some

others

Oak Wilt, a New Threat

Theodore W. Bretz

Oak wilt is a serious threat to our noble oaks, the lovely trees that since Colonial days have meant much to the economy and development of this country, account for one-third of the hardwood saw timber stand in eastern United States, and are highly valued as ornamentals and as sources of food for wildlife.

Oak wilt has received increasing attention since 1940 because of the damage it has caused in shade oaks and wood-lot and forest oaks in Iowa, Wisconsin, and Minnesota. Since 1947, it has been found in several other Midwestern and Appalachian States from Pennsylvania to North Carolina. It is infectious and destructive, and many foresters, arborists, conservationists, and plant pathologists are concerned about it.

We do not know how long it has been in the United States. An epidemic dying of oak, observed in southern Wisconsin and southern Minnesota 40 years ago, was ascribed to various causes—adverse climatic conditions, insects, and other diseases. But investigators of the Wisconsin Agricultural Experiment Station and the Department of Agriculture established the fungus nature of oak wilt in 1942, and it seems probable that the disease may have been responsible for at least some of the earlier mortality of oaks in that area.

Until the summer of 1949, oak wilt was known only in Wisconsin, Minne-

sota, Iowa, Illinois, and east central Missouri. In 1949, forest pathologists found it in the northern Ozark region of southern Missouri and in northwestern Indiana.

Funds obtained from the Forest Pest Control Act and the Arkansas Resources and Development Commission financed a limited survey by Federal forest pathologists in 1950, mainly in the Ozark Mountains. Low-flying airplanes were used to locate suspected infected trees in the forests. Ground crews then examined the suspected trees and collected specimens for laboratory confirmation of the field diagnosis. As a result of that work, the oak wilt was found to be widely scattered in southern Missouri and in northern Arkansas. Other workers found the disease in a few isolated locations in Kansas, Nebraska, Ohio, and Pennsylvania.

Airplane scouting for oak wilt has proved to be satisfactory, fast, and economical. An aerial observer's most effective survey for wilt is limited to about one-quarter mile. Two observers in a plane can usually survey a strip one-half mile wide through the forest. A third member of a crew can greatly speed the work by keeping the position located at all times on a map and indicating on it the location of suspected trees seen by the observers. Landmarks can also be noted that will be useful to a ground crew who may later visit the area to collect specimens for laboratory culturing.

The development of the aerial-scouting technique to locate oak wilt and the discovery of the disease in the Appalachians brought greatly expanded survey activities in 1951. Federal, State, and private organizations participated in the surveys, and their efforts disclosed the presence of the disease over much of our hardwood area. Besides the States I mentioned, oak wilt was found in Michigan, West Virginia, Kentucky, Tennessee, Virginia, Maryland, and North Carolina.

Oak wilt was not found in any additional States in 1952. It was found in many new locations throughout its range, however, with a notable increase in the number of known infection centers in Ohio and in Pennsylvania west of the Susquehanna River.

OAK WILT PRODUCES noticeable and characteristic leaf symptoms, which vary somewhat with the species affected. The diseased trees are most conspicuous from mid-June to mid-September. In species belonging to the red and black oak group, symptoms usually appear first in the top of the tree and at the ends of the lateral branches and progress rapidly downward and inward through the entire crown. The leaves first become dull or pale green, curl upward, and become stiff. They turn yellow or bronze from the apex and margins inward.

The blade tissue next to the petiole is the last to turn brown. Affected leaves may fall from the tree at any stage of symptom development. Defoliation may be slight or nearly complete. Some leaves may remain on the tree to the end of the season and a few may even persist until the following summer. Sometimes sucker growth, in the form of dense clusters of large, succulent leaves, develops on the trunk and larger branches before an infected tree dies. The bark of wilt-killed trees loosens rather rapidly and by the end of the second year following death may begin to shed from the bole.

In white and bur oaks the leaf symptoms are often much more localized. Usually the entire tree does not wilt at once. Individual branches in any part of the crown may develop leaf symptoms, while leaves on the unaffected parts of the tree remain green. The affected leaves may be tan or dark green and look water-soaked. They tend to remain on the branches after they die. The killing of individual branches over a period of years results in stagheading. A brown or black discoloration in the outer sapwood just under the bark is

sometimes present in the twigs and branches of infected trees. When one peels back the bark he can see a diffuse brownish discoloration or longitudinal streaks. In cross section it appears as a brown ring or circle of dark-colored spots just under the bark.

