IDENTIFICATION AND CONTROL OF OAK WILT IN TEXAS URBAN FORESTS

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Abstract. Oak wilt, caused by *Ceratocystis fagacearum*, annually causes losses of epidemic proportions in towns and cities throughout central Texas. Live oaks (*Quercus fusiformis* and *Q. virginiana*) are the most seriously affected *Quercus* spp. because of their prevalence and subsequent high values. Oak wilt epidemiology is influenced by host characteristics, climate, and reproductive traits of the pathogen. These factors have been studied to develop the key elements of a comprehensive, integrated oak wilt management program. Under most conditions, this program can be used in Texas to successfully minimize losses. The program relies on disruption of root connections, intravascular injection with propiconazole, proper pruning and wound treatments (including dressings), and various sanitation practices. Preventative measures are the most effective means of reducing losses, making prompt, reliable diagnoses of new oak wilt infection foci essential. A five step protocol is recommended to distinguish oak wilt in live oak from other diseases in Texas and facilitate disease control. Cooperation among federal, state, and local agencies working with commercial arborists has had a dramatic impact on public understanding of oak wilt and resulted in significantly controlling the disease.

The foliar symptoms of oak wilt, caused by *Ceratocystis fagacearum*, on semi-evergreen live oaks (*Quercus fusiformis* or *Q. virginiana*) in Texas differ from symptoms on diseased, deciduous oaks (1,14). In addition to symptoms, there are other differences between semi-evergreen and deciduous oaks that account for unique patterns of spread by *C. fagacearum* in live oaks (4,8). These differences influence the options available for disease control in live oaks when compared to traditional oak wilt management in the deciduous oaks (3,18).

Of the 22 states with oak wilt, only Texas has epidemics in live oak populations. Native live oaks, along with planted live oaks, account for the most valuable and largest proportions of trees in the urban forests of central Texas. Oak wilt management in live oak has been complicated by diagnostic difficulties as well as an incomplete understanding of pathogen transmission. Further complications in disease management arise in urban forests, when compared to rural live oak savannahs, by site disruptions and the sacrifices of apparently healthy trees needed to effectively contain the pathogen.

Oak wilt control has traditionally relied on interrupting two stages in the life cycle of the pathogen. The first stage involves inoculum production on the tree and subsequent transmission of fungal spores by insect vectors. This means of transmission has been called overland spread, and depends on the occurrence of fresh wounds as infection courts for inoculation by the insect vectors (16,24). Additional details on insect transmission are discussed below. The second critical stage for the pathogen involves vegetative growth in xylem tissues and movement of fungal spores through root connections between diseased and healthy trees. This transmission has been called local spread, and references distances of 50 ft. or less (30). Numerous strategies to address these means of transmission were developed during 40 years of research in the Mid-Atlantic and North Central U.S. (12,15,30). These same strategies also form the basis for oak wilt management in Texas. However, the growth habit of live oak and the composition of the oak forests in Central Texas require that adaptations and refinements be made to those traditional strategies to successfully reduce tree losses (23).

The majority of research in the Forest Pathology Laboratory at Texas A&M has focused on comparative epidemiology of oak wilt in Texas and occurrence of the disease elsewhere. The results of this research were then used to modify management programs from other states, or in some cases, develop new approaches to control the disease. Comprehensive disease control efforts are now underway, especially in urban areas

where trees can contribute as much as 13-19% of the tax appraised values of residential property (25) (Figure 1). The following discussion will highlight the diagnosis of oak wilt in live oaks and current recommendations for disease control in Texas.

