

RECOGNITION OF OAK WILT IN LIVE OAK

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Abstract. Over a two-year period, 77 suspected oak wilt centers were examined in 7 Texas counties. Fungal mats, formed by the oak wilt pathogen *Ceratocystis fagacearum*, were discovered under the bark of diseased Spanish oaks at 12 of the sites. Mats were not found on live oaks. Five types of symptoms were observed on 138 diseased trees selected for sampling and laboratory isolation of *C. fagacearum*. Veinal necrosis and tipburn of leaves occurred separately or in combination on 51% of the diseased live oaks. These symptoms were reliable predictors for isolation of *C. fagacearum*. Typical wilting was found only on diseased Spanish oaks. Interveinal chlorosis, non-specific decline symptoms, and various other combinations were also found on trees from which *C. fagacearum* was isolated. In one 36-acre oak wilt center, 45% of the oaks surveyed were dead or dying. The remaining trees, in various states of health, were scattered throughout the center. These observations have improved early detection and diagnosis of oak wilt in live oaks and contributed to the conclusion that oak wilt is the most serious oak disease in Texas.

There are 27 species of semi-evergreen, persistent oaks (*Quercus* spp.) in the southern and southwestern USA (Miller and Lamb, 1985). Two of these live oaks, *Q. virginiana* and *Q. fusiformis*, are highly valued as specimen and shade trees, and provide extensive, reliable, cover and mast on the central Texas rangelands. Relatively few pathogens have been reported as damaging live oaks. One exception is the documented occurrence of *Ceratocystis fagacearum*, the oak wilt fungus, in 31 central Texas counties (Appel et al., 1985).

The initial discovery of *C. fagacearum* in Texas was made in 1961 from diseased trees in Dallas (Dooling, 1961) and was well beyond the accepted range of oak wilt (Hepting, 1971). Subsequently, the pathogen has become a serious threat to the extensive live oak woodlands of central Texas. *Ceratocystis fagacearum* is inhibited or killed by temperatures exceeding 32 C° (90 F°) (Houston et al., 1965), but in Texas it can survive high summer temperatures in the roots and boles of infected trees (Lewis, 1985). Many characteristics of the oak wilt disease cycle, such as the formation of fungal mats for inoculum production, presence of insect vectors, and local spread of *C. fagacearum* through common root

connections, have been observed in Texas (Appel and Lewis, 1985; Appel et al., 1985; Lewis and Oliveria, 1979).

Typical foliar oak wilt symptoms in deciduous oaks include water soaking and browning or bronzing of leaf tips and margins (MacDonald and Hindal, 1981). Red oaks (*Quercus*; subgenus *Erythrobalanus*) are very susceptible to infection by *C. fagacearum* and usually die in the year of initial infection, while white oaks (*Quercus*; subgenus *Lepidobalanus*) either die more slowly or recover. The foliar symptoms in live oaks vary, but do not resemble symptoms on deciduous oaks. The most reliable, diagnostic foliar symptoms of oak wilt on live oaks are veinal necrosis and tipburn (Appel and Maggio, 1984). However, these symptoms are not always found in oak wilt centers. Another observation made during survey for live oak diseases was the presence of deteriorated, yet surviving trees, within active disease centers. The majority of the live oaks infected by *C. fagacearum*, however, defoliate and die rapidly following appearance of symptoms in the crown (Lewis and Oliveria, 1979).

This report describes and quantifies various oak wilt symptoms on oaks in Texas in relation to successful isolation of *C. fagacearum*. Also, the results of surveying a single, large oak wilt center are reported. An analysis of these observations may lead to improved diagnosis of the disease in live oaks.

