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OAK WILT BIOLOGY, IMPACT, AND HOST PATHOGEN RELATIONSHIPS: A TEXAS PERSPECTIVE

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ABSTRACT

Oak wilt, caused by *Ceratocystis fagacearum*, continues to be a significant issue for natural resource managers, ranchers, and homeowners in Texas. However, the impact of the disease in central Texas is difficult to quantify. Several efforts have been made to assess the risk of oak wilt and illustrate the consequences of the disease. These surveys clearly show the disease remains at epidemic levels in many areas. The reasons for the high epidemic levels in central Texas, relative to the impact of oak wilt in other parts of the range in the U.S. are not entirely clear, but certainly the dominance of live oak is an important factor. The success of *C. fagacearum* in the oak juniper woodlands illustrates the adaptability of the pathogen to different *Quercus* spp. growing under presumably inhospitable environmental conditions. In spite of the high level of disease, oak wilt control is regularly and successfully implemented in Texas. Yet further studies are needed to gain a better understanding of this enigmatic pathogen and improve our ability to manage the disease to prevent further catastrophic losses.

Key words: *Ceratocystis fagacearum*, disease cycle, live oak, *Quercus fusiformis*

The geographic range of oak wilt, caused by *Ceratocystis fagacearum* (Bretz) Hunt, has changed little in Texas over the past 15 years since the first National Oak Wilt Symposium (see website http://www.texasoakwilt.org). A small number of counties have been added, but they do not represent any significant expansion of the disease (Fig. 1). Nonetheless, oak wilt continues to be a significant problem for homeowners, landowners, and natural resource managers in Texas. Current surveys have shown the disease is present in extremely high levels, with little evidence that these localized epidemics are decreasing. In addition, epidemics have erupted in new locations within the range that were previously free of the disease.

Oak wilt extracts a heavy toll on the central Texas landscape through a variety of manners. As has always been the case, the huge losses in the numbers of trees have a detrimental impact on property values. Losses are not only measured in terms of dollars, however. Trees in Texas represent a connection to the past and are an attachment to the natural world. The mortality of valuable shade trees, historic trees, featured landscape specimens, or any oak has negative consequences for how people view their environment. Oak wilt also has an indirect impact on other ecosystem components that depend on the central Texas oak savannah for existence. The best understood of these components is the golden cheeked warbler, an endangered songbird with a unique dependency on certain attributes of the juniper-oak woodlands that are common to the region.

The key to successfully controlling oak wilt has always depended on understanding the disease cycle. Disease cycles represent life cycles of pathogens and how they interpose on those of their hosts. For oak wilt, key features of the disease cycle were initially described by a large

number of forest entomologists and pathologists working in the 1950s (Gibbs and French 1980, MacDonald and Hindal 1981). Those descriptions were sufficient to construct a management program for much of the range of oak wilt (O'Brien et al 2000). The discovery of widespread oak wilt in Texas opened a new chapter in oak wilt management. Differences in conditions between Texas and other states with oak wilt required additional research to improve disease control (Appel 1995). As a result of this work, oak wilt can be successfully controlled under most conditions where the disease occurs in Texas.

HOW MUCH OAK WILT IS THERE IN TEXAS?

Disease Incidence at the Local Level

We are often confronted with the question, "How many trees are killed by oak wilt in Texas?" The number is undoubtedly enormous, but it is difficult to quantify with any accuracy. Several approaches have been used to map and quantify oak wilt in Texas, but these are usually far too narrow to get a complete picture of the scope of the mortality. On a narrow scale, a survey was conducted in 2001 on the Fort Hood Military Installation in an attempt to quantify the impact of oak wilt on the live oak population. The survey area was 119,000 ha. and located approximately 160 km. south of Dallas-Fort Worth, Fort Hood is located largely in Coryell County where oak wilt is considered to be a common feature in the landscape. The survey utilized IKONOS 1-meter satellite imagery to photo interpret potential oak mortality (Fig. 2). These mapped mortality centers were then transferred to Orthophoto Quarter Quadrangles (DOQQs) in order to ground truth the photo interpretation efforts. There were 1,164 polygons interpreted as diseased oaks, and 119 randomly sampled polygons were visited for ground diagnosis (Fig. 3).