Host Influence on Pathogen Transmission

Live oaks are superior landscape trees for urban environments in Texas. They grow on a wide variety of soils, tolerate wide temperature extremes, and thrive under varying levels of annual precipitation (13). The foliage on live oaks is dense and semi-evergreen, providing excellent shade and cover during all months of the year except March, when defoliation occurs. Live oaks reach maximum heights of 60 ft., and have large lower branches and spreading crowns. Overgrazing, fire control, and selective management have contributed to the formation of large expanses of nearly pure live oak stands throughout the woodlands of central Texas. Live oaks tolerate the site disruptions associated with urban expansion very well, so that existing native trees usually comprise the most popular shade tree in central Texas towns and cities. These native stands growing in lawns and boulevards are commonly supplemented with planted live oaks. In towns such as Austin, San Antonio, Waco, Ft. Worth, Dallas, and hundreds of smaller communities, the

Figure 1. Impact of oak wilt with loss of live oaks on urban property in Austin, TX.

proportion of live oaks may far exceed 10%, the maximum level recommended to maintain a stable, healthy urban forest (17).

The abundance of live oaks in the native plant community relates in part to their habit of vegetative reproduction. Prolific root sprouting is a common trait of live oaks (26). Thousands of nuisance root sprouts are commonly observed in lawns and plantings. This characteristic is considered to be a selective advantage for trees colonizing disturbed sites under harsh environmental conditions (26). Many connections among trees are believed to be maintained through maturity. This development of a common root system, connecting numerous ramets as a clone, makes them highly vulnerable to a virulent, vascular inhabiting fungus such as C. fagacearum. Presumably, root grafts are common on the thin soils and further contribute to the risk of infection by a vascular parasite. Once the pathogen is introduced into a stand of live oaks by the insect vector, the focus of mortality expands rapidly from one tree to the next creating an ever growing patch of hundreds, and sometimes thousands, of dead and dying trees (8).

Sap-feeding nitidulid beetles (Coleoptera: nitidulidae) are suspected as primary vectors for overland transmission of C. fagacearum in Texas (6,7). The same group of insects have been also implicated as primary vectors in other states (16). However, the role of nitidulids in Texas is somewhat different. To obtain inoculum, nitidulids require formation of a fungal mat by the pathogen beneath the bark of diseased trees (16,24). Fungal mats attract the beetles with a strong, sweet odor and are covered with sticky spores which easily contaminate the insects when they feed and breed. Mats form on members of the red oak group (subgenus Erythrobalanus); they do not form on diseased white oaks (sub-genus Leucobalanus). In the Mid-Atlantic and North Central U.S., most diseased trees are potential mat formers because the primary hosts in those regions are red oaks. In contrast, mats have never been found on diseased live oaks, the predominant host in Texas. The opportunity for long distance spread is therefore limited in Texas by the proportion of red oaks growing in a disease center. The two most prevalent red oak species in Central Texas are Spanish oak

(*Q. texana*) and blackjack oak (*Q. marilandica*). These species are extremely susceptible to *C. fagacearum*, but they do not commonly comprise a large proportion of the central Texas oak savannahs.

Diagnostic Protocol

Few other live oak diseases resemble oak wilt. Herbicide damage may mimic the foliar symptoms and patterns of mortality, but herbicides are not species-specific and can usually be distinguished from oak wilt with little effort. Absolute certainty is necessary in diagnosing any destructive tree disease, especially when expensive management programs are to be implemented. A five step diagnostic protocol is recommended for routine tree diagnostics in central Texas. Although designed to distinguish oak wilt from other diseases, the protocol has proven useful in diagnosing any tree problem. The completion of all five steps is unnecessary to make an accurate diagnosis and may be impossible. The five steps are: 1) observe the pattern of dieback and mortality in time and space for the tree population, 2) observe the patterns of dieback and mortality in time and space for individual tree crowns, 3) examine specific organs, e.g. leaves, branches, or roots, for unique patterns of chlorosis and/or necrosis (symptoms), 4) examine specific organs for evidence of the pathogen (signs), and 5) attempt to obtain laboratory isolation of suspected pathogens. An analysis of these five steps also provides an excellent tool for better understanding oak wilt. A brief description is provided below for each of the steps.