Materials and Methods

During 8 Feb 1983 - 30 Oct 1985, 77 sites were visited and diagnosed as being oak wilt centers. Most sites (68) were chosen randomly during systematic survey of central Texas utilizing color-infrared aerial photography (Appel and Maggio, 1984). In addition, nine oak wilt centers were visited at the request of county agricultural extension agents or landowners. Diagnosis of oak wilt in the field depended on the presence of foliar symptoms and fungal mats, and the patterns of

mortality caused by fungal spread. For laboratory isolation of *C. fagacearum*, samples were removed from branches (greater than 1 inch dia.) and boles of symptomatic trees. Bole samples were chiseled from sapwood in trunks of diseased trees after removing the outer and inner bark. All samples were stored on ice in the field, returned to the laboratory, and cultured at room temperature on acidified potato-dextrose agar (PDA) prepared from fresh potatoes.

One oak wilt center, located at Round Rock, TX, was surveyed using a systematic line plot cruise (Figure 1). This site was previously confirmed as an oak wilt center by laboratory isolation of *C. fagacearum*, and was estimated to be 12-13 yr old at the time of survey. During the survey, trees were tallied and estimates made of the proportions of tree crowns lost to disease. The ten-percent line plot cruise used 0.2-acre plots spaced 4 chains apart on lines 5 chains apart (Figure 1).

Results

Fungal mats or mat scars (stained areas) were found on dying or dead Spanish oaks (*Q. texana*) at 12 disease centers visited (Table 1). No mats were found on live oaks. From the remaining 65 sites, 138 trees were sampled by removing 316 tissue specimens for culturing in the laboratory. There were 121 live oaks, 12 Spanish oaks, and 5 shin oaks (*Q. sinuata* var. *breviloba*) among the trees sampled. Bole samples were more reliable for isolation of *C. fagacearum* than branch samples (Table 1). Of the 170 bole samples collected, 38% were positive. Only 19% of the 191 branch samples were positive for isolation of the pathogen. The discovery of mats or isolation of *C. fagacearum* confirmed 55 of the 77 disease sites as oak wilt centers.

Symptoms on sampled trees were categorized into five types (Table 2). Symptom type 4, typical wilting, was observed only on diseased Spanish oaks and shin oaks. Type 5 symptoms were non-specific and could be caused by any of a number of tree disorders unrelated to oak wilt. *Ceratocystis fagacearum* was isolated from only seven of the 36 trees with type 5 symptoms (Table 3). In contrast, a much greater percentage of trees exhibiting symptom types 1 and 2 tested

positive (49% and 46%, respectively). The greatest isolation success (70%) was from diseased Spanish oaks with typical oak wilt symptoms (type 4).

Combinations of symptoms were also encountered on diseased trees (Table 3). The most common was veinal necrosis (type 1) associated with tipburn (type 2) in the same crown and sometimes on a single leaf. The frequency of isolating *C. fagacearum* from trees with symptom types 1 and 2 was 51%.

Six survey lines were sufficient to survey the entire Round Rock oak wilt center. The area of mortality was approximately 36 acres (Figure 1). There were 78 live oaks tallied in the 18 survey plots, giving an estimate of 22 trees/acre. This estimate is probably low, because occasionally dead trees in the center had been harvested for firewood. One sugar hackberry (*Celtis laevigata*) and one cedar elm (*Ulmus crassifolia*) were the only other tree species among the live oaks in the plots.

No discernable pattern in distribution of healthy and diseased trees throughout the Round Rock center was detected (Figure 1). Of 78 live oaks tallied, 35 (45%) were dead or nearly dead. The remaining live oaks exhibited various degrees of dieback. Twenty-two trees (28%) survived with less than 50% crown loss; seven of these appeared to have escaped infection entirely with no crown loss. Healthy, escape trees were located in both the oldest and youngest portions of the disease center. Also, many surviving live oaks with type 5 decline symptoms were found in the oldest portions on survey lines 1-3. Specific symptoms of oak wilt were observed only on diseased trees in survey lines 5 and 6 where trees were most recently infected. The oak wilt center was bordered by a highway and railroad track, restricting expansion of the center to only one direction.

