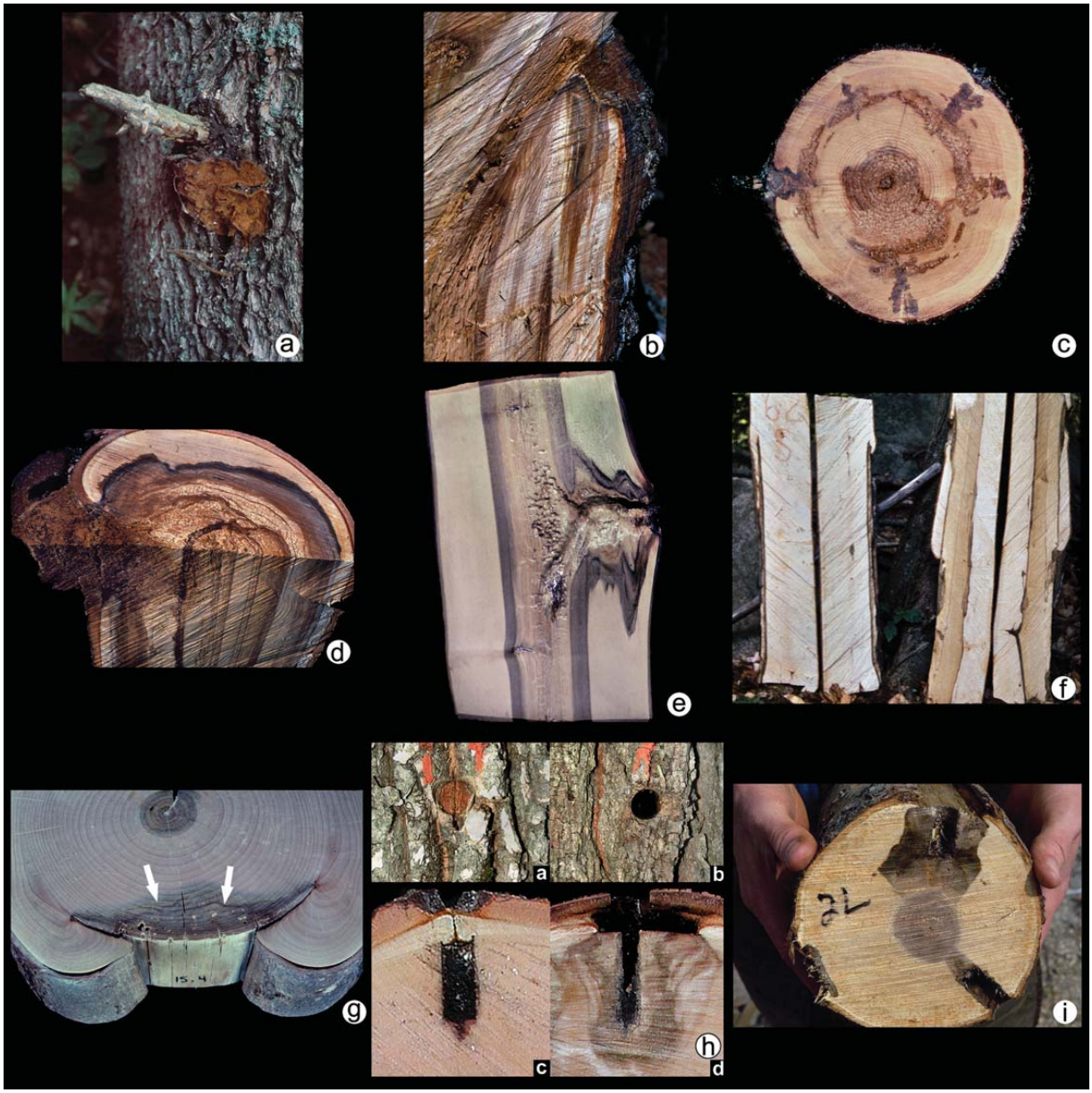

Figure 8.

