Cope With the Threat: Oak Wilt

by Dr. David N. Appel

Oak wilt is one of the more unusual diseases significantly affecting a major hardwood species in North America. Unlike more recognizable tree diseases, such as Dutch elm disease (DED) and Chestnut blight (CB), there is little known of the origin of the oak wilt pathogen, *Ceratocystis fagacaerum.* Although it is found in 22 states in the U.S.A. and is considered to be an extremely virulent fungus, the oak wilt pathogen has not caused the epidemic losses we previously experienced with DED and CB.

Oak wilt was first described in 1941, following the discovery of the pathogen killing red oaks in Wisconsin. There is some evidence that the disease was killing oaks in Minnestoa as early as the late 1800s. Oak wilt also may have been responsible for widespread live-oak mortality reported in 1931 in Austin, TX.

There are thousands of scientific and popular articles published over the past 50 years on oak wilt. The susceptibility of various oaks, the life cycle of the pathogen and the prospects for disease management all have been the focus of much research and discussion.

Most of the oak wilt literature deals with the disease in the Mid-Atlantic, Midwestern and North-Central states on deciduous oaks. More recently, the disease has been the focus of attention as it occurs on semi-evergreen live oaks (*Q. fusiformis, Q. virginiana*) in central Texas.

Red Oaks Are Most Susceptible

The genus *Quercus* encompasses an extremely large and diverse group of related species. The oak wilt fungus has been limited to the species that occur in the range depicted on the accompanying map. Throughout that range, deciduous red oaks (a large, sub-genus named Erythrobalanus within the genus *Quercus* spp.) are the most susceptible. Commonly affected red oaks include such species as northern red, northern pin, shumard and blackjack oaks. They are liable to die within a few weeks after appearance of the symptoms. They never recover from infection.

At the other extreme are white oaks of the sub-genus Leucobalanus, such as white oak, post oak and bur oak. They usually recover following minor crown loss and rarely die from infection.

In Texas, the semi-deciduous live oaks seem to have a low level of resistance to *C. fagacearum,* allowing 10 to 20 of the trees in many stands to survive indefinitely without any treatment.

There are several reasons for the relatively sporadic nature of oak wilt. Most of them relate to stages of the disease cycle considered to be "weak links" in the behavior of the fungus. For example, a number of conditions must be met before the fungus can be transmitted from a diseased tree to a healthy one by a beetle vector.

The first, and most important consideration, is a requirement for the production of pores on diseased trees. For the oak wilt fungus, this requirement is met by production of a sweet-smelling, spore-covered structure beneath the bark of the dying red oaks called a "fungal mat." However, fungal mats only form on a small portion of diseased red oaks and they are functional for only a few months during the spring. In Texas, fungal mats never form on live oaks; their occurrence is limited to diseased red oaks.

Sap-feeding "picnic" beetles are believed to be the primary insect vectors. Only a small proportion of beetles in an infested area are contaminated and capable of transmitting the pathogen. Further, these beetles require a fresh wound (less than two or three days old) to successfully introduce the pathogen into a new tree. These limitations make new infections relatively rare for such a virulent fungus.

In spite of the erratic means of insect spread, the oak wilt fungus can have devastating consequences for some species of oaks. This has proven particularly true in the live oak woodlands of central Texas. Live oaks grow in large, pure stands throughout a 50 to 60-county region, largely due to the ability to reproduce by prolific root-sprouting. The native live oaks tolerate home construction very well, making them the most popular shade trees in the state.

The "common root system," formed during growth of the root sprouts, may be maintained to the maturity of the tree. This provides an ideal means for the spread of the vascular-inhabiting fungus. The pathogen is believed to be vectored into the pure stands of live oak by the sap beetles, only to be rapidly transmitted to adjacent trees through the common root system.

