

THE OAK WILT SITUATION

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ABSTRACT. Although oak wilt has been studied for more than 30 years, there are many facets of the disease that are little understood. Continuing Federal-State cooperative studies are geared to predicting the overall effects of the disease on future forest-management programs, but much additional research is needed before present control programs can be expanded or discarded in a conscionable way.

OAK WILT, a vascular disease caused by the fungus *Ceratocystis fagacearum* (Bretz) Hunt, is potentially the most serious disease to affect oak trees in the 21 states where it has been found to occur. Widespread catastrophic losses have not yet been documented, although thousands of individual infection centers have been located by aerial and ground surveys in patchily infected forest stands. It is not known why the disease is limited to only part of the oak range in North America, but this phenomenon is thought to be related to ecological factors that directly or indirectly affect the pathogen or the vectors.

The origin of the disease remains a mystery. First described in Wisconsin in the early 1940's, it was almost immediately found in other states in the upper Mississippi Valley. By 1951 it had been found as far east as Pennsylvania, Virginia, and West Virginia. Estimates vary as to how long it has been present.

The pathogen is a vascular parasite that enters through any wound that ruptures

the outermost xylem tissue. Single spores have been used to artificially initiate infection, although the incubation period seems to be shortened when a large number of spores are used.

Artificial inoculations have shown that the causal agent is virulent on at least 51 species of *Quercus* and several close relatives in the genera *Castanea*, *Lithocarpus*, and *Castanopsis*. It also causes defoliation of the Jonathan apple (*Malus*) and it has survived for some time in sassafras. The lack of immunity by oaks suggests that the range may possible expand in the future.

SYMPTOMS

Generally, oak wilt symptoms are distinctive and may be recognized readily from the ground or from low-flying airplanes.

The leaves of infected trees droop or wilt, and some turn bronze or brown in color. They then fall from the tree. Often the leaves fall while still green, and in other instances a small green island is left

at the base of each. Sometimes one or more branches will remain green. About 6 weeks after defoliation, the larger branches produce sucker sprouts. Late fall infections may show no indication of the disease other than a premature fall coloration. Some of these trees refoliate normally the next spring and then produce symptoms, while others die over winter without producing visual symptoms.

Disease development often varies greatly between individuals, and it differs markedly between members of the red and white oak groups. Red oaks are thought to be more often infected. They exhibit symptoms over the entire tree, and the tree normally succumbs in 1 year. The foliar symptoms of white oaks, in contrast, are often restricted to localized branches, and they sometimes appear only during the season of infection.

In some instances white oak trees are able to "bury" the fungus with overgrowths of new wood. These hidden infections are seldom discovered unless the tree again develops foliar symptoms, as has happened, after a lapse of as much as 7 years. The role of such reservoirs of infection in continuation of the disease has not yet been fully accessed. Young chestnut oak trees may wilt and suddenly drop all of their leaves in a pattern reminiscent of young red oak, but older individuals of the same species may take somewhat longer—as is true for the white oak group as a whole.

The question about whether infected red oaks ever recover is often asked, but research has shown that the outer vessels of infected trees are plugged with gums and tyloses as much as 4 days before foliar symptoms develop; thus the disease is practically irreversible before the first leaf falls.

SPREAD

Oak wilt spreads both overland and underground. Overland dissemination is thought to be primarily due to certain sap-feeding beetles belonging to the family Nitidulidae, although *Pseudopityophthorus*, a genus of oak bark borers, has also been incriminated. The role of birds, squirrels, and other insects has been extensively dis-

cussed in the literature, but confirmation is lacking.

Nitidulid beetles are prime suspects in much of the northern part of the range because of their intimate relationship with the development of specialized fungal cushions. Bark borers are thought to be more logical vectors in southern areas, such as in Missouri, where the fungal cushions do not occur in abundance. These cushions begin with the development of large amounts of mycelium in the vessels. This mycelium then migrates intracellularly to the cambium, where it forms "mats" between the bark and wood. Endoconidia are produced in abundance on the mats as the bark is slowly cracked by the growth of opposing fungal cushions. This creates a cavity over the mat. A characteristic odor from the mat attracts insects which, while feeding on the self-sterile fungus, pick up conidia, both internally and externally, and later transport them to other mats where conidiation ensues and fertile perithecia result. Either ascospores or conidia can cause infections.