After several years we see a bore-hole column with strong compartmentalization on the outer (right) side where bark is intact (Fig. 9a). Note the lack of compartmentalization (no dark reaction zone) along the inner boundary (arrow) in which live sapwood is cut off from phloem by the dead, discolored wood. Note the development of decay by a white-rot fungus in a light zone within the discolored wood (left side).

Weak facultative decay pathogens remove dead branches by decaying them. This decay of dead branches is very important to remove stubs that prevent wound closure (Fig. 9b). If branches die when they are small, compartmentalization in branch sapwood will prevent decay infection from penetrating into the tree (Fig. 9c). If the branches are larger (not shown), decay will spread down the branch-wood to the center of the tree where decay will not adversely affect tree strength. Given good growth after branch shedding, the branch stub becomes sealed as a knot inside the tree. The wood pathogen stops producing decayed wood due to lack of aeration (Fig. 9d). The discolored wood associated with branch wounds may be infected. If infected, it will be wetter and have a greater mineral content than non-infected, discolored wood. If a canker-rot pathogen infects wood and bark and the branch stub remains open, decay will progress into the stem and degrade the wood, weakening the tree stem (Fig. 9e).

Proper pruning of living and dead branches favors good wound closure and compartmentalization that will limit decay activity (Fig. 9f). Improper pruning that exposes not just branch-wood to infection, but also stem-wood, creates conditions favorable for decay development (Fig. 9g). The split fork, a common type of injury due to glazing of branches during ice storms, leaves a wound that exposes a lot of stem-wood (Fig. 9h1) compared to broken tops (Fig. 9h2) or broken branches (Fig. 9h3). However, the decay that follows is limited by the capacity of the tree to compartmentalize the infection as in the case of basal scars (compare Figures 9i and 3d).

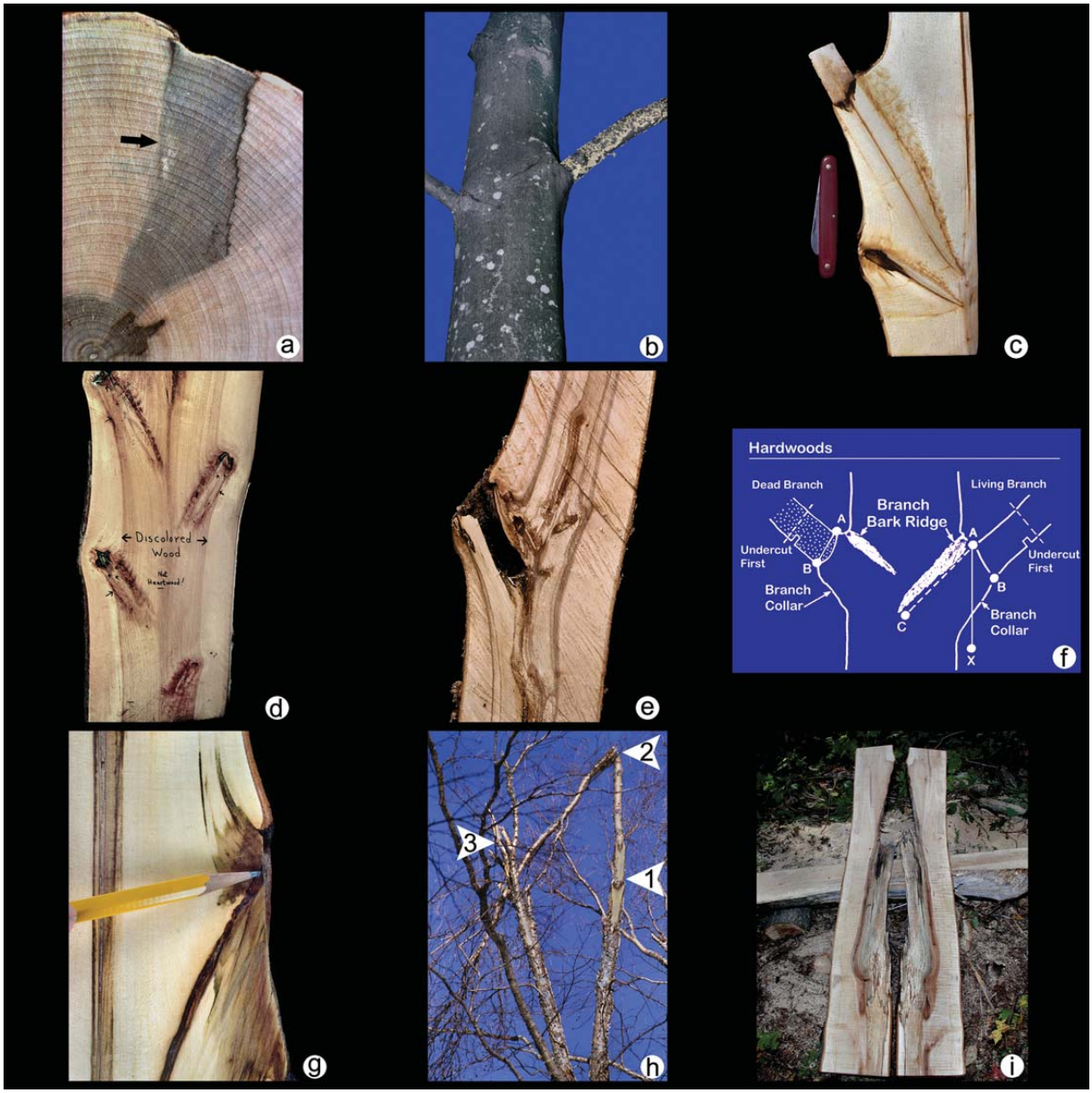
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Figure 9.