Of the 119 mortality centers identified on the aerial photography, 23 were brush piles resulting from the systematic removal of junipers (*Juniperus ashei*) (Table 1). The remaining 96 polygons consisted of dying oaks, of which 82 were clearly caused by oak wilt. By extrapolating from these figures for the Fort Hood survey, there are potentially more than 800 actively expanding oak wilt centers, or one disease center every 150 ha.

In another approach, geographic information systems were used to assess oak wilt and communicate the threat of the disease to homeowners in Dallas, TX. Disease centers in a 129.5 km^2 (50 mi^2) block on the north side of Dallas were originally located with the assistance of urban foresters, arborists, and landscape managers. The disease centers were delineated and a 50 m zone around the center was surveyed for tree condition and species composition. Several analyses were conducted to determine the influence of numerous factors on the incidence and severity of the disease. The spatial distribution of the disease centers was analyzed to test for spatial dependency and randomness in their occurrence. As a result of these tests, maps were generated to illustrate the risk of oak wilt assuming spread rates of 1.6 km/year (1 mi/year) by insect vectors and 50 m/yr (162 ft/year) by root connections. These maps dramatically illustrate why there is a critical need to implement control measures in a valuable urban forest (Fig. 4).

Regional Disease Incidence

An additional indicator of the intensity of oak mortality in Texas caused by oak wilt can be found at the website http://www.texasoakwilt.org/. This website is an excellent source of information concerning all aspects of oak wilt, and contains an Internet Map Server that can be used interactively to view the locations of disease centers throughout many parts of the oak wilt range in Texas. There are several locations in the state where oak mortality dominates the

landscape, such as the view of Kerr, Gillespie, and Kendall counties (Fig. 5). Although not a complete documentation, there is sufficient survey data to illustrate the incidence and severity of oak wilt in the central Texas woodlands.

By any measure, it is apparent that oak wilt at many places is having a lasting impact on the ecology of the oak-juniper savannahs. The disease is also causing urban foresters and homeowners to question the value of widespread planting of live oak, the most popular shade tree in the state.

WHY IS IT SO BAD IN CENTRAL TEXAS?

The course of an oak wilt epidemic is very much a product of the host species present and the structure of the tree stand. These factors can contribute to a circumstance where the pathogen spreads rapidly, killing countless trees. The disease then presents unique challenges in successfully being contained and controlled. Just such a disastrous scenario has arisen in central Texas, particularly when compared to oak wilt where it occurs in the mid-Atlantic, Midwestern and North Central states.

Host Influences

Oak species vary widely in their response to infection by *C. fagacearum*. This variation was noted soon after the pathogen was first described (Henry et al. 1944), and extends to mortality rates, symptom development, and potential production of inoculum. All members of the red oak group (subgenus *Erythrobalanus*) have repeatedly proven to be susceptible to the pathogen (Fig. 6). In contrast, members of the white oak group (subgenus *Leucobalanus*) are very resistant to the disease with little or no loss of crown to dieback (Rexrode and Lincoln 1965, MacDonald and Hindal 1980). Live oaks (subgenus *Leucobalanus*) are intermediate between these two extremes in their response to the pathogen (Appel 1986). Most infected live oaks either die or lose large proportions of their crowns to the disease. About 15% or the infected live oak population may survive intact or respond minimally with little dieback. Foliar symptoms of oak wilt in live oak are distinct, often exhibiting a striking chlorosis and necrosis of the veins (Fig. 7). Foliar symptoms in red oak are most often described as a bronzing of leaves with an accompanying marginal scorch. The foliar response of white oaks is similar to that of the red oak group.

The Disease Cycle

The primary vectors of the pathogen are sap-feeding nitidulid beetles (Coleoptera: Nitidulidae). Insect spread of the oak wilt pathogen is sometimes referred to as overland, or long distance spread. The *C. fagacearum* vector relationship is not a simple one and may be one reason for the limited losses from oak wilt relative to other notorious tree diseases such as Dutch elm disease and chestnut blight. The only known source of inoculum for acquisition by nitidulids forms underneath the bark on the surface of the sapwood (Fig. 8). As the tree dies from oak wilt, the pathogen undergoes a brief period of saprophytic growth and forms a mat of growth on which spores are formed. This mat pushes the bark outward to make cracks for the nitidulids to visit and become contaminated with the spores. The mats range from a few to several inches long, usually have an elliptical shape, and emit a very sweet smelling odor to attract insects. In addition to providing spores for long distance transmission to initiate new disease centers in healthy trees, the mats allow for cross fertilization of the two mating types of the pathogen and

subsequent sexual reproduction. For some unknown reason, fungal mats only form on diseased red oaks. They do not occur on infected white oaks.

