Oak Wilt Control — Preventive Medicine

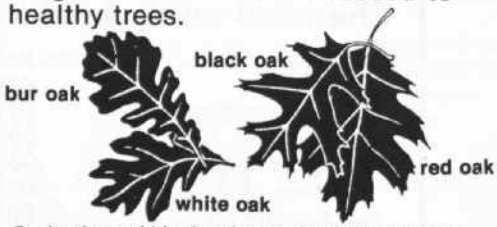
By Dr. James E. Kuntz

Healthy trees contribute greatly to the beauty of urban areas, including golf courses and other recreational areas. In Wisconsin, oaks are major shade and ornamental trees. But they suffer to varying degrees from attack by different insects and diseases. Fortunately, the many oak species vary greatly in their susceptibility or resistance to specific pests or to adverse environmental conditions. Preventive medicine, combining different control strategies, therefore, is possible by choice of the correct **Quercus** species, favorable soils and sites, and judicious management practices.

Oak wilt, a native, systemic, vascular wilt disease, incited by the fungus **Ceratocystis fagacearum**, is a serious lethal threat to oaks mainly east of the Great Plains. Every year in Wisconsin, oak wilt kills thousands of oaks in both urban and rural stands. Although there is no cure for diseased trees, much can be done to prevent initial infection and to limit further spread of the fungus pathogen. Oaks are not doomed; preventive medicine can be effective.

Disease Physiology. Before control measures can be formulated and applied, critical aspects of the disease must be understood. Continuing research has supplied much information. Microscopic spores of the causal fungus enter the water-conducting vessels, usually the large, open, long springwood vessels of the outer current annual wood ring. Especially in the spring (May and June), fresh, unprotected wounds are common entry points. Spores quickly multiply and are carried rapidly both upward and downward in the transpiration stream. Their metabolic products incite several internal host reactions including the proliferation of certain sapwood cells, the protrusion of balloon-like growths (tyloses) into infected vessels, the induction of gums and other dark staining materials, activation of dormant buds, and premature abscission of leaves. Vessels become plugged; water movement ceases; leaves wilt; and above ground parts of most trees die the first season. Roots, even infected ones, persist and remain active for some time, especially those roots grafted to roots of adjacent oaks.

In a dying oak, the fungus forms vegetative mycelium which grows outward to the cambium between the bark and wood. Under warm, moist conditions, the fungus destroys the cambium, loosens the bark, and forms many scattered mycelial mats or "pressure pads" which raise and crack the bark. The mats sporulate profusely and emit a "juicy fruit" odor which apparently attracts many insects including the common and familiar "picnic beetles." Such mat-invading insects become contaminated with spores and may serve as "vectors" to carry the fungus overland from diseased to healthy trees.



Red oaks and black oaks can be differentiated from the more resistant white and bur oaks by leaf characteristics. Leaf edges of reds and blacks have pointed tips; leaf edges of white and bur oaks are more curved and without points.

Host Symptoms. Red and black oaks are extremely susceptible to infection by the oak wilt fungus. Most die rapidly. Few, if any, recover. As commonly seen in June or July, leaves in the periphery of the upper crown wilt first, cure slightly, and pale to bronze or brown from their margins inward. Leaf symptoms progress rapidly downward through the crown. Leaves at all stages may defoliate prematurely. Occasionally, brown to black discoloration can be detected in the outer sapwood ring of a cut branch. Vessels, tightly plugged with tyloses, also can be seen with a handlens or microscope. Disease diagnosis can be confirmed in the laboratory by isolation of the fungus pathogen in pure culture from infected host tissues. Trees infected in late summer or fall may not develop symptoms until leaves begin to expand in the following spring.

White and bur oaks appear less susceptible to initial infection and resistant to subsequent disease development. Although some infected trees die the first season, many live for several years with more branches dying each year. Crown "stagheading" results. Some trees even recover. Their cambium remains active and lays down healthy wood over the infected annual ring, "burying" the infection.

In the current annual ring, initial infection is limited to arcs originating at the entry point of the fungus pathogen. Wilted and bronze leaves soon develop on branches with vascular connections to these infected areas in the trunk. The crown "symptom pattern" discloses the progressive development of the disease within the tree. Vessel plugging and dark brown streaking of outer sapwood are limited to infected arcs and associated branches. Premature defoliation is seldom heavy.

