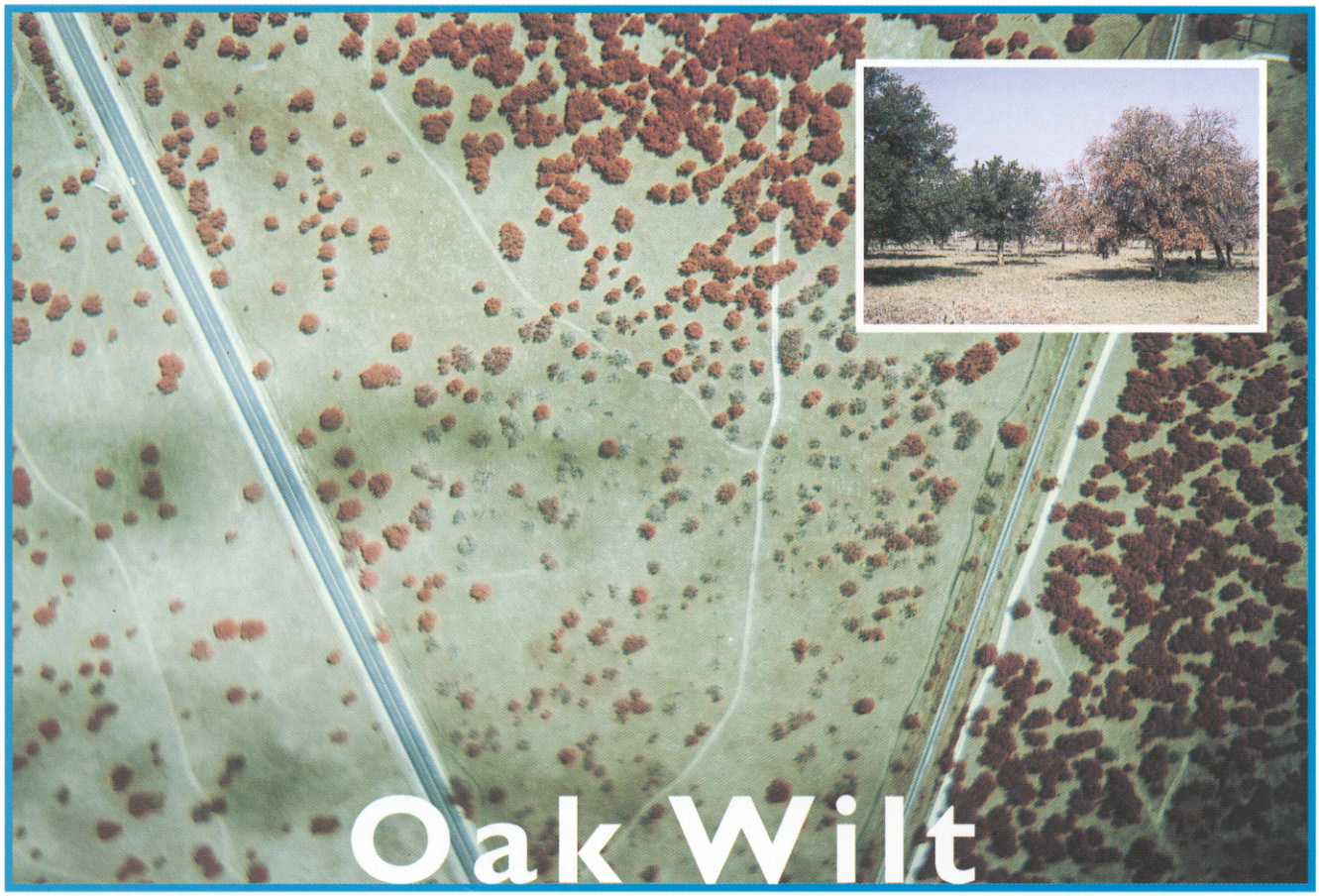

Photos courtesy of USDA Forest Service

Oak Wilt

A Potential Threat to Southern and Western Oak Forests

A. Dan Wilson

ABSTRACT

Oak wilt is recognized as one of the most destructive diseases to afflict oak species in the United States. The distribution and development of oak wilt in our eastern and midwestern oak forests has been closely linked to changes in forest stand composition, forest management practices, and pathogen dissemination facilitated by human and vector activity. The potential for oak wilt to spread into southern and western states and its possible impacts are discussed. The Texas experience with an oak wilt epidemic provides useful perspectives and suppression alternatives for other states not yet touched by this malady.