In addition to the fungal mats, nitidulids require a wound on the target tree in order to successfully vector the pathogen. To be a successful infection court, the wound must be less than a few days old. This additional requirement in the disease cycle is another reason why losses to oak wilt have been relatively limited.

There is another mechanism of spread for *C. fagacearum*, termed local or underground spread. This mechanism is by means of grafted roots (Fig. 8). Root grafts form when the roots of one tree fuse to those of an adjacent tree of the same or closely-related species. Since *C. fagacearum* is a vascular parasite, functional root grafts provide an effective avenue for spread of the fungus from a diseased to a healthy tree.

The dominance of live oak in central Texas woodlands and its popularity as a shade tree changed the formula for assessing the epidemic potential of *C. fagacearum*. In addition to making acorns, live oak has the ability to reproduce clonally by the formation of root sprouts. This means of vegetative reproduction has several ecological advantages over other tree species when compared to conventional seed production. For example, live oaks have been able to efficiently colonize the former grasslands of central Texas following the control of fires and overgrazing. This ability to rapidly colonize disturbed sites is the reason there are huge monocultures of live oak in central Texas with limited species diversity. These stands are made up of highly interconnected clonal trees with common root systems supplemented by root grafting.

Although vegetative root sprouting may be an efficient means of reproduction, it is particularly detrimental when connected trees are exposed to a vascular parasite such as *C. fagacearum*. Root graft spread is common to oak wilt throughout the range in the U.S., but spread through the live oak common root systems in Texas adds a whole new dimension to oak wilt epidemiology (Fig. 9). Red oaks, such as Spanish oak (*Quercus buckleyii*) and blackjack oak (*Q. marilandica*), play the same role in Texas as they do elsewhere. Fungal mats form on them, but the high heat and dry conditions sometimes diminish the numbers of potential mat-bearing trees. When a contaminated nitidulid introduces *C. fagacearum* into an interconnected stand of live oaks, the pathogen spreads rapidly from tree to tree within the stand. This sort of spread results in the production of very large disease centers (Fig. 10).

The Significance of Live Oaks

The unique response of live oak to oak wilt in Texas has had many implications for our understanding of the disease. Oak wilt went undiscovered in Texas, perhaps at least for 40 years, after the disease was initially described in Wisconsin in 1941. This failure to recognize the disease in Texas probably derived from the unfamiliar symptoms exhibited by live oak. It should also be noted that *C. fagacearum* is a heat sensitive fungus, so the oak forests of the southern U.S. were presumably safe from the disease (Schmidt 1978, Gibbs and French 1980). Although live oak exhibits a high degree of susceptibility to the pathogen in the manner of red oaks, no fungal mats form on live oaks. The ability to reach epidemic proportions in a host population where no external inoculum sources are present for insect transmission reveals the great resilience of the oak wilt pathogen. The ability to adapt to a region with intense high temperature extremes and relatively limited insect transmission clearly shows this pathogen can adapt to new oak forests, regardless of our attempts to predict risk based on our current understanding of the pathogen.

CAN WE CONTROL OAK WILT?

The predominance of live oak as a primary host for *C. fagacearum* also has implications for how we approach the control of the disease in Texas. As with all plant diseases, our ability to control oak wilt is based on our understanding of the biology of the host and the pathogen. Overland spread by nitidulids may be accomplished by avoiding wounding of oaks in the spring, the use of wound paints on fresh wounds, and the cautious movement of firewood. Underground or local spread may be prevented by trenching to break up root grafts and common root systems. Roguing of trees, in order to further damage existing root systems in hopes of destroying pathogen habitat, is also recommended. Intravascular injection of high risk trees with fungicides does not successfully prevent a tree from becoming infected, but it has been shown to be effective in protecting high risk trees from extensive pathogen colonization. As a result, treated trees survive, often with all or most of their crowns intact. New fungicide products continue to be introduced for injections, but most are based on the originally-tested fungicide propiconazole or some closely-related compound. There are numerous trees, including some oak species that are recommended for replanting once the epidemic has abated. Experience has shown that even live oaks planted in remnant oak wilt centers seem to escape the disease indefinitely. Oak wilt control and the related issues of inoculum sources, infection courts, insect vectors, and potential resistance in the live oak population are addressed in greater detail in several of the presentations in this Symposium.