In Texas, oak wilt is easily recognizable in live oaks due to the formation of large, rapidly-expanding "centers" of infection, sometimes reaching hundreds of acres. This means of transmission is similar to spread through root grafts, a common occurrence among deciduous red oaks. Root transmission of the oak wilt fungus is responsible for the death of far greater numbers of trees when compared to spread via the insect vector.

Save Healthy Trees

A successful oak-wilt-control program must take into account all potential means of transmission. As is the case with any tree disease, the best approach to reduce the losses is to prevent infections from occurring in unaffected stands.

There are a numbers of strategies to prevent the occurrence of a new infection center, as well as reduce the losses caused by the fungus spreading in an existing disease center. For example, insect transmission can be prevented by destroying the diseased red oaks before the fungal mats are able to form.

Another method to stop spread by insects is to avoid wounding the trees (including pruning) during the spring, when trees are most susceptible and contaminated beetles

are at their peak. If an oak is wounded during the spring in a high-hazard area for oak wilt, wound paint should be used to prevent the beetle from introducing the fungus into the wound. The wound paint needs only be effective for a few days, because only wounds less than two to three days old are susceptible to infection. Once oak wilt is introduced to an area, root transmission must be contended with. This is especially true in live oak, where no fungal mats are formed and root transmission is so efficient. In practice, root transmission is easily prevented simply by mechanically breaking the roots between diseased and healthy trees. Any trenching machine capable of chiseling through existing soil types is suitable.

Trench placement must be planned carefully, so that the fungus is successfully contained. If the trench is dug too close to a diseased tree, the pathogen may already to beyond the intended treatment area. Therefore, the position to the trench must be sufficiently far from the symptomatic oak in order to control spread of the fungus.

For oak wilt management in the North-Central U.S., tables are available to aid with trench placement by considering sizes (dbh, or diameter at breast height) and distances between the diseased tree and adjacent healthy trees. The tables provide a probability of spread between two trees within a year, and are even adjusted for different soil types.

Trench placement is not as sophisticated for pathogen control in live oak. In Texas, a 100-foot barrier is recommended between the symptomatic trees on the perimeter of the disease center and the trench. Further, best control is achieved when all trees within the 100-foot barrier are rogued to create maximum disruption of the root systems. Failure to control oak wilt in Texas with trenching is usually attributed to the roots growing beneath the recommended 4-foot level, or placement of the trench too near to an inconspicuous, yet diseased, tree.

Trenching and pulling trees out by their roots to save valuable shade trees are often unacceptable practices in the urban environment. This is particularly true when symptomless trees must be sacrificed in order to prevent further fungal spread. An alternative control for individual, high-value trees is tree injection. There is experimental evidence for recommending the fungicide, propiconozole (Alamo), as a high-volume, root-flare injection, propiconazole has been as high as 90 to 100 percent effective in saving live oaks on a preventative basis. Encouraging results also have been demonstrated in deciduous oaks. Therapeutic treatments are successful if the tree is injected before the pathogen has extensively colonized the tree.

There are, however, limitations to the use of injection to control oak wilt. When done properly, the process is time-consuming and necessitates extensive wounding of the tree. Also, injections with propiconazole do not prevent the pathogen from spreading further through the stand, but only protect the treated trees from succumbing to the disease with significant loss of crown. When injection is combined with the other measures for controlling oak wilt, such as trenching, a successful management program may be designed for most situations under which the disease if found.

The oak wilt fungus might have originated from a closely related species in the relatively recent past, thus explaining the limited range on a world-wide basis. On the

other hand, it might have been introduced from elsewhere, just as the DED fungus might have evolved in Europe or Asia, where the CB blight pathogen also is believed to have originated.

Regardless of where it originated, there are still valuable oak species in the U.S. that may yet be threatened by *C. fagacearum*. Our experiences with control practices developed in the current oak wilt range will undoubtedly be useful as the fungus expands into new oak forests. However, the Texas oak wilt epidemic has served to illustrate how progressive approaches in disease control will be needed, as new epidemics arise in previously unaffected forest types, climates and geographic locations.

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