Keywords: entomology; pathology

Oak wilt has plagued hardwood forests in the United States for many years, probably long before it was first described as a fatal disease of oaks in Wisconsin (Anonymous 1942). The effects of the disease were likely observed by woodsmen who reported dying oaks, without knowing the cause, as early as the late 1800s and early 1900s in the upper Mississippi River valley (Gibbs and French 1980). It also was the probable disease reported to be killing live oaks in the Texas Hill country in the 1930s (Taubenhaus 1934, 1935), 27 years before it was officially identified in that state (Dooling 1961). The causal agent of oak wilt, *Ceratocystis fagacearum*, is a fungus that has since caused widespread damage as it has dispersed to its current range in 22 states *(fig. 1)*. The fungus continues to be responsible for killing a large number of oaks annually in this country. Although it was previously reported to occur in Louisiana, Mississippi, and Alabama (Peacher et

Above: **An infrared image of an oak wilt infection center in the hill country of central Texas; the bright red trees are healthy, the pink trees are declining due to oak wilt, and the gray trees are dead and totally lack foliage.** ***Inset:*** **Live oak trees in central Texas along the advancing edge of an oak wilt infection center.**

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al. 1975), these observations were based on symptoms alone and were not confirmed by isolation. Hence, there are still no confirmed incidences of its occurrence in the six southernmost states, except Texas. Nevertheless, its ominous and devastating presence in Texas confirms that oak wilt is still posing major problems for our nation's oak forests.

The objective of this article is to increase awareness among foresters, landowners, and forest managers of the nature and destructive potential of this disease, particularly in the southern states and in California, where oak wilt has not occurred but where highly susceptible oaks are abundant and vulnerable. The rationale for this perceived risk, the probable means by which the oak wilt fungus would likely be introduced and subsequently spread within these regions, and likely impacts of the disease on forests are discussed. Regional factors such as climatic, environmental, land management practices, and stand species composition that can influence disease development also are addressed. Finally, an example is provided of a currently operational oak wilt suppression program that describes what is involved in developing a control program should this disease become a problem in your state. The example also provides some insight into suppression strategies that have been successful.

Disease Recognition and Susceptible Hosts

The oak wilt fungus causes a root-transmitted disease of fagaceous hosts including oaks (*Quercus* spp.), chestnuts (*Castanea* spp.), chinkapins (*Castanopsis* spp.), and tanoak (*Lithocarpus* spp.) (Bretz and Long 1950; Sinclair et al. 1987). The oaks are the most seriously affected and common hosts. The pathogen infects oak species in both the white oak group *(Q. quercus)* and the red–black oak group *(Q. lobatae)* defined by Nixon (1993). Members of the red oak group generally are more susceptible and are killed by the disease faster and more frequently than are members of the white oak group. White oaks rarely die from oak wilt (MacDonald and Hindal 1981) and are often tolerant of the disease, although no oak species is known to be immune.

Few forest pathogens have killed their hosts as quickly as *C. fagacearum.* Susceptible trees expressing oak wilt symptoms often die within a few months after symptoms first appear, and some highly susceptible oaks may succumb within weeks. Early foliar symptoms of the disease are characterized by leaves that turn dull green, yellow, or bronze, followed by marginal and apical leaf browning. Veinal necrosis is a diagnostic symptom in southern live oaks. These foliar symptoms usually progress inward and downward from the outer and upper limbs of the crown until all foliage is dead. In advanced stages of the disease, leaf drop of symptomatic leaves may result in defoliation of the crown, followed by branch mortality and death if the tree is unable to refoliate. Suckers often develop on the bole and on larger limbs in the same year or in the year following defoliation as apical dominance is lost because of the death of growing tips. The fungus may spread from infected trees through root grafts into healthy adjacent trees to create expanding infection centers. Root grafts mostly form between oaks of the same species, and red oaks form root grafts more commonly than do white oaks. Root grafts are very rare between red oaks and white oaks.

