

DISCOLORATION & DECAY IN OAK

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ABSTRACT. Diseases that result in discoloration and decay of wood are major problems affecting all species of oak. Wounds often start the processes that can lead to these diseases. The type and severity of the wound, the vigor of the tree, the environment, and the aggressiveness of microorganisms that infect are some of the most important factors that determine the nature of the disease. Decay following fire wounds and decay in sprout stems have been the major defects in oak. Now discolorations and other defects once considered minor are becoming more important.

THE OAKS ARE important not only in the United States, but also in many other countries. Because of their importance in central Europe, where forest pathology emerged as a science, the species of oak there were among the first trees studied by early forest pathologists. Some of the first studies by these investigators were on decay, which was considered a major problem. Studies on decay by Robert Hartig at that time set firmly the foundation for forest pathology (*Hartig 1878, 1894*).

When forest pathology began to emerge in the United States at the turn of the 19th century, decay was considered a major problem in need of research attention. And decay in oak was high on the list of priorities. George H. Hepting was the first to develop a sound research program on decay in oaks. Some of his early studies were made with George G. Hedgcock, who was one of the first forest pathologists in the United States. In 1935, Hepting published

on decay following fire in young Mississippi delta hardwoods (*Hepting 1935*). This important work set the stage for many decay studies that were to follow.

Many investigators have since studied decay in oak, and many valuable contributions have been made; but the results of studies by George H. Hepting, E. Richard Toole, and Elmer R. Roth still account for most of our present information about decay in oak. A detailed literature review and brief accounts of all the important discoloration and decay studies on the major species of oak in the United States are given by Hepting (*1970*). This publication will give anyone interested in discoloration and decay in oak an immediate exposure to all the important literature and to an accurate summary of the information available. Because this publication exists and is readily available, I will only outline briefly the information given by Hepting and will present some other information about discoloration and decay in oak.

DISCOLORATION AND DECAY PROCESSES IN GENERAL

Diseases that result in discoloration and decay of wood are major problems affecting all species of oak. Wounds often start the processes that can lead to these diseases. The type and severity of wound, the vigor of the tree, the environment, and the aggressiveness of microorganisms that may infect are some of the most important factors that determine the nature of the disease.

The events that follow wounds on trees of high vigor are different from those that follow wounds on trees of low vigor. Trees have repair systems that heal wounds. And most wounds do heal. The processes that can result in discoloration and decay start when the force of the repair system is overcome by the force of invading microorganisms. Anything that lowers the vigor of the entire tree or a portion of it will then lower the force of the repair system.

Wounds on low-vigor trees are the most serious problem.

The many species of oak in the United States grow on a wide variety of sites. Some of these sites are very poor, and the trees on them are low in vigor. Add to this the declines, diebacks, oak wilt, insect defoliations, insect borers, droughts, and many other problems that reduce the vigor of the trees; then add further wounds caused by fires, logging operations, animals, and other agents: and it seems a wonder that oak can even survive at all.

Oaks are different in many ways from the northern hardwoods—beech, birch, and maple. A few of the important differences center about the true heartwood core and the ring-porous wood in oaks. These differences play an important part in the way discolorations and decays develop in oak. The heartwood is impregnated with many substances that make it resistant to attack by many microorganisms. Yet some microorganisms under certain conditions can infect heartwood. The vessels in oaks often form tyloses in response to injury. These tyloses block the passage of many microorganisms. Yet some microorganisms invade tissues containing tyloses.

Discoloration

Wounds may expose sapwood or both sapwood and heartwood in oaks. The depth of the wounds may then determine whether microorganisms that attack sapwood or heartwood will infect. The living cells in the sapwood elicit a dynamic response, while the dead cells impregnated with various substances in the heartwood elicit a passive response.