UNANSWERED QUESTIONS

As measures to manage oak wilt improve, it allows us time to consider some of the broader issues concerning this important disease. One of those issues is the origin of the pathogen and the potential for expansion into valuable, unaffected resources. The analysis of the range of a plant pathogen often reveals clues as to where that pathogen may have originated, but in the case of oak wilt this has not been the case. The current range in Texas reflects some revealing attributes of the disease that also occur in the oak wilt range nationally. Oak wilt in Texas continues to be a problem largely in the central portion of the State, where the tree types are dominated by the oak-juniper woodlands. There has been no encroachment into the east Texas pineywoods, where susceptible oak species occur in large numbers (Fig. 1).

The lack of expansion to the west and south in Texas is understandable due to sparse host type, but the failure of the pathogen to disperse into east Texas remains a mystery. A similar phenomenon occurs throughout the range of oak wilt in the U.S. In several states, the pathogen has failed to encroach into forests where there are susceptible trees and no perceptible climatic limits. If we do not have a good understanding of where *C. fagacearum* might have come from, then our ability to predict the impact it may continue to have elsewhere may also be flawed. This issue is particularly timely due to the increasing concern for regulating and assessing the risks of exotic, invasive species. Other lines of evidence on the origins of *C. fagacearum* are discussed by authors elsewhere in this Symposium.

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Table 1. Results of ground truthing randomly selected polygons at Fort Hood, TX.

Cause	No. Centers	% Total
Oak Wilt	82	69
Military Ops	1	0.8
Unknown	8	6.7
Brush Piles	23	19.3
Blow down	1	0.8
Fire	4	3.3

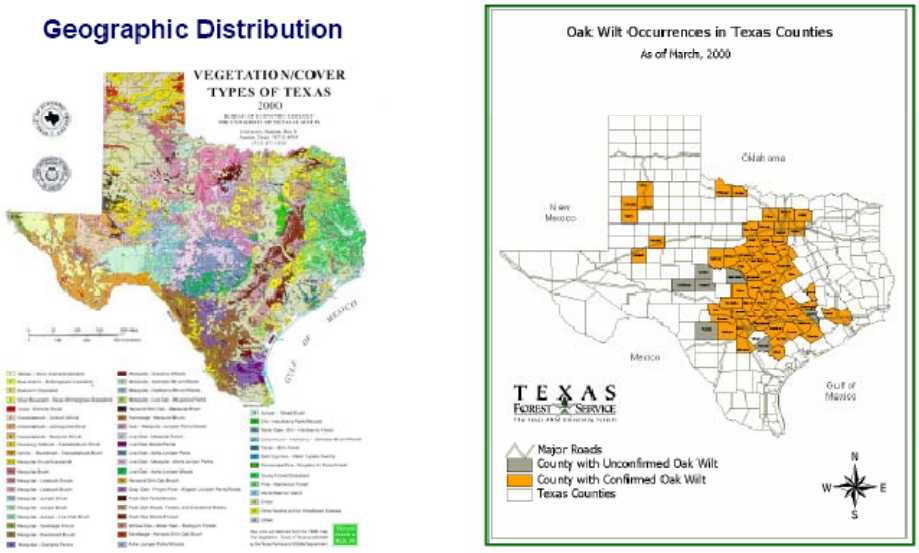


Figure 1. Vegetative cover types and range of oak wilt in Texas.