In the southern states, a number of red oak species are highly susceptible to oak wilt. Susceptible stands may be divided into coastal, upland, and bottomland forest types. The large coastal populations of live oak *(Q. virginiana),* often planted as landscape trees with water oaks *(Q. nigra)* and other red oaks in coastal communities, are at considerable risk. Within interior upland stands, black oak *(Q. velutina),* northern red oak *(Q. rubra),* scarlet oak *(Q. coccinea),* southern red oak *(Q. falcata),* blackjack oak *(Q. marilandica),* laurel oak *(Q. laurifolia),* and turkey oak *(Q. laevis)* are susceptible species. Inland bottomland species such as cherrybark oak *(Q. falcata var. pagodifolia),* Nuttall oak *(Q. nuttallii),* Shumard oak *(Q. shumardii),* water oak, and willow oak *(Q. phellos)* are equally susceptible. Any incidental introduction of *C. fagacearum* into hardwood stands containing high densities of these species or having high propor-

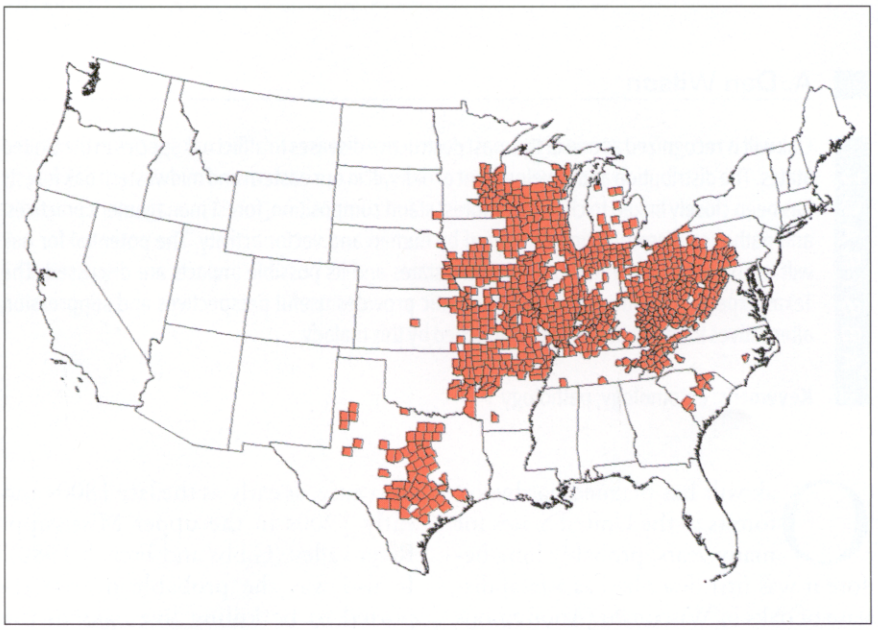

Figure 1. **Distribution of oak wilt incidence by county in the United States as of December 1999. Shaded counties have confirmed cases of oak wilt where *C. fagacearum*, the oak wilt fungus, was isolated in pure culture from oak trees with diagnostic symptoms.**

tions of total basal area consisting of susceptible red oaks will likely sustain heavy damage. The creation of new infection centers jeopardizes adjacent stands for years.

Five major tree species in California probably would be at greatest risk if oak wilt were introduced into that state. Four common red oak species found in low-elevation mixed evergreen forests, foothill woodlands, and southern oak woodlands of California would likely be most susceptible to oak wilt. These include three semi-evergreen live oaks, interior live oak *(Q. wislizenii)*, coast live oak *(Q. agrifolia)* and canyon live oak *(Q. chrysolepis)*, and California black oak *(Q. kelloggii)*. In addition, tanoak *(Lithocarpus densiflorus)* is susceptible to oak wilt. All of these species occur over a large range and account for significant proportions of basal area in inland hardwood forest stands of California. In some forests and foothill woodlands, specific species compose nearly pure stands.

Introduction and Spread

Human activity is arguably the most important means by which the oak wilt fungus has been introduced into new areas. Harvesting firewood from dead trees killed by oak wilt is a common and compelling way to use otherwise unmerchantable wood. But when infested firewood is transported to other areas, there is risk of spreading infection. Firewood often contains masses of the fungus (fungal mats) that form under the bark of diseased red oaks. Insect vectors that often breed and form galleries in the wood are attracted to the sweet aroma of these mats and pick up spores produced on these structures, which they carry to wounds on healthy trees and thus initiate new infections. Tree-pruning in urban areas creates wounds that increase the susceptibility of trees to insect transmission, particularly during spring months when insect activity is highest. New outbreaks of the disease around homes often are traced to piles of insect-infested firewood with active fungal mats.