The response of the sapwood to wounding in oak is not so different from the response to wounding of the wood in a northern hardwood. The first events that follow wounding of the sapwood are chemical responses, in which cellular materials are altered as a result of exposure to the atmosphere. The altered materials resulting from these processes can impart a slight discoloration to the wood. When the wound remains open and conditions for infection by microorganisms remain favorable, microorganisms infect and the discoloration of the wood intensifies.

The discolored wood that results after wounding appears similar to heartwood and it is often called a type of heartwood: "... an alternation of the wood exactly similar to normal heartwood formation also occurs as a pathological phenomenon in the vicinity of wounds" (*Büsgen and Münch 1931*), page 126). Hart (1965) showed that discolored sapwood was higher in moisture content, ash content, and pH, and was lower in its cold-water-solubility than was heartwood. The Committee on Nomenclature (1964) defined heartwood as:

... the inner layers of wood which, in the growing tree, have ceased to contain living cells and in which the reserve materials (e.g., starch) have been removed or converted into heartwood substances. It is generally darker in color than sapwood, though not always clearly differentiated.

This definition is based on three major conditions: cell death, depletion of nutrients, and deposits in the cells; and a minor condition, darkening of the tissues. These four conditions occur in discolored wood also, depending on the stage of development. The processes that resulted in the discolored wood may continue as micro-

organisms invade further, and decay may result. This does not occur in heartwood that is not injured and where no microorganisms are involved. This is an important difference between heartwood and discolored wood (Shigo 1967).

Heartwood may also discolor after injuries. Insect galleries are commonly surrounded by discolored heartwood.

Discoloration following increment borings developed slower in oaks (Hepting et al. 1949) than in other hardwoods (Lorenz 1944). However, Toole and Gammage (1959) pointed out that the discoloration resulting from wounds made in the spring in Nuttall oak extended vertically farther than in four other species of trees (eastern cottonwood, green ash, sweetgum, and sugarberry). Following such wounds the discoloration developed as a streak along the sapwood-heartwood boundary (Shigo and Sharon 1968).

In the past, discolorations in oak were not considered important defects. Slight discolorations did not reduce the value of wood used for railroad ties, construction materials, and the like. When defect-free wood was wanted, there was always a large supply to choose from. Now the situation is different. Discolorations in oak are becoming an important problem as more high-quality products are made and as more oak is used for veneer. For example, wooden floors, furniture, wall panels, and many other products must be free of discolorations to satisfy the modern market. We know relatively little about discolorations in oaks. We need to know more.

Decay

No doubt more is known about decay in species of *Quercus* than in species of any other genera of hardwood trees. The wood has been used primarily in products where strength over a long period is essential. Anything that caused a reduction in the strength of the wood caused problems. No one wanted a house, a ship, a railroad bed, or a bridge built with wood that might fail under stress, use, or time.

Although in many cases the decay recognized by most people was decay of the

product, this still called attention to decay. And, when enough pressure is exerted by people, some action must follow. In the United States, this pressure began to build early in the 20th century. The vast forests of oak in the Appalachian Region were considered of high value. Information was needed to help predict the development of decay over large areas and to estimate internal defects associated with external indicators. At the same time oak was receiving this attention, other hardwoods in the northeastern United States were being girdled in favor of the conifers.

Early studies carried out by Hepting and Hedgcock (1935a, 1935b, 1937) on 5,882 trees cut from 1924 to 1928 in the Appalachians showed that 77 percent of all the defects were due to butt rot, 2 percent were due to top rot, and 3 percent were due to other defects. And 94 percent of the butt rot was due to microorganisms entering through fire wounds. These studies showed clearly the extreme importance of fire wounds.

Fire Wounds

The amount of butt rot in any area depends on the history of fire in that area. Information for predicting cull following fire in Appalachian oaks is given by Hepting (1941). The predicting equation for individual trees was based on the width of the basal wound 12 inches above ground and the age of the wound. The variation in the amount of decay associated with the wounds was due to the different fungi invading the wood. *Stereum frustulosum* and *Hydnum erinaceus* were the most common fungi associated with advanced decay.