Pattern of tree mortality at the population level. An expanding patch of dead and dying live oaks spreading from diseased trees to adjacent, healthy trees on the perimeter is a familiar characteristic of oak wilt in Texas. Sequential, aerial surveys of the disease in large, homogenous live oak stands in rangelands have been useful for estimating fungal spread (8). Typical expansion rates of 14 - 16 meters/yr. were recorded. The fungus is able to spread similarly in urban trees, in spite of the expected obstructions resulting from construction in the native stands. In Figure 2, oak mortality can be followed over a 5 year period for

a disease center in an Austin, TX, neighborhood designated as Mesa I. The initial infection probably occurred at the northern corner of the block in 1981. Tree crowns in Figure 2 are comprised only of live oak. The urban infrastructure for Mesa I is also depicted, showing how *C. fagacearum* may spread under sidewalks, driveways, construction slabs and sewer lines. The average rates of spread, sizes, and relative densities of live oak for Mesa I and other disease centers are given in Table 1. Relative density refers to total crown cover divided by total area of the focus in 1987. The relative densities for all four disease centers were comparable, but the rate of spread observed in Mesa I was the least of the four. Urban development may influence the occurrence of root grafting and common root systems and in turn reduce opportunities for root transmission. The mortality in Mesa I expanded to affect 12 property owners, comprising 3,280 m^2 of crown cover and 1.26 ha before steps were taken to minimize further losses.

In Texas, the application of soil sterilants is a popular method for controlling weeds in the landscape. As is the case with foliar symptoms, the effects of these compounds are sometimes confused with oak wilt because they may result in expanding patches of tree mortality. Eventually the patch ceases to expand, in contrast to the inevitable spread of *C. fagacearum* into adjacent live oaks on the focus perimeter. Other than herbicides, there are no known disease-causing agents that resemble the typical oak wilt pattern in a live oak population.

Rates of individual tree death. Unlike the distinct contrast between red and white oak susceptibility, live oaks exhibit an intermediate response to infection by *C. fagacearum*. Live oaks do not all die at the same rates (1). Most are extremely susceptible and will die within 3 - 6 mos. following infection. However, a small proportion (15 - 20%) of live oaks in an infected stand will survive with varying levels of crown loss (8). These trees appear to survive indefinitely, but do not readily recover to become specimen landscape trees. Live oaks debilitated by oak wilt may further decline as a result of infection and attack by non-aggressive fungi and insects. However, they do not normally develop new symptoms of oak wilt

after 2 - 3 yrs., and *C. fagacearum* cannot be isolated from their tissues. Following defoliation, the terminals never resprout, as trees often do when affected by herbicides.

Red oaks cannot be expected to live 4 - 6 weeks beyond the initial appearance of symptoms. Their crowns succumb rapidly to the disease and they only survive to the following growing season if the infection occurs in late summer or early fall. In contrast, white oaks are resistant to the pathogen, rarely die, and usually recover with little crown loss.

Patterns of foliar symptoms. Detailed documentation has revealed a variety of foliar symptoms in live oaks affected with live oak (4,5). A few of the symptoms are considered diagnostic for the disease, but their occurrence is transitory. The most reliable symptom involves a distinct chlorotic (yellowing) and necrotic (browning) of the mid-vein and lateral veins on the leaf, called veinal necrosis. Following rapid defoliation, these leaves may be found under the tree for months with discernable brown veins. When present, it is considered to be a strong indicator of oak wilt and appears to reflect an advanced stage of the disease; branches exhibiting this symptom inevitably die.

Other symptoms may also be found throughout a population of live oaks affected by oak wilt. These include tipburn, interveinal chlorosis (yellowing), and wilting of young tissues during early spring. At times, there may be general foliar chlorosis and necrosis with no discernable patterns. Combinations of all the various symptom types are often found on the same tree, or even the same branch. No seasonal trends have been documented, but the symptoms appear to be more prevalent during those times when the fungus is most active, such as the cooler months of spring and fall. Diagnostic foliar symptoms are most likely found on trees on the perimeters of disease centers where the fungus is actively colonizing new trees, rather than on the debilitated survivors on the interior of the disease center.

Symptoms on deciduous red oaks, i.e. Spanish and blackjack oaks, are similar to those exhibited by red oaks in other states. Leaves on diseased trees first become dull green, curl upward, and eventually turn chlorotic or bronze from the apex and margins inward (15,30).