Because the harvest and transport of oak firewood from oak wilt–endemic areas across county and state lines is not controlled by quarantine regulations, *C. fagacearum* may be spread freely in this manner. The long-distance dissemination of the oak wilt fungus similarly may occur directly by various insect vectors carrying spores from fungal mats of unharvested dead trees. Oak sap beetles (Nitidulidae) and bark beetles (Scolytidae) have been the most implicated in insect transmission, although these insects generally are considered inefficient vectors. Other contributors to vector transmission could include root-feeding and bark-feeding insects, rodents, and woodpeckers that cut into and expose sapwood to potential inoculum sources. The movement of oak wilt–infected oak planting stock to new areas is another possibility; raising oak nursery stock in outside beds provides opportunities for trees to become infected prior to commercial sale.

Once introduced, the fungus can spread vegetatively through root transmission and by repeated cycles of sporulation on dying red oaks and dispersal by insect vectors and human activity within the new area. The rate of root transmission largely depends on stand density, species composition, soil depth, and the tendency of oak species to form root grafts.

Regional Factors

The southward progression of oak wilt into the southern tier of states along the Gulf Coast from Louisiana to Florida has been hindered by the existence of mixed hardwoods and hardwood-conifer stands. Mixed, high-diversity forests create space barriers that break potential avenues for root transmission of the pathogen, and inhibit expansion of infection centers through root grafts between diseased and uninfected trees. For this reason, oak wilt is not likely to have the same impact on the other southern states that it has had in Texas. However, forest stands altered by land-management practices in these areas, resulting in more homogenous stands of highly susceptible red oaks, are vulnerable to the disease.

Indeed, the most severe oak wilt epidemics have occurred in stands with reduced species diversity. This was the case in both the upper Mississippi Valley and in Texas, where highly susceptible oak populations developed as a result of selective logging, thinning operations, and land management practices (Appel 1995). A similar scenario was observed in the aftermath of the Dutch elm disease and chestnut blight epidemics that eliminated elms and chestnuts as dominant species in many of our eastern deciduous forests. The removal of these species created empty niches that were largely filled by susceptible red oaks. This transition in species composition helped create hardwood forest stands vastly more susceptible to oak wilt. The oak wilt fungus quickly took advantage of this shift in stand composition, as evidenced by the continuous rise in oak wilt incidence and expanded distribution throughout the eastern United States in the second half of the 20th century.

The situation is different in California. Accidental introduction of the oak wilt fungus is considerably less likely there than in the South because of geographical isolation and the remoteness of susceptible trees to infection centers in midwestern states. Nevertheless, the recent introduction of a *Phytophthora* species, a pathogenic fungus that causes a fatal new disease known as "sudden oak death," is responsible for devastating losses to coast live oak, tanoak, and California black oak populations in coastal forests of central and southern California. The unthinkable can and sometimes does happen; for example, the situation would change dramatically if a population of *C. fagacearum* were to become established in this region. The abundance of often pure stands of tanoak and live oaks in many coastal foothill woodlands could create conditions similar to the pure stands of live oaks in central Texas. In addition, high tree mortality would create conditions favorable for catastrophic fires, and this combination could cause significant alterations in the landscape. This scenario is already expected to occur with the current *Phytophthora* epidemic.

Regional climatic and environmental factors such as rainfall, ambient temperatures, relative humidity, soil texture, and soil depth can have dra-

How to Detect and Manage Oak Wilt

Detection

Red oaks usually show rapid leaf discoloration and wilting. Often the initial symptom is a subtle off-green color shift that may be visible in the upper portion of the tree crown. This symptom is apparent in the northern part of the disease range in late June to early July. Shortly after this initial color shift, the leaves begin to wilt from the top of the crown downward. As the disease progresses, individual leaves quickly discolor, taking on a "bronzed" appearance. The discoloration progresses around the margins of the leaf from the tip to the base. The progressing discoloration may be interrupted by the leaf veins or may affect the entire upper portion of the leaf. Leaves are cast rapidly as the infection progresses; in most cases, infected trees are almost entirely defoliated within a few weeks of symptom onset. Fallen leaves usually are brown at the tips and margins, and sometimes green at the base and along the lower veins.

Occasionally the outer ring of vessels of diseased trees will be plugged with a brown substance that may be visible in cross sections as a ring or a series of dark spots through the outer sapwood, and in tangential cuts as longitudinal streaking of wood exposed after removing the bark. Discoloration is most readily seen in tangential cuts on branches.

White oaks usually die slowly, one branch at a time, over a period of years. Wilting and death of leaves on individual branches occurs in a similar fashion to that of diseased red oaks, but usually progresses much more slowly. Affected leaves exhibit a pattern of discoloration similar to that seen in red oaks, with discoloration proceeding from the margins to the base, sometimes interrupted by the leaf veins. Brown streaking in the outer growth rings is often readily apparent, even to an untrained observer, in infected white oaks and bur oaks; however, this streaking may not occur in all diseased trees.