QUICK REVIEW: THE DEFINITION OF A TREE AND THE BASIC UNDERSTANDING OF TREE BIOLOGY NEEDED TO EVALUATE TREE DECAY

A tree is a large, long-lived, perennial, compartmented, woody, shedding, walling plant. Some trees get larger and live longer than any other organisms on Earth (Fig. 10a). A tree is a green plant with leaves, a stem, and roots (Fig. 10b). As trees grow larger, they become mostly stems of wood and bark. As trees grow from 1 to 20 m in height, the stem increases from about 5 to 75 percent of total dry mass, foliage decreases from 60 to 5 percent, branches from 20 to 5 percent, and roots remain at about 20 percent (Fig. 10c).

Food made in the leaves is distributed about equally to aboveground and belowground parts when a tree is small, but as a tree becomes larger, 80 percent or more of its food goes to maintain belowground roots. Many tree problems begin in the root zone. At maturity the massive stem supports the crown of branches where leaves make food to sustain life as long as sufficient water and essential elements are provided by the roots belowground (Fig. 10d). Trees are perennial and must survive from one season to the next (Fig. 10e). Trees growing in temperate regions must work hard to sustain life as they pass from aboveground activity in summer when shoots elongate, to belowground activity in winter when roots elongate. A record of annual stem growth can be seen in tree rings (Fig. 10f). We see there are good years (wide rings) and poor years (narrow rings) for growth depending on stand density (in forests), climate, and nutritional factors (in the root zone). Some of these factors can be regulated by silvicultural practices and by proper tree care.

Tree stems are compartmented into networks of living cells (the symplast includes outer wood, inner bark, and the cambium) and networks of dead cells (the apoplast includes vessels and fibers in hardwoods and tracheids in softwoods). Open vessels in this sugar maple (Fig. 10g) make excellent pathways for the spread of wood pathogens, but notice that this network of dead cells contacts the network of living cells (ray cells and marginal parenchyma cells between rings) at several points. These living cells store food reserves and are ready to protect against the spread of wood pathogens by compartmentalization.