Hepting gives also a good account of a succession of decay fungi following fire wounds (Hepting 1941). *Schizophyllum commune*, *Panus stipticus*, *Daldinia concentrica*, *Nummularia* sp. (*Hypoxylon*), and some species of *Stereum* and *Polyporus* produced sporophores on the wound surface the first summer after the fire. The next summer *Stereum rameale*, *S. lobatum*, and *Lenzites betulina* produced sporophores, and the third year sporophores of *P. gilvus* began to appear. The heartrotting fungi then followed.

For oaks in the Southern bottomland, Kaufert reported on decay following fire in 1933 (Kaufert 1933). Of 261 red oaks examined in a 70-year-old stand, 72 percent had decay. Later Toole and Furnival (1957) and Toole (1959) extended the studies of Kaufert and Hepting to include larger trees, other species, and additional information on the rate of decay induced by various species of fungi.

Toole (1959a) also gave information on a succession of fungi infecting fire wounds. His observations were similar to those of Hepting (1941). Toole (1959a) stated that, although 30 species of fungi were identified, only 5 species were associated with half of the rot: *Pleurotus ostreatus*, *Hydnum erinaceus*, *Polyporus fissilis*, *P. lucidus*, and *P. sulphureus*. In a recent study on decay in upland oak stands of Kentucky, Berry (1969) found that 26 percent of 490 trees had fire wounds and that most of the advanced decay was caused by two species of fungi: *Poria cocos* and *P. nigra*. *Poria cocos* was limited to the butt, while *P. nigra* invaded the butt and trunk.

Although better methods for fire prevention during the last decade have reduced considerably the number of new fire wounds, there are still many trees in our forests that have active decay columns associated with old fire wounds. The forester still needs all the information he can get on estimating defect associated with fire wounds. Because the history of fire will be different from area to area, and because different aggressive fungi will be active in the different areas, the information must pertain to specific areas to be useful.

Rot in Sprout Oak

In 1936 Elmer R. Roth and Bailey Sleeth pointed out in a preliminary report (Roth and Sleeth 1936) that a very high percentage of decay in sprouts in fire-free oak stands was traced to the parent stump. *Stereum gausapatum* was the principal fungus associated with the decay. In 1937, Hepting and Hedgcock stated that there was more decay in stump sprouts than in seedlings and seedling sprouts (Hepting and Hedgcock 1937). Subsequent work gave valuable information on butt rot in unburned sprout

oak stands (Roth and Sleeth 1939), on wounds and decay caused by removing larger companion sprouts of oaks (Roth and Hepting 1943a), and on origin and development of oak stump sprouts as affecting their likelihood to decay (Roth and Hepting 1943b).

Recommendations for managing oak sprout stands came primarily from these studies. This work is summarized by Hepting and Fowler (1962). To minimize decay in sprouts, they give three general recommendations: (1) favor low-origin sprouts; (2) favor those that are separated from a companion sprout by a low U-shaped crotch; and (3) make a flush saw cut when removing a companion sprout. Hepting and Fowler (1962) diagrammed the procedures.

The paper by Roth and Hepting (1969) on the prediction of butt rot in newly regenerated sprout oak stands culminated the series of sprout oak studies begun in the early 1930's. This study showed clearly that the information gained from past studies was indeed applicable. The authors also pointed out reasons why decay in sprouts will be on the decrease in future stands. The modern chainsaw makes it possible to cut trees low on the stump. Fewer small trees are being cut for fuelwood, and large stumps do not sprout as readily as small stumps. Burning after clearcutting insures low-origin sprouts. And—most important—the information gained from all the studies on sprout stems has found its way into practice.

Root Rots

Hepting (1970) divides the root rot into three groups:

- (1) Those few minor rots not caused by Hymenomycetes;
- (2) those Hymenomycetes which play a secondary role in the death of trees and cause little butt rot;
- and (3) those which play a secondary role in tree death, but can rot not only the woody tissue of roots, but can cause extensive butt rot.