The fungus has been shown to move underground through natural root grafts between diseased and healthy trees. Starting with an initial infection, the disease, particularly in shallow or sandy soils, moves progressively outward in a more or less concentric pattern, although on steep hillsides the disease often follows the contour. Root-graft spread is most prevalent in Midwestern States, but it does occur to some extent wherever the disease has been found. Grafting is infrequent between members of the white and red oak groups, but it is known to occur.

CONTROL PROGRAMS

Oak wilt control programs have been characterized by small-scale experiments in some states and by statewide efforts in others. Some Eastern States conduct their programs in cooperation with the USDA Forest Service under auspices of the Forest Pest Control Act of 1947. In the upper Midwestern States, the disease was thought to be too widespread, when initially discovered, to be controlled.

For example, experiments in Wisconsin indicated that the rate of progress of the

disease was such that virtually all oak would be removed within 75 to 100 years. In some states, control was attempted and abandoned. In others, control was not attempted because of financial or other problems.

Two exceptions are Pennsylvania and West Virginia, where statewide control programs have been vigorously pursued for years. The control techniques used in these states are quite different, yet both have as their goal the elimination of the mat-like fruiting structures as sources of inoculum.

Before embarking on systematic control programs, discussion by Federal and State officials in the above-mentioned states revolved around whether or not the costs could be justified. Also, as the potential losses and the chances of control were not immediately known, it became important to decide whether controls based upon admittedly insufficient knowledge should be attempted. This decision had to rest upon the danger involved if control should be delayed, because there was the presumed potential of the disease for eliminating the number one timber tree in the region. Consequently it was decided to move into control as rapidly as possible, but to continually adjust or modify the control program as new information became available.

The techniques used in the control programs varied from the beginning. As Federal funds were being used in several of the programs, a cooperative study with four states was initiated in 1958 to check on the effectiveness of the differing techniques being used and the economic implications of each. This study eventually became an appraisal of Oak Wilt Control Programs in Pennsylvania and West Virginia.

In Pennsylvania, all diseased oaks and healthy trees of the same species group within 50 feet of diseased trees are felled, and the stumps are treated with Ammate. The diseased trees are felled to hasten drying, which in turn lessens mat formation; the healthy trees are removed to prevent possible spread through root grafts.

The West Virginia technique involves chopping a girdle to the heartwood at a convenient height. The bark is then re-

moved from the girdle to the soil line. This hastens drying of the tree as well as allowing for a rapid ingress of competing fungi such as *Hypoxylon*. Infection centers are located from low-flying airplanes in both states. Nontreatment or check plots were located in Maryland and Kentucky.

The initial study was conducted from 1958 through 1963, and the results indicated a reduction of diseased trees on plots treated by both methods. More importantly, the data showed that the disease intensified with time on untreated plots. As the different treatments and untreated checks were segregated by state, ecological factors could not be assessed; and this fact caused reservations about the validity of the study. A second study, designed to remove past criticisms, is now being conducted. The results of the first few years will be available soon. One very important aspect of this continuing control appraisal is the surveillance of quadrangle-sized plots instead of the more limited areas previously used.

Data compiled by West Virginia workers indicate that there is very little cost differential between the Pennsylvania and West Virginia treatments if the tree location is first plotted on topographic quadrangles and if only one trip is made to each infection center. There is, however, an overall cost differential due to the use of chemicals and to the greater number of trees cut in the Pennsylvania method.

RESEARCH NEEDED

The future of all oak wilt control programs hangs on research—research on the effectiveness of present techniques and research on untried comparisons. There is also a great need for research into the ecological aspects of the disease. For example, why is the problem restricted to only a part of the overall range of oak in North America? Is this a climatic limitation or is it correlative to vector distribution?

There are several holes such as this in our knowledge. For example, the causal fungus varies in appearance, physiological activity, and possibly in virulence. Thus, can we develop a system of continued monitoring of the organism to forecast the

appearance of new and more virulent strains, or strains adaptable to different environmental conditions, which could conceivably extend the present geographic range?