When pathogens introduced through wounds contact living cells, this triggers compartmentalization, which limits the spread of wood pathogens while the trees continue to grow. Sometimes the system works well to limit the size of columns of discolored and decaying wood (Fig. 10h), other times it does not (Fig. 10i). The degree to which column size is limited depends on a combination of genetic and environmental factors. A limited degree of control can be afforded by proper tree care and silviculture. Regardless, some trees survive, other do not. Of all the 4-inch diameter saplings in a hardwood stand, only 10 percent on average survive to become mature 16-inch trees (Fig. 11a). As the younger, smaller trees die, they are decayed to enrich the soil with organic matter and nutrients for the surviving trees.

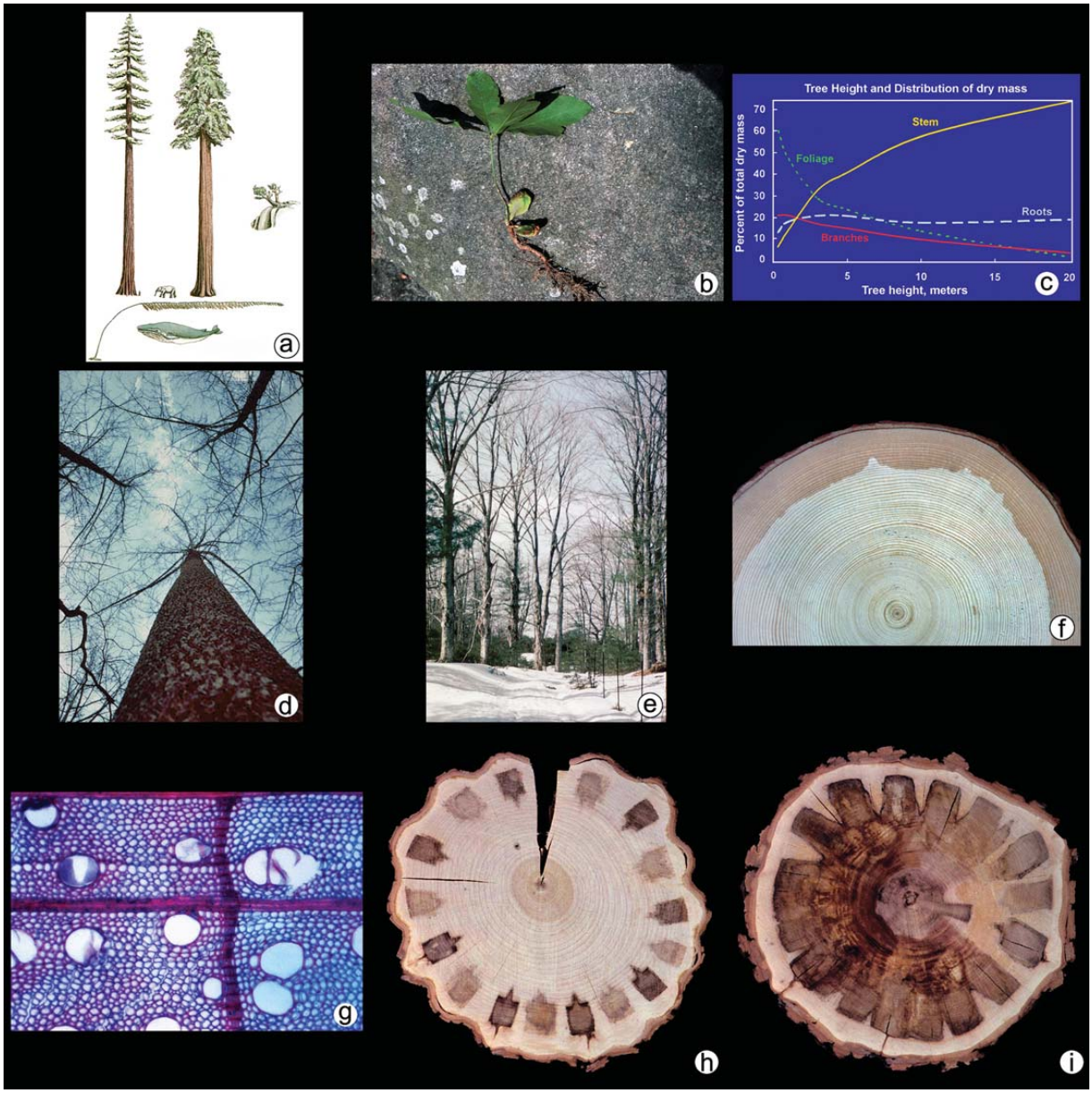
a
b
Tree Height and Distribution of dry mass
Percent of total dry mass
70
60
50
40
30
20
10
0
Stem
Foliage
Roots
Branches
0
5
10
15
20
Tree height, meters
c
d
e
f
g
h
i