Presence of signs of the pathogen. Fungal mats are the only signs of oak wilt produced by *C. fagacearum*, and are considered to be unquestionable indicators of the disease. However, there are severe limitations in relying on mats for routine diagnosis. Most importantly, they do not form on live oaks, nor do they form on diseased white oaks (7). Those red oaks that form mats must be in the proper stage of colonization by the fungus. Early season infections (April - June) do not result in mat formation because wood of the tree is too dry by the time conditions become conducive to formation. Also, late season infections (August - September) may not be sufficiently colonized by the pathogen and will resprout the following season. The crowns of potential mat-forming red oaks will be entirely symptomatic in September, but must have green cambial layers going into winter. Due to these requirements, only a low proportion of red oaks affected by oak wilt develop fungal mats.

Mats are thin, cushion-like structures formed on the surface of the sapwood. They range 3 - 4 in. x 6 - 8 in. and are usually elliptical. They have a sweet smell reminiscent of rotting melons and are pale grey or brown. If left undisturbed, the bark over the mat will crack and can be best detected by tapping on the tree to detect a hollow sound. The mats deteriorate within weeks, but can be seen as black stains on the surface of the sapwood for up to a year following formation. Numerous wood-rotting fungi may also cause black "scars" of various shapes and sizes on the sapwood surface and should not be confused with dried *C. fagacearum* mats.

Laboratory isolation of *C. fagacearum*. Isolation and identification of *C. fagacearum* requires a properly equipped laboratory and trained technician. This service is usually provided by a plant disease clinic at a state agricultural college or through a private company. Presumably personnel in those labs will be knowledgeable in the standard culture conditions necessary to isolate a pathogenic fungus. However, there are steps that must be followed during the sample collection procedure that will strongly influence the outcome of the isolation attempt. In the tree, the fungus

colonizes the outermost rings of the sapwood. It is therefore important to collect samples of living sapwood, preferably from trees with active symptom development. Samples can include branches, greater than 1 in. diameter, or strips of sapwood removed directly from the trunk. Following collection, all reasonable precautions should be taken to prevent the tissues from heating to greater than 85 - 90°F. These precautions necessitate placing samples on ice immediately following removal from the tree and storing them in plastic bags. Cool temperatures should be maintained through shipment to the diagnostic laboratory.

Branch samples should always come from portions of the crown exhibiting symptomatic foliage, and should never be dried and discolored. If bole samples are to be taken, a "window" must be cut through the outer and inner bark, exposing a clean sapwood surface. Strips of tissue can then be cut from the outer growth rings with a chisel. Ceratocystis fagacearum is a poor competitor compared to common fungal contaminants found in the inner bark, so successful isolation is favored by obtaining clean sapwood. Care should be taken to avoid including bark in the sample bag. Also, sapwood discolored by prior wounds or borer activity should be avoided due to the likelihood of colonization of these tissues by unwanted fungi and bacteria.

Options For Disease Control

The rapid rate of fungal transmission and tree mortality in Mesa I (Table 1) illustrate the need for prompt diagnosis while oak wilt centers are small and relatively easy to manage. Most oak wilt management practices are preventative; the selection of a suitable practice depends on whether the goal is to prevent overland transmission or local spread through root systems. The affected oak species, topography, tree density, and tree values also must be considered. For example, when oak wilt is being controlled in a stand of live oaks, control efforts can focus on root transmission due to the lack of fungal mat formation and insect transmission. This is best accomplished by creating a "barrier zone" around the perimeter of the disease center. The zone is established by digging a trench to break root connections at a

Table 1. Rates of spread for _Ceratocystis fagacearum_, sizes, and relative live oak densities[x] for two urban and two rural oak wilt centers in Central Texas.