The symptoms are sufficiently distinctive so that a diagnosis can usually be made in the field. We know of no other disease of oak with those symptoms or effects. For accurate identification, however, particularly in areas in which the disease has not previously been known, laboratory isolation of the causal fungus from diseased specimens is advisable.

The oak wilt fungus is closely related to the fungi that cause the Dutch elm disease and the canker stain disease of the London planetree, and is a near relative of the important blue stain fungi of forest products. It was described and named *Chalara quercina* by Berch W. Henry in 1944. Later, having discovered new facts regarding its life history, I reclassified and named it *Endoconidiophora fagacearum*.

All species of oak thus far subjected to infection under natural conditions or by artificial inoculation have proved susceptible to oak wilt. They include the following species native to the region in which oak wilt occurs—white (*Quercus alba*), swamp white (*Q. bicolor*), northern red (*Q. borealis*), eastern red (*Q. borealis maxima*), scarlet (*Q. coccinea*), northern pin or Hill's yellow (*Q. ellipsoidalis*), southern red or Spanish (*Q. falcata*), swamp red (*Q. falcata* var. *pagodaefolia*), shingle (*Q. imbricaria*), bur (*Q. macrocarpa*), blackjack (*Q. marilandica*), chestnut (*Q. montana*), chinquapin (*Q. muehlenbergii*), water (*Q. nigra* and *Q. nigra* f. *hemisphaerica*), pin (*Q. palustris*), willow (*Q. phellos*), basket or swamp chestnut (*Q. prinus*), post (*Q. stellata*), Shumard (*Q. shumardii*), and black (*Q. velutina*).

Turkey (*Q. laevis*), laurel (*Q. laurifolia*), Texas red (*Q. shumardii* var. *texana*), and live (*Q. virginiana*) oaks from the Gulf Coast, Gambel (*Q. gambelii*) and Oregon white (*Q. garryana*) oaks from the west coast, cork (*Q. suber*) and English (*Q. robur*) oaks from Europe, and sawtooth (*Q. acutissima*) and Daimyo (*Q. dentata*) oaks from Asia developed typical symptoms of oak wilt when they were artificially inoculated with the fungus.

In nature, species of the red and black oak group usually die the same season that they first show symptoms, often within a few weeks after the first symptoms appear. When symptoms first appear late in the season, however, diseased trees may survive the winter. The following spring sparse, dwarfed, and short-lived foliage develops. Species of the white oak group die more slowly. Bur oaks have been reported to lose all their leaves and die in a single season. The relative susceptibility to the disease of many of the species when growing in their natural environment has not yet been determined. Such information can be obtained only by observation of the behavior of the disease in nature.

Trees closely related to oaks are also known to be susceptible to the disease. Naturally infected Chinese chestnuts (*Castanea mollissima*) have been found and it has been shown by artificial inoculation that the American chestnut (*C. dentata*), European chestnut (*C. sativa*), tanoak (*Lithocarpus densiflorus*), and bush chinquapin (*Castanopsis sempervirens*) are susceptible to oak wilt in the greenhouse.

Experimental evidence indicates that the oak wilt fungus can infect only through wounds that penetrate the bark of the tree. Once established, the fungus multiplies rapidly and is distributed widely in the sapwood of the tree by the time the symptoms first appear. That may happen within 3 weeks after inoculation. A direct and early response of the host to the infection is the plugging of the water-conducting vessels in the sapwood. That response may be actuated by a toxin, which the fungus may produce

and which alone will induce wilt symptoms. The obstructions that form in the water-conducting tubes in the sapwood may limit the water supply available to the leaves, and the shortage may contribute to the development of the leaf symptoms. No important plugging of the roots of diseased trees has been observed, and it has been demonstrated that the fungus can move from diseased to nearby healthy oaks through natural root grafts.

Natural root grafting is common in stands of red and black oaks. The grafts provide a highway through which the fungus can pass from tree to tree. Such spread probably accounts for the somewhat radial manner in which the disease extends from an initially infected individual. Progress of the disease in that way is slow. Bur and white oaks in mixed stands often remain unaffected even after the population of red and black oaks has been largely wiped out. A probable explanation might lie in the infrequency of root grafting between different oak species. Root grafts from bur to bur and white to white are reported to be uncommon.

How oak wilt spreads over distances greater than those we can attribute to natural root grafting is not known. Such above-ground spread does occur, but how far from old centers of disease the fungus may be carried in a single passage has not been determined. By aerial surveys oak wilt has been located many miles from any other known center of infection. Such trees often are the dominant or codominant trees in the stand.