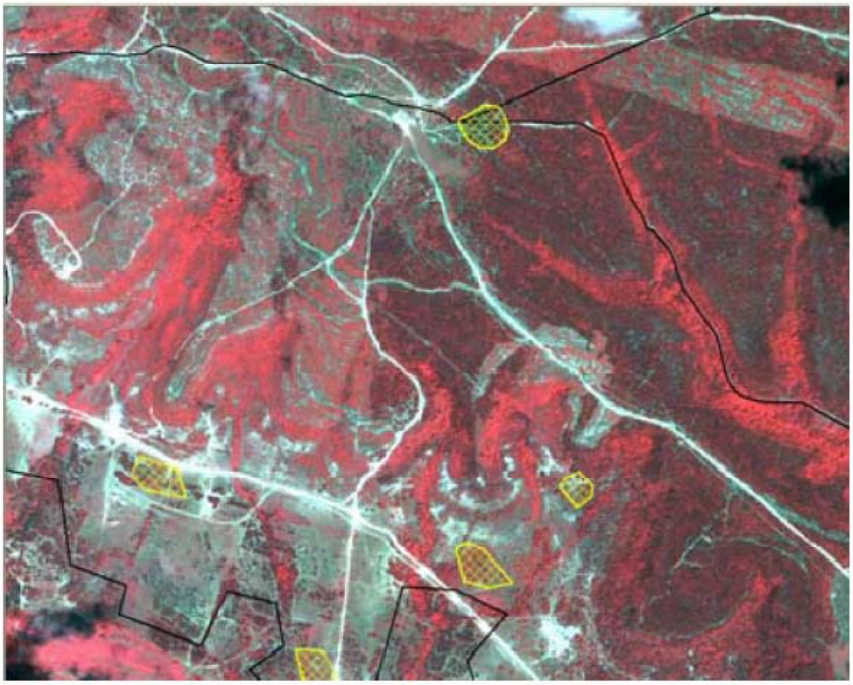
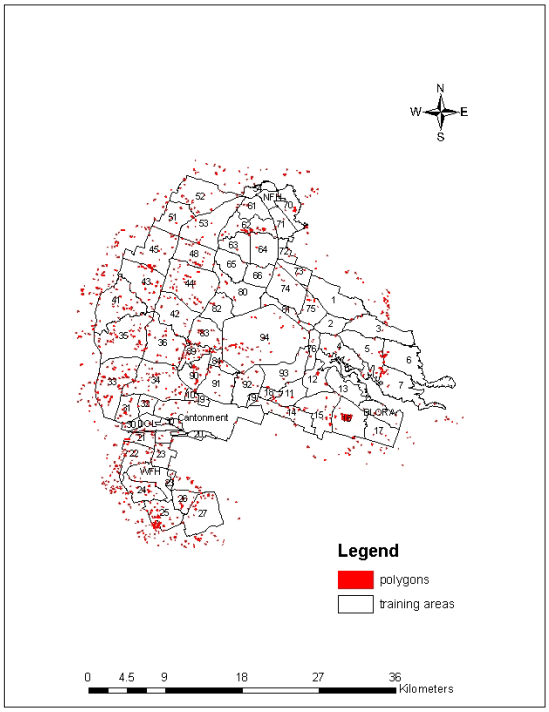


Figure 2. Example of the IKONOS 1-meter satellite imagery used to survey for oak wilt at Fort Hood, TX. Yellow polygons are areas identified by the photointerpreter as oak mortality.

A



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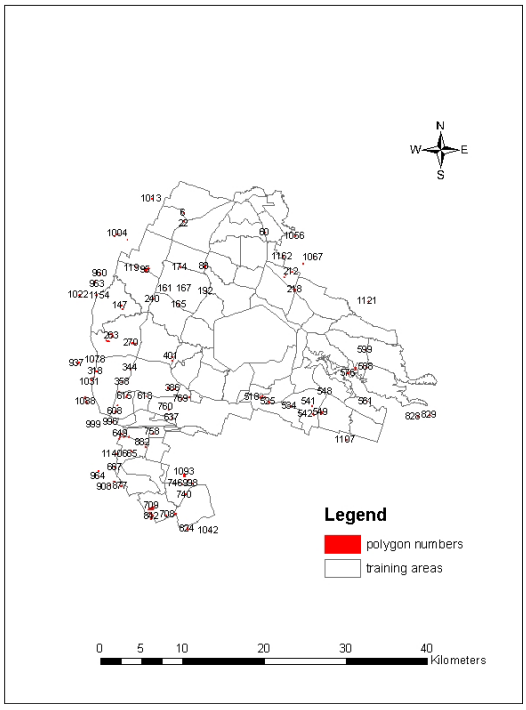

Figure 3. Map of Fort Hood, TX, with training areas and the locations of the 1164 polygons interpreted as oak mortality (A) and the 119 randomly selected polygons selected for ground truthing (B).