Center	Average yearly spread (m)	Total crown cover (m²)	Relative density
Urban			
Mesa I	7.9 (± 8.0)	3277	0.26
Travis Heights	19.6 (± 9.8)	15413	0.27
Rural			
Monte Verde	30.5 (±32.9)	29658	0.19
Bandera Creek	19.9 (±14.5)	16697	0.28

[x] Relative density refers to total crown cover divided by total area of the focus.

minimum of 100 ft. beyond the outermost symptomatic live oaks located on the perimeter of the disease center. This was the reasoning used for placement of the trench in Mesa I (Figure 2) along Graystone Dr. and Rockpoint Dr. to prevent further spread to the south and southwest. The greater width of Mesa Dr. and recent placement of relatively deep water lines were considered to be sufficient to protect neighborhoods to the north. There are no oaks to the east at risk of root transmission. Trenching should always be to a minimum of 48 in. deep, but greater depths are preferable (Figure 3). Symptomless trees will exist within the barrier zone, some of which are infected but not yet responding. The best approach is to rogue these trees, including stumps, with a bulldozer to further break root connections, but this measure is rarely followed in urban environments due to the site disruption and high tree values. Additionally, several secondary trenches may be dug within the barrier to isolate the diseased, symptomless trees while saving the unidentifiable healthy trees.

In urban environments, barrier trees are usually sufficiently valuable to consider treatment by intravascular injection with the fungicide Alamo®. Alamo® (propiconazole) is an ergosterol-biosynthesis- inhibiting fungicide with several qualities that make it ideal for injecting trees to control
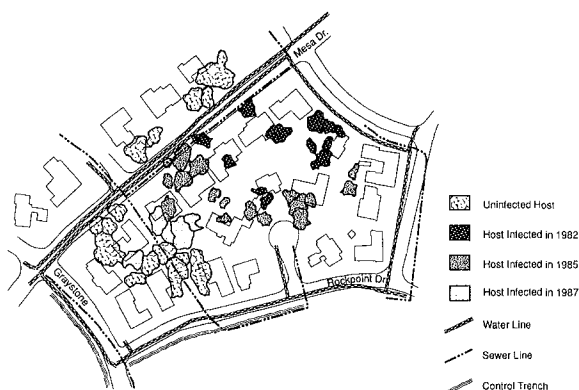
Figure 2. Photo interpretation of an oak wilt center (MESA I) in Austin, TX, made from 1:24,000 scale cole-infrared aerial photography. Tree crowns, houses, streets, and locations of underground utilities are depicted in the drawing. Expansion of the disease center can be seen during the years 1982-1987.

vascular- inhabiting pathogens (9,10,22,29). In addition to being readily systemic with low phytotoxicity, propiconazole also has growth regulator properties that may promote disease control through an influence on tree physiology (11).

Figure 3. Trenching to break root connections for oak wilt control in the urban environment.

Research conducted during 1987 - 1990 demonstrated propiconazole was very effective as a preventative treatment and also had some therapeutic value for live oaks with oak wilt (2,28). A total of 100, native-grown live oaks were involved in the study to compare disease development between treated and untreated trees under natural infection (Table 2). Treated and untreated controls were grouped in 9 plots located in urban and rural oak wilt centers. The plots consisted of groups of trees in yards, or rural stands, measuring no more than 100 ft. across. Six of the plots were complete treatments (no. 3,4,5,6,8, and 9), and 3 were split plots (1A and B, 2A and B, and 7A and B). In split plots, treated trees were intermingled with untreated trees. All trees were located on the perimeters of disease centers, and some plots contained both preventative and therapeutic treatments.

Table 2. Locations, tree attributes and results for plots and sub-plots containing native live oaks used for testing the efficacy of propiconazole for control of oak wilt.

Plot no.[w]	Location	No. trees	dbh[x] (in.)	Crown loss (%)[y]	No. dead
		Injected			
1A	Round Rock	8	11.3	2 (±)	0
2A	N.W. Hills	8	8.3	28(±14)	0
3	N.W. Hills	6	17.3	0	0
4	Camp Mabry	9	16.4	22(±44)	2
5	N.W. Hills	14	19.9	41(±36)	3
6	Camp Mabry	6	25.4	0	0
7A	Comfort	6	12.9	41(±27)	0
		Uninjected			
1B	Round Rock	8	6.1	88(±20)	5
2B	N.W. Hills	8	11.4	61(±34)	3
8	N.W. Hills	6	- [z]	79(±38)	4
7B	Comfort	6	14.0	88(±20)	3
9	Camp Mabry	15	7.0	100	15

[w] Plot numbers labelled with A or B and listed as treated and untreated denote a split plot in which treated and controls are intermingled.
[x] dbh = diameter breast height, as the average for all trees in the plot.
[y] Average crown loss assessment and standard deviation for all trees in a plot.
[z] Indicates no measurement taken.