Phytophthora cinnamomi and *Phymatotrichum omnivorum* are in the first group. They are most active on roots of young nursery seedlings.

Armillaria mellea and *Clitocybe tabescens* are the major fungi of the second group.

They are active on roots of oaks of all ages. Actually, little is known about the role of *A. mellea* in the death and decay of roots.

The major fungi in the third group are *Polyporus berkeleyi*, *P. curtisii*, *P. dryadeus*, *P. lucidus*, *P. spraguei*, and *P. sulphureus*.

Root rots in oak may be more important than is now recognized. More studies that entail digging and dissecting roots of mature trees must be done.

Top Rots

Hepting, Garren, and Warlick (1940) studied the external features correlated with top rot in 333 Appalachian oaks. Poorly healed large branch stubs were the major infection courts correlated with top rot. Roth (1948) pointed out that the decay hazard increased with the width of the wound. If pruning is to be done, he recommended that the branch should be less than 1.5 inches in diameter when cut. *Stereum gausapatum* and *S. rameale* were isolated commonly from the decay associated with large open branch wounds.

Canker Rots

The principal decay fungi that incite cankers on oaks are *Polyporus hispidis*, *Poria*

spiculosa, *P. laevigata*, and *Irpex mollis* (Sleeth and Bidwell 1937, Campbell and Davidson 1942, Roth 1950, and Toole 1955, 1959b). Roth (1950) points out that there was no relation between tree vigor and the occurrence of *Irpex mollis* cankers, since they were found on both slow-growing and fast-growing oaks on both good and poor sites.

Decay Fungi

As studies on decay in oaks progressed, it became necessary to know more about the fungi associated with the decays. It became evident that more information was needed on the sporophores and on the identification of the fungi in culture. The outstanding investigators who worked on the sporophores were Lee Oras Overholts, who wrote "The Polyporaceae of the United States, Alaska, and Canada" (Overholts 1953), and Josiah L. Lowe, who wrote "Polyporaceae of North America: The Genus *Fomes*" (Lowe 1957) and many other works, especially on the genus *Poria*. The outstanding investigators who worked on the cultural characteristics of the decay fungi were Ross W. Davidson (Davidson et al. 1938); Davidson et al. 1942; and W. A. Campbell (1938).

CONCLUSION

The great amount of work done on decay in oak by only a few dedicated researchers is very impressive—especially when you consider some of the conditions they had to work under. Their work set a firm foundation for work that must now follow.

The changing world is changing priorities constantly. New problems are demanding new solutions. More emphasis is being placed on individual trees, on discolorations, and on many other defects considered minor in the past, but now of major importance. More people are looking at trees. There are green belts, recreation sites,

street trees, backyard trees, and watershed trees—and the list goes on and on. In the future it will not be so important where the tree is, but the fact that the plant is a tree. And, in the future, trees will need all the help they can get to survive.

We are now in a position somewhat similar to the one that started in the early 1930's. More information will be needed on the processes that result in discolorations and decays in individual trees. More information will be needed on better ways to prevent and stall the processes that lead to these defects. Now is the time to take the next step.