Figure 10.

HOW TO EVALUATE TREE DECAY

Sometimes evaluating tree decay problems is obvious (Fig. 11b). Sometimes it is more difficult to determine how decay is progressing within the living tree from what can be seen externally (Fig. 11c). Comparing what is observed externally to the general patterns of decay following two types of wounding (Fig. 1i), scar wounds (Fig. 11d) and stub wounds (Fig. 11e), gives an idea of what is happening internally. If the trees being observed are being grown where they present a potential risk to people and property, key questions to ask are how much wood is being removed and how much is being added, is stem growth and wound closure sufficient to maintain tree strength, are there cracks in the outer several inches of wood that must carry the weight of the crown (especially in wind), what can be done to prevent further damage, etc. If the trees are being grown for wood products, key questions to ask are how long to wait to harvest trees after major wounding events before losses of wood quality and yield due to wounding exceeds gain from wood formed after wounding, are the wounded trees infected with strong pathogens that kill bark and decay wood (root-rot and canker-rot fungi) or with weak pathogens with limited activity, etc. Rapid growth, strong wound-wood formation for wound closure, and strong compartmentalization to wall-off infections initiated by wounds mean years of gain of sound high quality wood. However, slow growth, weak wound-wood formation delaying wound closure, and weak compartmentalization that fails to limit the spread of wood pathogens means rapid loss of wood quality. Knowledge and skills needed to assess risk of lost wood quality and tree survival begin with learning the basics of tree biology and how to apply that knowledge to common tree species growing in a changing environment.

Compartmentalization of decay in trees is a powerful system that protects the living parts of a tree, allowing it to survive many wounds and infectious wood pathogens. But like any system, it has its limits (Fig. 11f). It is important to learn about these limits and work within them. Actively maintain good tree health and protect trees from wounds whenever possible. Learn how to properly treat wounds and how to detect high risk trees. Take prompt action when a hazard tree has the potential to harm people or property. With a sound knowledge of tree biology, strive to balance the economic and ecological effects of stand management. Manage to improve wood quality associated with discoloration and decay while recognizing the importance of some loss of quality to sustain a healthy ecosystem.