A standard high volume, low pressure (20 -25 psi), root-flare injection technique was used to inject propiconazole into the trees (Figure 4). As the pathogen colonized root systems and symptoms developed, trees were rated according percentage crown loss from 9 - 36 months following treatment. Survival and crown retention was consistently greater in treated trees than untreated trees (Table 2). The mortality rate in the 57 treated trees was 8% in contrast to 70% in untreated trees. Crown loss ranged from none (Plots 3 and 6) to 41% (Plot 5) in injected trees, compared to a range of 61% (Plot 2B) to 100% (Plot 9) in the untreated controls. Based on these results, propiconazole was labelled under the trade name Alamo® in 1991. Since then, injection has become a routine practice for homeowners and commercial arborists involved in the control of oak wilt.

Although the research results justify recommending injection, the study revealed many limitations to using propiconazole for disease control.

Figure 4. Root flare, high volume injection with propiconazole for oak wilt control in live oak.

For example, injection does not control the spread of the pathogen through a stand. Injected trees appear to sustain root colonization and transmit the pathogen to other connected trees, although there may be little or no crown loss. Also, preventative treatments were more effective than treating symptomatic trees, although there was some efficacy to therapeutic treatments (2). In Texas, live oaks adjacent to symptomatic trees are considered to be diseased even though they may have no symptoms, due to the likelihood of latent root infections. These limitations, combined with other drawbacks inherent in the injection process (21,27), must be considered when formulating oak wilt control programs. Injection should be just one step in a total program to control the pathogen and reduce losses of trees.

When red oaks are diseased, there are several measures available to prevent overland spread by insect vectors. The safest approach is to remove and destroy all diseased red oaks immediately upon detection. Mat formation is then eliminated as an inoculum source. Nitidulid transmission can also be prevented by avoiding wounds on trunks, limbs and branches of healthy trees, especially during the spring. Fresh wounds attract nitidulids during feeding and if contaminated, the beetles will inoculate the wound (19,20). Wound paints can also be used to prevent the beetle from coming in contact with the exposed sapwood (14,15). The wound paints need only to protect the wound for 2 - 3 days, after which the wound is no longer susceptible to infection.

In Texas, the risk of infection by nitidulids is considered to be greatest during March 1 through June 1. Free-flying nitidulid populations are highest during this period. At the same time, peaks in mat formation occur on diseased red oaks. Finally, live oaks are most susceptible to infection during the spring (6,7). These lines of evidence, combined with research conducted in other states, justify an active campaign to encourage the use of wound paints and minimize pruning of trees during the spring.

Long distance overland transmission may also be prevented by avoiding the transport of firewood. Of greatest concern is the formation of fungal mats on logs cut from diseased red oaks. How-

ever, the possibility exists that contaminated nitidulid beetles emerging form fungal mats may be harbored on live oak logs removed from disease centers. Therefore, all oak firewood originating from areas with oak wilt epidemics should be treated with caution. All of the wood should be burned prior to spring, and covering wood piles with clear plastic to prevent emergence of insects is warranted.