Literature Cited

- Berry, Frederick H.
1969. DECAY IN THE UPLAND OAK STANDS OF KENTUCKY. USDA Forest Serv. Res. Paper NE-126. 16 pp., illus. NE. Forest Exp. Sta., Upper Darby, Pa.
- Büsgen, Moritz, and E. Münch.
1929. THE STRUCTURE AND LIFE OF FOREST TREES. 436 pp. (Translation by Thomas Thomson.) Chapman & Hall, Ltd., London.
- Campbell, W. A.
1938. THE CULTURAL CHARACTERISTICS OF THE SPECIES OF *Fomes*. Torrey Bot. Club Bull. 65:31-69.
- Campbell, W. A., and Ross W. Davidson.
1942. A SPECIES OF *Poria* CAUSING ROT AND CANKERS OF HICKORY AND OAK. Mycologia 34:17-26.
- Committee on Nomenclature.
1964. MULTILINGUAL GLOSSARY OF TERMS USED IN WOOD ANATOMY. Comm. Nomen. Int. Assoc. Wood Anat. Mitt. Schweiz. Anst. forstl. Versuchsw. 40: 1. 196 pp.
- Davidson, Ross W., W. A. Campbell, and Dorothy J. Blaisdell.
1938. DIFFERENTIATION OF WOOD-DECAYING FUNGI BY THEIR REACTIONS ON GALLIC OR TANNIC ACID MEDIUM. J. Agr. Res. 57:683-695.
- Davidson, Ross W., W. A. Campbell, and Dorothy Blaisdell Vaughn.
1942. FUNGI CAUSING DECAY OF LIVING OAKS IN THE EASTERN UNITED STATES AND THEIR CULTURAL IDENTIFICATION. USDA Tech. Bull. 785. 67 pp.
- Hart, J. H.
1965. FORMATION OF DISCOLORED SAPWOOD IN THREE SPECIES OF HARDWOOD. Mich. Agric. Exp. Sta. Quart. Bull. 48:101-116.
1965. FORMATION OF DISCOLORED SAPWOOD. Mich. Agric. Exp. Sta. Quart. Bull. 48:101-116.
- Hartig, Robert.
1878. ZERSETZUNGSERSCHEINUNGEN DES HOLZES DER NADELHOLZBÄUME UND DER EICHE IN FORSTLICHER BOTANISCHER UND CHEMISCHER RICHTUNG. 151 pp., illus. Julius Springer Verlag, Berlin.
- Hartig, Robert.
1894. TEXT-BOOK OF THE DISEASES OF TREES. 331 pp., illus. (Translation by William Somerville, Rev. and ed. with a preface by H. Marshall Ward.) Macmillan and Co., London.
- Hepting, George H.
1935. DECAY FOLLOWING FIRE IN YOUNG MISSISSIPPI DELTA HARDWOODS. USDA Tech. Bull. 494. 32 pp.
- Hepting, George H.
1941. PREDICTION OF CULL FOLLOWING FIRE IN APPALACHIAN OAKS. J. Agr. Res. 62:109-120.
- Hepting, George H.
1970. DIAGNOSIS OF DISEASE IN AMERICAN FOREST AND SHADE TREES. (In press.)
- Hepting, George H., and Marvin E. Fowler.
1962. TREE DISEASES OF EASTERN FORESTS AND FARM WOODLANDS. USDA Forest Serv. Agr. Info. Bull. 254. 48 pp.
- Hepting, George H., Kenneth H. Garren, and Paul W. Warlick.
1940. EXTERNAL FEATURES CORRELATED WITH TOP ROT IN APPALACHIAN OAKS. J. Forestry 38:873-876.
- Hepting, George H., and George G. Hedgcock.
1935a. RELATION BETWEEN TREE DIAMETER AND PERCENTAGE OF CULL IN SOME EASTERN HARDWOODS. USDA Forest Serv. Tech. Note 13. 7 pp. Appalachian Forest Exp. Sta., Asheville, N.C.
- Hepting, George H., and George G. Hedgcock.
1935b. RELATION OF CULL PERCENT TO TREE DIAMETER AND TO PERCENTAGE OF TREES WITH BASAL WOUNDS IN SOME EASTERN HARDWOODS. USDA Forest Serv. Tech. Note 16. 4 pp. Appalachian Forest Exp. Sta., Asheville, N.C.
- Hepting, George H., and George G. Hedgcock.
1937. DECAY IN MERCHANTABLE OAK, YELLOW POPLAR, AND BASSWOOD IN THE APPALACHIAN REGION. USDA Tech. Bull. 570. 30 pp.
- Hepting, George H., Elmer R. Roth, and Bailey Sleeth.
1949. DISCOLORATIONS AND DECAY FROM INCREMENT BORINGS. J. Forestry 47:366-370.
- Kaufert, F.
1933. FIRE AND DECAY INJURY IN THE SOUTHERN BOTTOMLAND HARDWOODS. J. Forestry 31:64-67.
- Lorenz, Rolland C.
1944. DISCOLORATIONS AND DECAY RESULTING FROM INCREMENT BORINGS IN HARDWOODS. J. Forestry 42:37-43.
- Lowe, Josiah L.
1957. POLYPORACEAE OF NORTH AMERICA: THE GENUS *Fomes*. N. Y. State Univ. Coll. Forestry Tech. Pub. 80. 97 pp.
- Overholts, Lee Oras.
1953. THE POLYPORACEAE OF THE UNITED STATES, ALASKA, AND CANADA. 466 pp. Univ. Mich. Sci. Ser. vol. 19. Univ. Mich. Press, Ann Arbor.
- Roth, Elmer R.
1948. HEALING AND DEFECTS FOLLOWING OAK PRUNING. J. Forestry 46:500-504.
- Roth, Elmer R.
1950. CANKERS AND DECAY OF OAK ASSOCIATED WITH *Irpex mollis*. Plant Dis. Rep. 34:347-348.
- Roth, Elmer R., and George H. Hepting.
1943a. WOUNDS AND DECAY CAUSED BY REMOVING LARGE COMPANION SPROUTS OF OAKS. J. Forestry 41:190-195.
- Roth, Elmer R., and George H. Hepting.
1943b. ORIGIN AND DEVELOPMENT OF OAK STUMP SPROUTS AS AFFECTING THEIR LIKELIHOOD TO DECAY. J. Forestry 41:27-36.
- Roth, Elmer R., and George H. Hepting.
1969. PREDICTION OF BUTT ROT IN NEWLY REGENERATED SPROUT OAK STANDS. J. Forestry 67:756-760.
- Roth, Elmer R., and Bailey Sleeth.
1936. DECAY IN SPROUT OAK STANDS. USDA Bur. Plant Ind., Emerg. Conserv. Work. 4 pp.
- Roth, Elmer R., and Bailey Sleeth.
1939. BUTT ROT IN UNBURNED SPROUT OAK STANDS. USDA Tech. Bull. 684. 43 pp.
- Shigo, Alex L.
1967. THE EARLY STAGES OF DISCOLORATION AND DECAY IN LIVING HARDWOODS IN NORTHEASTERN UNITED STATES: A CONSIDERATION OF WOUND-INITIATED DISCOLORATION AND HEARTWOOD. XIV IUFRO Cong. vol. IX, sect. 41: 117-133. Munich, Germany.