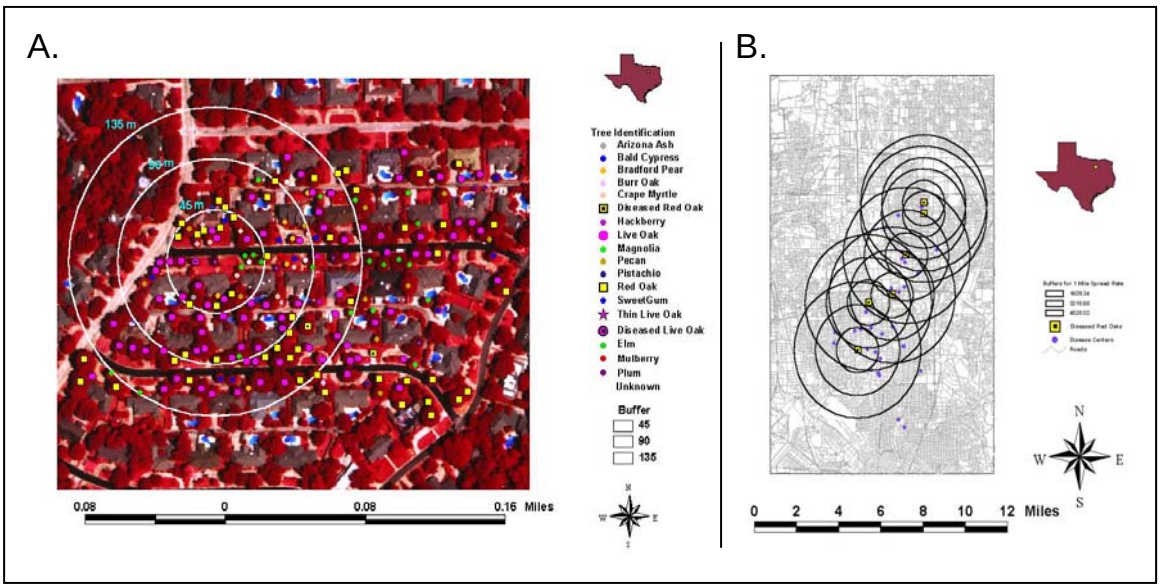

Figure 4. Illustration of the risk of annual spread of *C. fagacearum* locally through root connections (A) and by overland by insect vectors (B) in Dallas, TX.