Future Considerations

There are many facets of oak wilt requiring further research in order to better control the disease. The nature of disease resistance in the genus *Quercus* has not been thoroughly investigated, so that selection and breeding for improved trees is impaired. There are undoubtedly gaps in our information on potential vectors such as oak bark beetles, especially in Texas where there has been little work on insects other than nitidulids. As a result, the sources of localized disease outbreaks in previously unaffected areas are occasionally impossible to explain. On a broader scale, the origins of the pathogen in the U.S. are unknown, making it difficult to predict where and when *C. fagacearum* will ultimately spread and reach epidemic proportions. In addition to the continued expansion of the oak wilt epidemic into new parts of Texas, the disease may yet spread into western and Pacific Coast states where several *Quercus* spp. comprise important components of forests. During the developing oak wilt epidemic in Texas, *C. fagacearum* has proven extremely adaptable to environmental conditions, such as a hot climate, previously considered to be limiting to disease development. Just as methods to control oak wilt in the Mid-Atlantic and North Central States has limitations for use in Texas, new approaches will probably be needed as the range of the fungus continues to expand.

The successes achieved in minimizing oak wilt losses in Texas can be attributed in part to the cooperation of private arborists with several different municipal, state and federal agencies. Many individuals and institutions have shared time and resources toward conducting surveys, research projects and public educational efforts throughout a 50 county region in central Texas. Public under-standing of the disease and the technical basis for control is therefor excellent. The USDA sponsored, Texas Forest Service Oak Wilt Suppression project provides homeowner-assistance in designing and implementing disease control strategies. County agents and extension specialists with the Texas Agricultural Extension Service have made oak wilt a high priority for public education workshops and programs to enhance the decision-making process needed to control the disease. The City of Austin, Parks and Recreation Department has played a key role in testing and demonstrating options for disease control in urban settings. These cooperative efforts have provided a model for coping with a destructive, urban tree epidemic while illustrating to the public the value of good tree maintenance practices.

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Résumé. La flétrissure du chêne, causée par *Ceratocystis fagacearum*, provoque, de façon annuelle, des épidémies dans les villages et les villes du centre du Texas. Les chênes verts (*Quercus fusiformis et Q. virginiana*) sont les plus sérieusement affectés en raison de leur prédominance et de leur grande valeur d'avenir. L'épidémiologie de la flétrissure du chêne est influencée par les caractéristiques de l'hôte, les conditions climatiques et les caractéristiques de reproduction de l'organisme pathogène. Ces facteurs ont été étudiés afin de développer les éléments clés d'un programme complet de lutte intégrée contre la flétrissure du chêne. Dans les plupart des situations, ce programme peut être utilisé au Texas pour minimiser les pertes avec succès. Ce programme s'appuie sur une perturbation des relations entre les racines, l'injection intravasculaire de propiconazole, l'élagage et le traitement des blessures (incluant l'application d'un enduit), ainsi que diverses pratiques sanitaires. Un protocole d'intervention en cinq étapes est recommandé pour bien séparer la flétrissure du chêne sur les chênes verts des autres maladies présentes au Texas et ainsi faciliter le contrôle de cette maladie.

Zusammenfassung. Die Eichenwelke, verursacht durch *Ceratocystis fagacearum*, führt alljährlich in zentraltexanischen Städten und Gemeinden zu Epidemien. Die Lebenseichen (*Quercus fusiformis* und *Q. virginiana*) sind durch ihre Verbreitung und dem damit verbundenen hohen Wert hiervon besonders betroffen. Die Epidemiologie der Eichenwelke wird bestimmt durch die Wirtseigenschaften, das Klima und die Reproduktionsmechanismen des Erregers. Diese Faktoren wurden erforscht, um die Schlüsselelemente eines umfassenden integrierten Pflanzenschutzprogramms gegen die Eichenwelke zu entwickeln. Unter den meisten Bedingungen in Texas kann dieses Programm erfolgreich angewendet werden, um die Verluste zu minimieren. Das Programm stützt sich auf eine Unterbrechung der Wurzelverbindungen, auf intravaskuläre Injektionen mit Propionazol, ienen gründlichen Rückschnitt mit Wundbehandlung (inkl. Wurdverschlußmittle) und verschiedenen pflanzenhygienischen Maßnahmen. Um die Eichenwelke in der Lebenseiche in Texas von anderen Krankheitne zu unterscheiden und um die Kontrolle der Krankheit zu ermöglichen, wurde ein Fünf-Punkte-Protokol vorgeschlagen.