Texas live oaks can wilt and die rapidly or slowly, depending on the timing of infection and weather conditions, but generally succumb from one to six months after infection. Diagnostic leaf symptoms are usually produced somewhere on the tree, especially in spring and fall. Leaves develop yellow veins which eventually turn brown, a symptom known as veinal necrosis. Affected leaves fall, and the tree crown progressively thins out until the entire tree is dead. Fallen leaves under the tree may show darker brown veins for months. Sometimes just the tips, margins, or interveinal portions of leaves will turn yellow or brown, but these symptoms are not necessarily the result of oak wilt and are not as useful in diagnosing the disease. A small percentage of Texas live oaks may survive oak wilt infection indefinitely, while suffering varying degrees of crown loss.

Prevention

Remove infected trees. Trees that are infected with or have died from oak wilt should be removed and properly treated to prevent development of spore mats. These treatments include debarking, chipping or splitting, and drying the wood. Covering dead wood with plastic, burying the edges for six months, and then air-drying for another six months will kill the fungus and any associated insects. Trees that die in summer should be removed and treated before the following spring, when new fungal mats can develop. If the wood is sufficiently dried, however, fungal mats will not develop. A word of caution: Removing a diseased tree that is still living may actually spread the infection by accelerating the movement of the fungus into adjacent trees that are grafted to it by the roots. To avoid this problem, disrupt interconnected roots before removing diseased living trees.

Avoid injury to healthy trees. It is important to avoid injury to oaks during favorable conditions for infection, which in the North occur in spring and early summer, when fungal mats are present and the beetles are flying. Favorable conditions usually occur between April 15 and July 1 in the Lake States, and over a correspondingly longer period of time to the south. In Texas, avoid damage to oak trees from February through June.

If construction activity or pruning are unavoidable, or if storms injure oak trees during the critical period, the wounds should be treated immediately with a commercial tree paint or wound dressing. In the North, if trees are wounded during the dormant season tree paints are not necessary, but judicious use during the rest of the year is acceptable. From Missouri to Texas, tree paint should be used immediately after trees are wounded, regardless of the time of the year.

Control

Disrupting root connections between infected and healthy trees through trenching limits the spread of oak wilt, and is an effective control measure. Trenches are usually cut completely around infection centers. Because the oak wilt fungus usually has already moved through roots in advance of visible symptoms, a buffer zone is required between the visible edge of the infection center and the trench. The size of the buffer zone is determined by the rate at which the fungus is known to spread through the grafted root system of a given oak species. For example, in Texas live oaks, the oak wilt fungus is known to move at a rate of 75 to 100 feet or more per year in advance of symptomatic trees, and therefore a 100-foot buffer zone is used. In oaks of the upper Midwest, the fungus usually moves at less than 50 feet per year, so the buffer zone can be smaller. In either case, unless infected and healthy trees are already spaced at a distance wide enough to serve as a buffer zone, a trench is usually cut between healthy trees at the outer edge of the buffer zone to sever root grafts between them before the fungus gets there. Interconnected root systems can be disrupted with a trencher, vibratory plow, backhoe, rock saw, or other equipment, depending on soil type.

For Further Information

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Table 1. Allocation of US Forest Service cooperative pest-suppression funds, FY 2000.

Pest or disease	Funding allocation ($1,000s)	Percent of total allocation
Insects		
Gypsy moth	3,803	64.27%
Mountain pine beetle	1,076	18.18
Southern pine beetle	315	5.32
Hemlock woolly adelgid	116	1.96
Ips beetles	42	0.71
Spruce beetle	10	0.17
Total	5,362	90.61
Weeds		
Miscellaneous alien plants	193	3.26
Miconia	85	1.44
Pennisetum	7	0.12
Total	285	4.82
Fungi		
Oak wilt disease	270	4.56

NOTE: Cooperative federal funds allocated to state governments are matched by state funds to support pest-suppression projects administered by affiliated state forestry agencies. Funding usually is allocated at a percentage of the requested funding level.

matic effects on oak wilt disease development. Hot, dry conditions generally exacerbate symptom development in individual trees; additional stress is placed on trees already suffering from water deficit caused by the disease. Areas with more rainfall, higher humidity, and cooler temperatures, however, favor fungal mat formation and increased insect activity that may result in the creation of new infection centers at faster rates. Shallow soils facilitate root grafting by concentrating roots into a narrow soil profile; sandy soils promote drought stress in susceptible trees because of reduced water retention. Increases in stand density also can promote root grafting in relatively pure stands of susceptible oaks.