Infection by wind-blown spores may possibly occur but appears to be relatively unimportant, even though the fungus has been found to sporulate abundantly under the loosened bark of some wilt-killed trees. If such spread did occur, a much more definite pattern of distribution of the disease within a given area and a much greater mortality in our oak population would be expected than has been the case. Some other agent or agents of dissemination appear more likely. The discovery that the fungus possesses a spore form (ascospores) admirably suited to transmission by insects or birds, because of its sticky nature, may provide a clue to the mystery of long-distance spread. The ascospore form, which develops only when two compatible strains of the fungus are brought together, has been observed beneath loosening bark of trees killed by oak wilt. The frequency with which it occurs in nature and whether it may develop on other tree parts or other host plants remain to be determined.

THE SPREAD OF THE DISEASE in local areas can be retarded by interrupting the movement of the fungus from diseased to healthy trees through root grafts. In situations in which individual trees are of little value, this may be most readily accomplished by poisoning the healthy trees around the perimeter of diseased trees. A 50-foot buffer zone of killed trees around the infection center is recommended.

Among street, lawn, and park trees, mechanical severance of roots connecting diseased and healthy trees may be more desirable. That may be done by trenching or (in some types of soil) by the use of an especially designed, tractor-drawn knife blade. A cut 30 inches deep is believed sufficient to sever all connecting roots between trees. The effectiveness of the method depends largely upon the promptness with which the root cutting is done after the disease is detected. Root severance should not be delayed more than 3 weeks after the first symptoms appear.

To what extent standing, wilt-killed trees may remain a source of infection is unknown. As the fungus may sporulate under the loosened bark of wilt-killed trees and has been isolated from trees almost a year after death, disposal of such trees by burning or sawing them into lumber and burning the slabs and brush seems to be advisable as a supplementary practice.

We have begun experiments to get more exact information on the danger

of leaving oaks that are poisoned and, if they are a danger, on other methods of making certain that they cannot serve as sources of disease. Since wilt symptoms on white and bur oaks may be limited to a few isolated branches, careful and rigorous pruning of all wilting branches has been reported to prolong the life of an occasional tree.

The development of measures (other than the sanitation of known infection centers) for the control of long-distance, above-ground spread of oak wilt will probably await the discovery of the means of such spread. In many regions, however, oak wilt is not yet firmly established and the number of infection centers are few and small. Prompt elimination of those centers might prevent serious spread of the disease.

The fungus will kill small oak trees, but whether the disease may be spread through shipment of nursery-size trees is not known. There is but little evidence with regard to the hazard dimensional lumber may present in the spread of the disease. However, the fungus has not been isolated from heartwood. Kiln drying should kill the fungus if it were present in sapwood. The fungus is reported to have survived for almost a year in logs with bark on them.

Although the disease is widely scattered through much of our commercially important timber in the Ozarks and Appalachians, the percentage of trees infected is low and no significant losses have occurred. But oak wilt is capable of much destruction; should an efficient vector for the fungus appear, it could become a catastrophe. Research on all aspects of the disease has been started and we hope that a control of this menace can be found.

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Littleleaf in Pines in the Southeast

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Littleleaf, a disease of shortleaf (*Pinus echinata*) and loblolly (*P. taeda*) pines, was first observed in Alabama in 1934. The early symptoms are a yellowing of the foliage and a reduction in growth. Shoot growth and needle length become greatly reduced as the disease progresses, and often the affected trees produce an abundant crop of small cones. Trees with advanced symptoms are conspicuous because of their short, yellow needles, which are confined to the ends of the branches. The disease rarely affects trees younger than 20 years of age and is most prevalent in stands that are 30 to 50 years old.

Littleleaf occurs in varying amounts in Mississippi, Alabama, Georgia, South Carolina, North Carolina, Virginia, and Tennessee. The littleleaf belt, from Virginia to Mississippi, embraces approximately 30 million acres, of which over half is in forests. Areas of abundant littleleaf cover about 5 million acres of this forest land. But even in areas of greatest abundance, littleleaf is not uniformly distributed throughout stands of shortleaf pine. Age of stand, soil conditions, and degree of erosion are important factors in the distribution of the disease. Stands with severe littleleaf may be found close to healthy stands. The spotty occurrence of littleleaf in many areas and often the relative lack of the disease in nearby stands are generally determined by soil relationships.