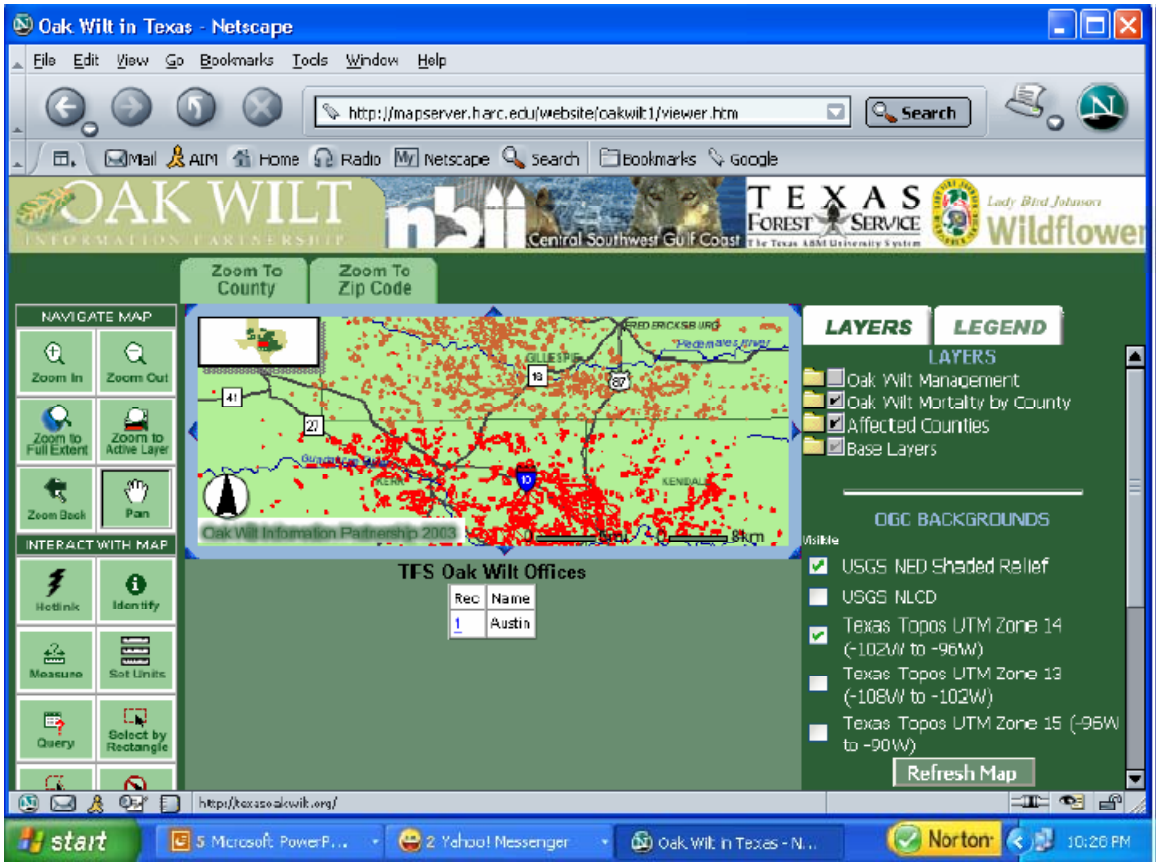

Figure 5. Interactive website (www.texasoakwilt.org) illustrating the incidence of oak wilt in Kerr/Kendall counties, TX. Red polygons are areas of oak mortality identified by Texas Forest Service foresters during aerial surveys.

Figure 6. Dead red oaks near Lampasas, TX, and typical red oak foliar symptoms (insert).

Figure 7. Dying live oaks near Lampasas, TX, and typical live oak foliar symptoms of infection (inserts).