Forecast for Oak Wilt in the South

The question is not whether oak wilt disease will eventually occur at visible levels in the oak forests of the southernmost states, but rather when it will occur. In fact, it is very likely that the disease already exists in this region. Several examples of its possible imminent incursion into the area are known. The disease was positively identified in a live oak population in Houston in 1993. This represents an expansion of the disease into the valuable live oak population that extends along the Gulf Coast and lower Atlantic coast north to Virginia (Appel 1995). Similarly, in 1998 it was detected in live oaks in Aiken County, South Carolina, the first known occurrence of the disease on this host outside of Texas (USDA Forest Service 1999). The disease is consistently reported in annual disease surveys of several Arkansas and Tennessee counties near or bordering northern Mississippi, but no confirmed reports have been made in that state.

The oak wilt fungus probably does exist in small, sporadic populations in southern oak forests but has not yet built up to sufficient levels, other than in Texas, to become readily visible. This was the situation in American hardwood forests during the first half of the 20th century; by the time oak wilt disease was recognized, it had already become well established in many eastern states.

Another problem lies in recognizing the disease. A number of biotic and abiotic factors (e.g., drought stress, lightning strikes, nutrient deficiencies, salt damage, soil compaction, bacterial leaf scorch *(Xylella fastidiosa)*, and some herbicides—can induce oak wilt-like foliar symptoms in oaks. In addition, *C. fagacearum* may actually be self-limiting early on, after initial colonization of mixed hardwood stands, by rapidly killing the small number of available hosts and thus limiting further dispersal.

Suppressing Oak Wilt in Texas

Texas currently has the distinction of being the southernmost US state affected by oak wilt. The Texas experience with the disease shows the complexities of the problem and demonstrates the devastating potential of this disease to damage oak forests.

The first observations of oak wilt disease in Texas were probably recorded in the 1930s in the central Texas hill country. At that time, unusually high live oak mortality was reported in the Austin area (Taubenhaus 1934, 1935). This mortality continued over the next 40 years, resulting in widespread damage that received considerable attention (Dunlap and Harrison 1949; Halliwell 1966). It appears that populations of the oak wilt fungus slowly developed during that period until they reached a critical mass by the late 1970s, culminating in the epidemic that rages today. Apparently, the disease was already well established in that region by the time the causal agent was identified (Lewis 1977). Consequently, the significant buildup of *C. fagacearum* populations in susceptible oak stands presented a formidable task for the disease suppression program that was established a decade later.

Currently, oak wilt is causing increasingly devastating losses to oaks in Texas; total cumulative damages, primarily owing to reductions in property values and costs for dead tree removal, have been estimated to be in the hundreds of millions of dollars statewide (Wilson and Forse 1997). Oak wilt has now been confirmed in 60 Texas counties, although most of the damage attributed to the disease is occurring within a high-risk 22-county area extending from San Antonio to Dallas (Texas Forest Service 1998, pers. commun.).

Texas is the most recent state to develop a comprehensive oak wilt suppression program in the United States. The need for federal assistance to combat the disease in Texas was realized early in the process. Two projects were initiated to address the problem with cooperation among the USDA Forest

Table 2. Incidence, severity, and impact of oak wilt disease in Texas, 1988–2000.

Year	Counties affected[1]	Cumulative confirmed infection centers	Cumulative estimated acres affected in infection centers[2]	Biannual estimated damage[3] ($1,000s)	Cumulative total estimated damage[4] ($1,000s)
1988	35	362	1,118	645	48,653
1990	46	714	2,050	1,225	49,878
1992	51	1,261	5,962	4,892	54,770
1994	54	2,084	9,133	7,337	62,106
1996	55	2,781	11,456	8,229	70,335
1998	59	3,285	12,621	15,746	86,081
2000	60	3,903*	14,347	16,083	102,165

[1]Includes only those counties with confirmed cases of oak wilt as determined by isolation of the oak wilt fungus from symptomatic trees and identification in pure culture.
[2]Tree values range from $750 per acre in rural, nonresidential areas up to $60,000 per acre in urban settings (McKinney and Billings 1995).
[3]Damage estimates are based on total expenditures for oak wilt control, the percentage of infection centers treated, an average cost:benefit ratio of 1:6, and the assumption that 80 percent of the trees within trenched infection centers died of oak wilt.
[4]Cumulative estimates take into account only damage that occurred within confirmed infection centers. Cumulative damage for 1988 includes estimated losses that accrued within infection centers during the years prior to and including 1988.
**Estimate based on detection rates in previous years.*
NOTE: All values indicate estimates of documented damage only from ground surveys of confirmed oak wilt centers, and probably represent less than 10 percent of the damage that would be detected if unlimited resources made it possible to accurately determine estimates for all areas affected in the state.