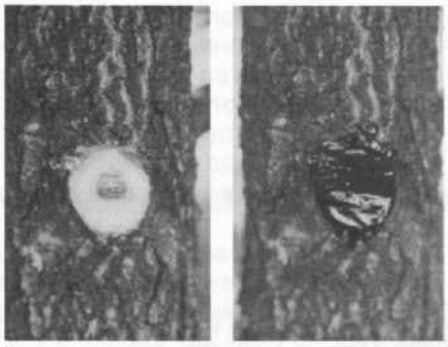
Disease Spread. The fungus pathogen is carried from infected oaks to healthy oaks in two ways. First, insect vectors contaminated with spores from sporulating mats infest fresh wounds where infections begin. Later, the fungus is drawn through root grafts from infected to adjacent healthy oaks. Present control depends on preventing such initial infection and subsequent spread.

Root grafts often unite red and black oaks growing within 50 feet of one another. Root grafts are less common among white and bur oaks, and rare, or not at all, between roots of members of the red and white oak groups. Root grafts are natural "pipelines" among adjacent oaks for water

and nutrients—but also, unfortunately, for the fungus pathogen. When vascular plugging reduces upward transpiration in an infected tree, the fungus is drawn through grafted roots into adjacent healthy oaks. New infections begin. An expanding oak wilt "pocket" of dead and dying trees results. In mixed oak stands, white and bur oaks may escape this "tree-to-tree" spread of the fungus among red and black oaks.

Insects, especially "picnic" beetles (sap-feeding nitidulid beetles) carry spores from fungal mats on infected oaks to fresh wounds on stems and branches of healthy oaks. Such wound infections on single scattered trees may initiate new infection centers in healthy oak stands.

Control.* Avoid initial infections through fresh wounds. Oaks are particularly susceptible in the spring and early summer, especially from budbreak to full-leaf expansion. Occasional infection through fresh wounds may follow summer rains. Consequently, prune and remove oaks during the winter. If spring or summer wounding does occur, protect wounds against spore-bearing insect vectors by prompt and thorough wound treatment with thick paint or wound dressing.



A fresh, flush-cut—vulnerable to infection (left). The cut on the right was painted immediately and thoroughly to prevent infection.

Sever root grafts. Sever either mechanically or chemically, root grafts connecting healthy trees to infected trees. A series of "barriers" among adjacent healthy oaks may be necessary since some trees beyond the first barrier may be infected already. Various mechanical equipment can be used to cut roots underground. Grafted roots can be severed chemically by soil fumigants (Vapam). Killed root sections block spread of the fungus.

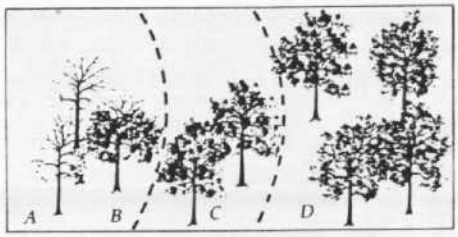


Illustration of a typical oak wilt "pocket," showing where barriers should be put. (A) Oaks killed by wilt. (B) Oaks with current symptoms. (C) Oaks without symptoms but near diseased trees (may already be infected). (D) Healthy oaks permitted by two rows of barriers.

Reduce mat formation by drying infected wood. Completely girdle the trunk base through the outer sapwood of diseased red and black oaks. Girdling hastens bark and wood drying which reduces or prevents fungus mat formation. Avoid moving infected materials,

When girdling diseased trees, cut deeply into the heartwood as shown.

including firewood, into areas free of oak wilt. Even where oak wilt is present, infected firewood should be dried rapidly. Cut, split, and stack off the ground as a single tier in a dry, open, sunny area. If possible, remove the bark. Protect from moisture.

In remote "roughs" where individual trees are of less value, local spread of oak wilt can be stopped by **creating a barrier of poisoned trees** around the oak wilt pocket. Root kill is essential. Removal and drying of infected material will improve control.

Meanwhile, continued research suggests that disease resistance may be found even among red and black oaks. Moreover, systemic chemotherapy may prove helpful in aiding the recovery of infected oaks, especially bur and white oaks.

*For details, see University of Wisconsin-Extension Circular A1693, Oak (**Quercus**) Disorder: Oak Wilt.

*Editor's Note: Jim Kuntz retired from the Plant Pathology Department in December of 1984, but in his position as Emeritus Professor he has hardly slowed down. Known for his exuberance for the study of plant diseases (this editor enjoyed having Dr. Kuntz as a lab instructor in PP 300, **many** years ago!), he continues his research on walnut canker, wilt and seed rot.*

Dr. Kuntz received a B.A. degree from Ohio Wesleyan in 1941 and both M.S. and Ph.D. degrees from Wisconsin in 1942 and 1945, respectively. He achieved international recognition for his contributions in the study of the oak wilt disease. He also has made major contributions in the general area of urban forest problems, those kinds of diseases of major concern to Golf Course Superintendents.