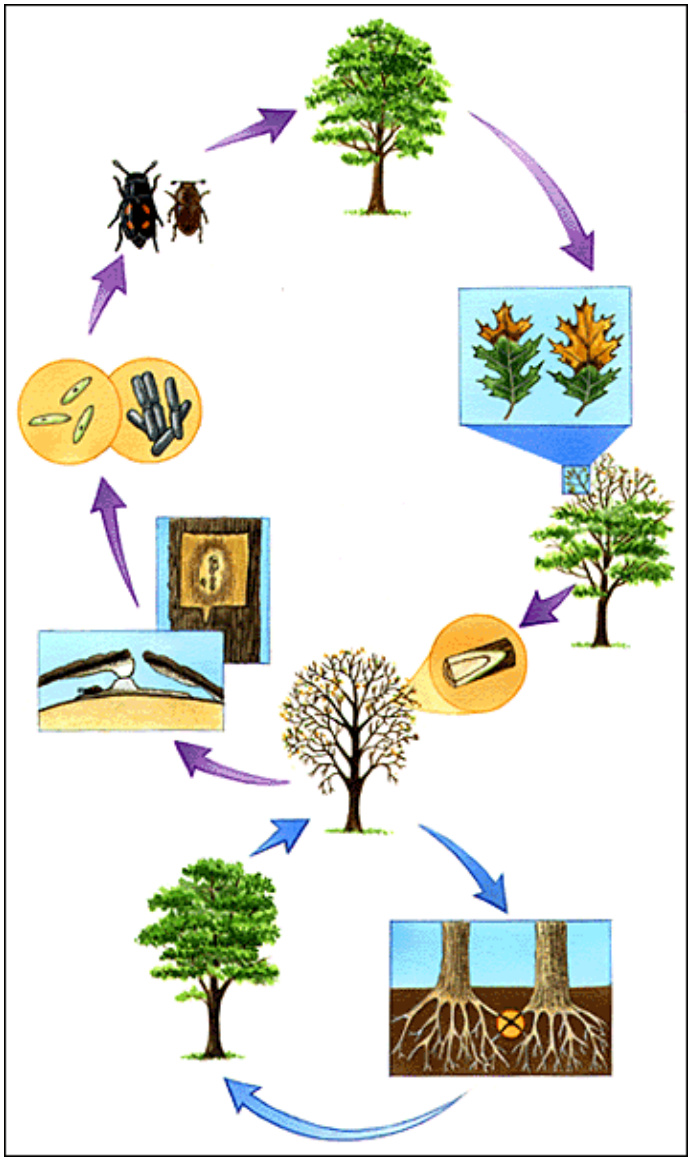

Figure 8. Oak wilt disease cycle (O'Brien et al. 2000).

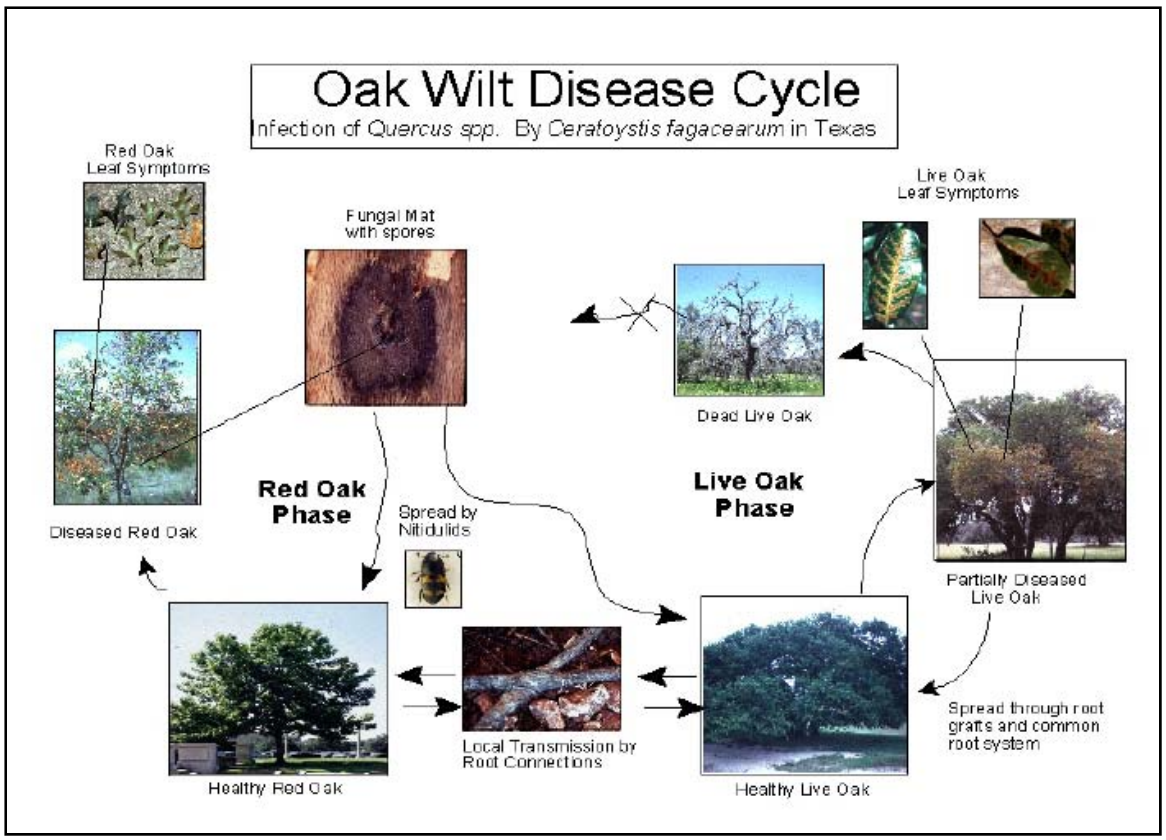

Figure 9. Oak wilt cycle in Texas, with emphasis on the live oak phase of the disease.

Figure 10. Remnants of a large, old oak wilt center near Lampasas, TX.