Shigo, Alex L., and Edward M. Sharon.

1968. DISCOLORATION AND DECAY IN HARDWOODS FOLLOWING INOCULATIONS WITH HYMENOMYCETES. *Phytopathology* 58:1493-1498.

Sleeth, B., and C. B. Bidwell.

1937. *Polyporus hispidus* AND A CANKER OF OAKS. *J. Forestry* 35:778-785.

Toole, E. Richard.

1955. *Polyporus hispidus* ON SOUTHERN BOTTOMLAND OAKS. *Phytopathology* 45:177-180.

Toole, E. Richard.

1959a. DECAY AFTER FIRE INJURY TO SOUTHERN

BOTTOM-LAND HARDWOODS. USDA Forest Serv. Tech. Bull. 1189. 25 pp.

Toole, E. Richard.

1959b. CANKER-ROTS IN SOUTHERN HARDWOODS. USDA Forest Serv. Forest Pest Leaflet. 33. 4 pp.

Toole, E. Richard, and George M. Furnival.

1957. PROGRESS OF HEART ROT FOLLOWING FIRE IN BOTTOMLAND RED OAKS. *J. Forestry* 55:20-24.

Toole, E. Richard, and John L. Gammage.

1959. DAMAGE FROM INCREMENT BORINGS IN BOTTOMLAND HARDWOODS. *J. Forestry* 57:909-911.