Service, the Texas Forest Service (TFS), and the Texas Agricultural Experiment Station. The Texas Oak Wilt Demonstration Project, administered by TFS and funded in part by USDA Forest Service Forest Health Protection (FHP) from 1982–87, evaluated the distribution, severity, and rate of expansion of the disease, as well as pilot-test control strategies. The Texas Oak Wilt Suppression Project (TOWSP) was funded beginning in 1988 by a combination of FHP funds and matching state funds. Federal funding for TOWSP accounts for about 4.6 percent of the FY 2000 cooperative pest suppression allocation in the United States *(table 1)*.

Although federal research on the oak wilt problem in Texas has diminished, federal funding for oak wilt suppression in the state continues; FY 2000 cooperative funding for TOWSP represents the 13th year that federal assistance has supported oak wilt suppression efforts in that state. Other states received federal assistance for oak wilt suppression some years ago, but Texas is now the only state that continues to receive suppression funds because of the continuous devastating impact of the disease. In fact, TOWSP is the only cooperative disease suppression program in the country receiving federal assistance.

TOWSP maintains a computerized database of suppression activities and results using ground survey data from confirmed disease centers, primarily in central Texas. In addition, unofficial aerial surveys, intended to provide tentative, broader regional perspectives of disease impacts, were conducted over more than 16 million acres from 1996 to 2000. These surveys have recorded more than 7,000 mortality centers throughout the region. Although many of these mortality centers may be and probably are caused by oak wilt, many are not confirmed by ground crews and thus include mortality from other natural causes, such as recent extended droughts occurring over several consecutive years. Consequently, the values presented here from ground surveys of infection centers are the most accurate numbers available to document the progression of the disease. However, the values only represent the portion of the problem addressed by TOWSP, which is probably less that 10 percent of the disease that would be recorded if sufficient resources were available to accurately measure it statewide. Nevertheless, the numbers effectively illustrate the proportional increase in impact of the disease on oaks in Texas.

A chronological summary of the incidence and impact of oak wilt in Texas since inception of TOWSP demonstrates the dramatic progression and devastating potential of oak wilt *(table 2)*. Between 1988 and 2000, the number of confirmed infection centers increased almost 1100 percent and the area affected within centers increased from 1,000 to more than 14,000 acres. The estimated rate of damage accumulation within infection centers nearly doubled every two years from 1988 to 1994. The damage estimates are based on the total expenditures for control, the percentages of centers treated, a cost:benefit ratio of 1:6, and the assumption that 80 percent of trees within centers will die of oak wilt (Gunter and Billings 1996).

For the benefits of controls, it is further assumed that infection centers are roughly circular in shape, that the centers will continue to expand for five years at a radial rate of 75 feet per year in the absence of controls, and that the controls will stop the spread of the disease during that time. The benefits are defined in terms of the value of trees protected computed from the estimated acreage affected and the tree value per acre from each land-use category, as well as the costs avoided for removal of dead trees and replacement costs. Cumulative total damage takes into account all economic losses that had occurred within known infection centers to date through 1988 and additional losses that were added every two years after 1988. The area within an infection center is defined as the land area encircled by a control trench. Four land use categories—urban, suburban, rural residential, and rural nonresiden-

Table 3. Summary and costs of oak wilt suppression efforts in Texas, 1988–2000.