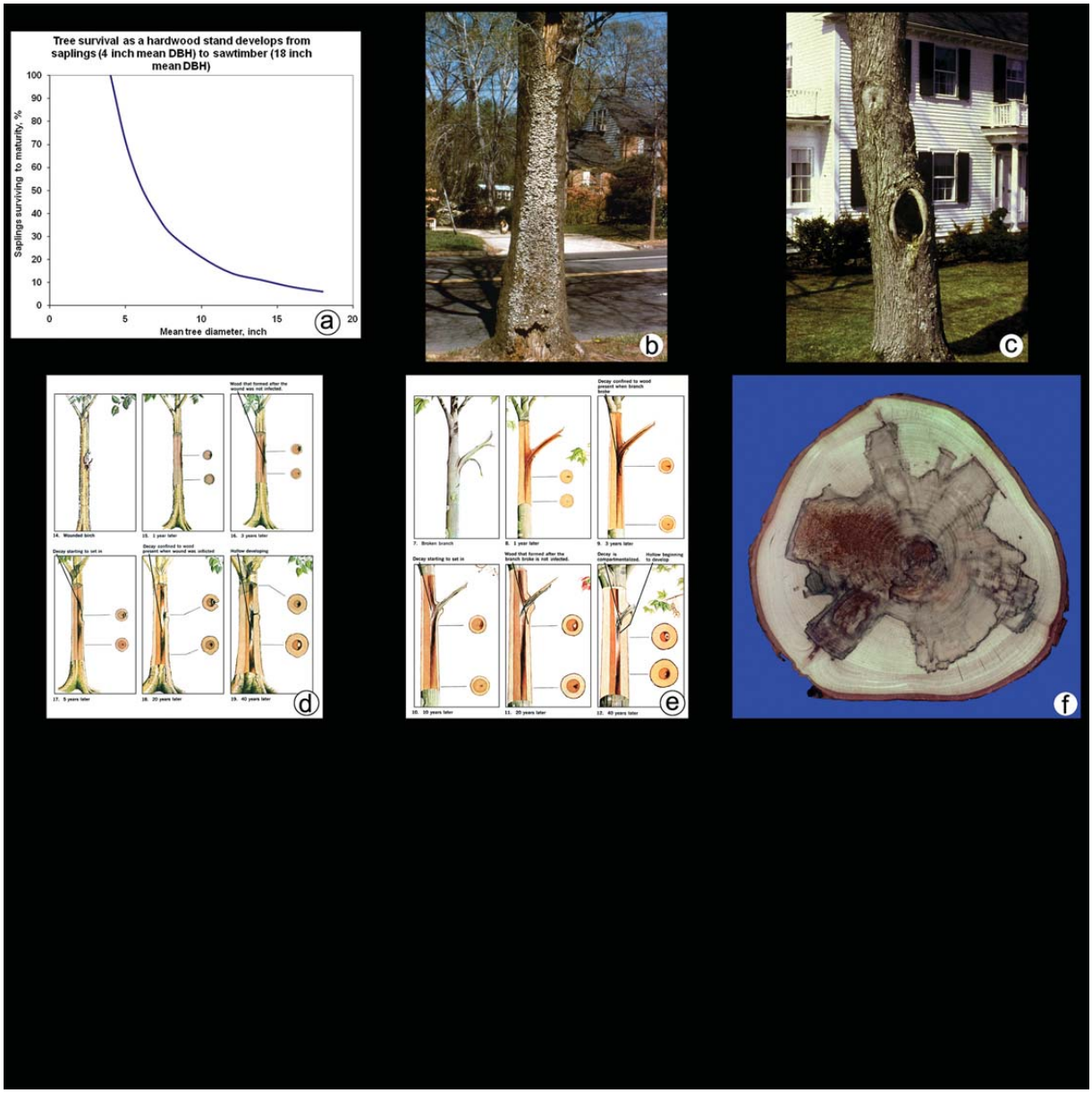
Tree survival as a hardwood stand develops from saplings (4 inch mean DBH) to sawtimber (18 inch mean DBH)
Saplings surviving to maturity, %
Mean tree diameter, inch
a
b
c
d
e
f

Figure 11.

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Pioneering research by Alex L. Shigo and his associates has produced a series of pictorial guidelines to provide a better understanding of how trees respond to wounding and subsequent microbial infections that lead to wood decay. The purpose of this paper is to visually summarize through the use of 96 color photographs and illustrations, the varied patterns of wood discoloration and decay observed in the dissection of thousands of trees. This information has served as a conceptual framework for understanding the biochemical processes that limit the spread of wood-destroying infections initiated by wounding during the maturation of all trees. This understanding has helped those who work with forests, trees, and wood to resolve practical problems and improve the health and productivity of trees and forests.

KEYWORDS: wounds, discoloration, compartmentalization, wood-decay fungi, pioneer fungi

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