It is doubtful that the fungus is limited to distribution by a single vector, or even to two or three; but present knowledge on this matter is inconclusive.

The role that antibiotics and systemic fungicides will ultimately play in the treatment of infected trees in residential or other high-value properties needs to be more adequately evaluated. Will soil sterilants be developed that will inhibit root-graft transmission in the Appalachians as methyl bromide apparently does in the sandy soils of Wisconsin?

What are the respective roles of spring and fall mats? Is fall mat production important? If so, does the infection occur so that the fungus overwinters in the host? Or do the spores overwinter in the soil, in or on insects, etc.?

QUESTIONS, QUESTIONS

Natural regeneration in disease centers does not seem to be a problem in the Appalachians, but perhaps not enough time has elapsed to show certainly. Will this situation change when infection centers expand and unite so as to remove oak from acres in contrast to the present small centers? What is happening on large infection centers in the Midwest? Will the disease ultimately cause a degeneration of stand productivity throughout its range, even though there may not be any discernable influence on the establishment, survival, and development of individual oaks?

Will the mere presence of the disease continue to inhibit the exportation of oak logs to foreign countries? Will it be possible to exploit the fumigation treatments worked out by the USDA Forest Service as a technique to obviate present quarantine restrictions?

Are there unrecognized inoculum sources? If so, what are they, and how is the fungus transferred?

What factors govern the establishment of the disease in an otherwise healthy tree? Do we understand the overall ecologic fac-

tors that influence the susceptible-pathogen-vector relationships? Do we really understand the disease as it exists in nature?

There are dozens of similar basic questions that need to be asked and answered before we can assess the true potential of oak wilt. Will the fact that the requirements for successful transmission are very exacting and that the requirements may vary from region to region work to the detriment of control activities? Perhaps different control measures will need to be developed that will consider ecological peculiarities.

Perhaps we will never know the answers to all the questions. Perhaps the disease will silently disappear just as it arrived. Or perhaps it will intensify and devastate the resource before we can solve the riddle or develop the necessary control programs. It is ironic, but the overall pace of research effort has slowed in recent years. Can it be accelerated? Has it slowed because the easier answers have been found? Or is it because we have tired of the problem and have not yet been sufficiently remotivated? Could it be that many of the past problems have been solved in the laboratory, while most of the current questions will be answerable only in the field?

The evaluation of oak wilt intensities and the location of diseased trees through use of aerial surveying techniques is a field of recent interest. Much can be said for color and for infrared photography, but these are as yet strictly experimental and cannot be used on routine programs.

One problem with aerial techniques, both in surveying for oak wilt and for beetle-damaged pines, is that more or less overlapping contour flying is necessary. It has been shown that it is virtually impossible, at altitudes of 500 feet above the terrain and at speeds of 90 miles per hour, to locate single trees or even clumps of two or three infected trees more than $\frac{1}{4}$ mile distant. Therefore, surveys flown on 1 mile grids, or flown at 500 feet above the highest terrain, or flown at speeds in excess of 100 miles per hour, are suspect. Even if flown the exact same way every time, they will not give similar results, especially if new or

untrained personnel are used. In addition, grid flights never give 100-percent coverage, and they should not be reported as such.

WHAT NEXT?

In summary, the oak wilt situation is now virtually static; and program directors are badly in need of research information. It is anticipated that within the next 2 years the cooperative Federal-State analysis will provide guidance on how to proceed with present programs.

But, if the analysis indicates that control efforts should cease, then the true problem will rapidly emerge. Will we be able to develop information on how to predict the

total effect on forest management in time? Will we be able to recommend harvesting practices that will slow the disease? Will we be able to develop a statistical model to predict future intensities instead of relying upon deterministic calculations?

Oak is such a valuable resource that every possible effort must be expended to prevent its loss if the loss indeed seems imminent. The surveys and resultant control programs may have to be extended indefinitely. We just do not know at present. We hope some sort of biological control will finally be developed on the basis of complete ecological data. Only then will those entrusted with the management of our forest resources for future generations be able to relax.