Year	Cumulative percentage of confirmed oak wilt centers trenched	Cumulative length of trenches installed[1] (1,000 feet)	Cumulative number of infected red oaks removed	Cumulative number of oaks injected with Alamo[2]	Cumulative cost of suppression[3] ($1,000s)
1988	3.3	15.6	74	0	336
1990	15.7	140.4	142	77	1,630
1992	33.4	677.3	1,336	2,135	3,092
1994	40.0	1,291.7	1,939	5,862	5,178
1996	44.0	1,836.5	2,157	7,188	6,444
1998	44.8	2,134.7	2,796	7,234	8,036
2000	45.3	2,506.5	3,042	7,234	9,636

[1]Cumulative total linear feet of trench installed to control root transmission from infected trees within infection centers.
[2]Alamo® was the only registered fungicide used by TOWSP (Texas Oak Wilt Suppression Project) personnel to control oak wilt.
[3]Cumulative total costs for oak wilt suppression by the Texas Forest Service are based on expended funds from a combination of sources including: 50 percent from the federal government through the USDA Forest Service (budgeted funds only, exclusive of supplemental year-end money), 28 percent from the State of Texas, 19 percent from private landowners, and 3 percent from the City of Austin. Costs include direct suppression costs, operating expenses, administrative overhead, staff salaries, and employee benefits.

tial—determine the average value of trees per acre used to convert area to dollar values.

The major control strategies used by TOWSP have included trenching, removal of symptomatic red oaks, and tree injections with fungicide. The oak wilt fungus commonly moves from infected trees to adjacent healthy trees through root grafts or fused common root systems. To prevent root transmission, trenches are cut into the soil to a depth of 3 to 5 feet completely around infection centers to sever root graft connections at least 100 feet in advance of symptomatic trees. Trenching continues to be the most effective and predominant method used to control the disease. Over 2.5 million feet of trench was installed through 2000, treating about 45 percent of confirmed infection centers *(table 3)*. Trenches on average have been about 70 percent successful in stopping further spread of infection centers since the inception of TOWSP (Texas Forest Service 1999, pers. commun.).

Red oaks generally have been considered a main source of inoculum from which new infection centers are started by insect vectors. The sticky, sweet-smelling fungal mats of *C. fagacearum* that form on infected red oaks are visited by nitidulid sap beetles, which are believed to transmit inoculum from these mats to wounds on healthy trees. More than 3,000 infected red oaks were removed in hopes of slowing the formation of new infection centers, although red oak removal has not yet been demonstrated to significantly affect disease epidemiology in Texas. However, red oak removal apparently has helped in some states. Injections of more than 7,000 high-valued trees with a systemic fungicide (Alamo®) saved some trees from further decline, but in many cases only delayed the disease process. TOWSP discontinued fungicide injections in 1997 because they were no longer considered cost-effective.

Despite the inefficiencies of the control methods, the program has resulted in significant benefits well worth the costs incurred. The total expenditures for all suppression activities totaled $8 million as of December 1998 and provided estimated benefits of $48 million solely in saved costs for tree removal and replacement (Texas Forest service 1998, pers. commun.).

A number of problems have been associated with developing and implementing an effective oak wilt control program in Texas. Some of the key problems include the large buildup of the disease before a control program was initiated, the late diagnosis of the disease (not recognized until 1977), the learning curve associated with implementing suppression measures, and problems with specific control methods. At this time, however, the most immediate problem is insufficient resources. The TOWSP control program simply is not large enough to handle the problem. With the current approach, it is estimated that the program is treating only 150 to 200 new oak wilt centers each year (Texas Forest Service 1999, pers. commun.). However, an estimated 250 to 400 new centers are confirmed each year. At the current rate of treatment, the program will not be able to keep up with the growth of the epidemic. Therefore, either a different approach must be taken to more effectively leverage available funds for more efficient controls, or more resources must be allocated to contain the epidemic.

Conclusions

The increasing oak mortality caused by oak wilt is a grim reminder of the potential damage that virulent pathogens can inflict on hardwood forests. Although oak wilt currently occurs only in the United States, there is concern that it could spread to other countries. The destructive potential of this disease has been recognized internationally for many years because of the threat it poses to oak resources in Europe and Asia (Balder 1991; Pinon et al. 1995). Quarantine regulations restricting the export of unfumigated oak lumber and logs to Europe and Asia were established soon after the disease was discovered here. Unfortunately, efforts to curtail the importation of exotic pests into the United States, although well grounded and important, have shifted attention and emphasis away from endemic virulent pests such as *C. fagacearum* that have the capacity to devastate forests, both domestic and foreign, with equal voracity.

The threat to red oaks in the United States is equally significant. The presence of many highly susceptible red

oaks in the southern states and in California makes it highly probable that the vast oak resources in these regions will eventually become exposed to oak wilt. The damage to uniform, low-diversity red oak stands could be severe. Since the oak wilt fungus is considered potentially the most destructive of all forest pathogens (Young 1949; Gibbs and French 1980), any threat of its expansion into oak forests in unaffected areas should be taken seriously.

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