Discussion

The symptoms described for oak wilt in live oaks and the oak wilt center at Round Rock were typical of the disease in central Texas. Diseased stands are often large, discrete, and comprised of hundreds of trees in various stages of disease development. Three types of distinct,

recognizable foliar symptoms are encountered on diseased live oaks. The pathogen is most easily isolated from diseased live oaks when veinal necrosis, tipburn, or combinations of these are found on the same tree. These symptoms are usually found only in diseased trees on the periphery of oak wilt centers, rather than on deteriorating, surviving trees in the older portions. The confirmation of oak wilt in live oaks, therefore, depends on sampling boles of the most recently infected, symptomatic trees. If symptomatic branches must be used, or trees with non-specific symptoms are sampled, the probability of recovering the pathogen from diseased tissues is diminished.

The reasons for the variety of oak wilt symptoms on live oak are unknown. The necrotic patterns observed on leaves do not appear to reflect stages in colonization of a tree by *C. fagacearum*. Also, no association between season and appearance of a specific symptom was observed.

Species composition of forests probably has a strong influence on oak wilt incidence (MacDonald and Hindal, 1981). The disease is prevalent in Texas where species diversity is low, as in the homogenous live oak stand observed at Round

Rock (Figure 1). In central Wisconsin, oak wilt is severe in coppice stands of pin oak (*Quercus palustris*) that regenerate in woodlands destroyed by logging and fire (MacDonald and Hindal, 1981). Live oaks in central Texas have similarly invaded former grassland prairies following fire control and overgrazing (Buechner, 1944; Scifres, 1985). Live oak is well suited for rapid regeneration on altered sites because it can propagate vegetatively by production of suckers on stem-like rhizomes (Muller, 1951).

The formation of common root systems as a result of the vegetative reproductive habit of live oaks is distinct from root connections that arise when roots of two trees graft. If common root systems are maintained through maturity of the tree, they may provide a more efficient means of transmission than root grafts, a common mechanism for local spread in deciduous oaks (MacDonald and Hindal, 1981). Assuming live oaks may also form root grafts and the two types of connections are combined, the fungus could rapidly colonize very large centers such as those in central Texas. The potential size of clonal live oak stands is unknown. Quaking aspen (*Populus tremuloides*) is an early successional tree that

Table 1. Attempted isolation of *Ceratocystis fagacearum* from 361 branch and bole samples removed from live oaks, Spanish oaks, and shin oaks with oak wilt symptoms.

Diagnostic Status	No. Sites	No. Trees	Type of sample removed			
			Branch		Bole	
			Pos	Neg ^b	Pos	Neg
Mats or mat scars found	12	-- ^a	--	--	--	--
<i>C. fagacearum</i> isolated	43	54	37	86	65	63
<i>C. fagacearum</i> not isolated	<u>22</u>	<u>84</u>	<u>0</u>	<u>58</u>	<u>0</u>	<u>41</u>
Total	77	138	37	154	65	105

^a Samples were not collected when fungal mats or mat scars (stained areas) were found.

^b Pos = positive; Neg = negative.

reproduces by root suckering, and sometimes it produces individual root systems of several acres (Barnes, 1975).

There are additional agents of transmission for *C. fagacearum*. One of these is the sap-feeding nitidulid beetle. These small beetles may visit fungal mats, acquire inoculum, and subsequently deposit the inoculum in wounds on another tree (Gibbs and French, 1980). The unexpected discovery of fungal mats on Spanish oaks in the extreme southwest range of oak wilt indicates that

the pathogen survives both high temperatures and the competitive presence of the oak canker fungus, *Hypoxyton atropunctatum*. These factors were formerly believed to limit the survival of *C. fagacearum* in the South (Gibbs and French, 1980).

The control of oak wilt in Texas will rely heavily on recommendations previously developed in other states. The disease in live oak, however, is unique to previous experiences with oak wilt and some of the conditions in Texas will limit the

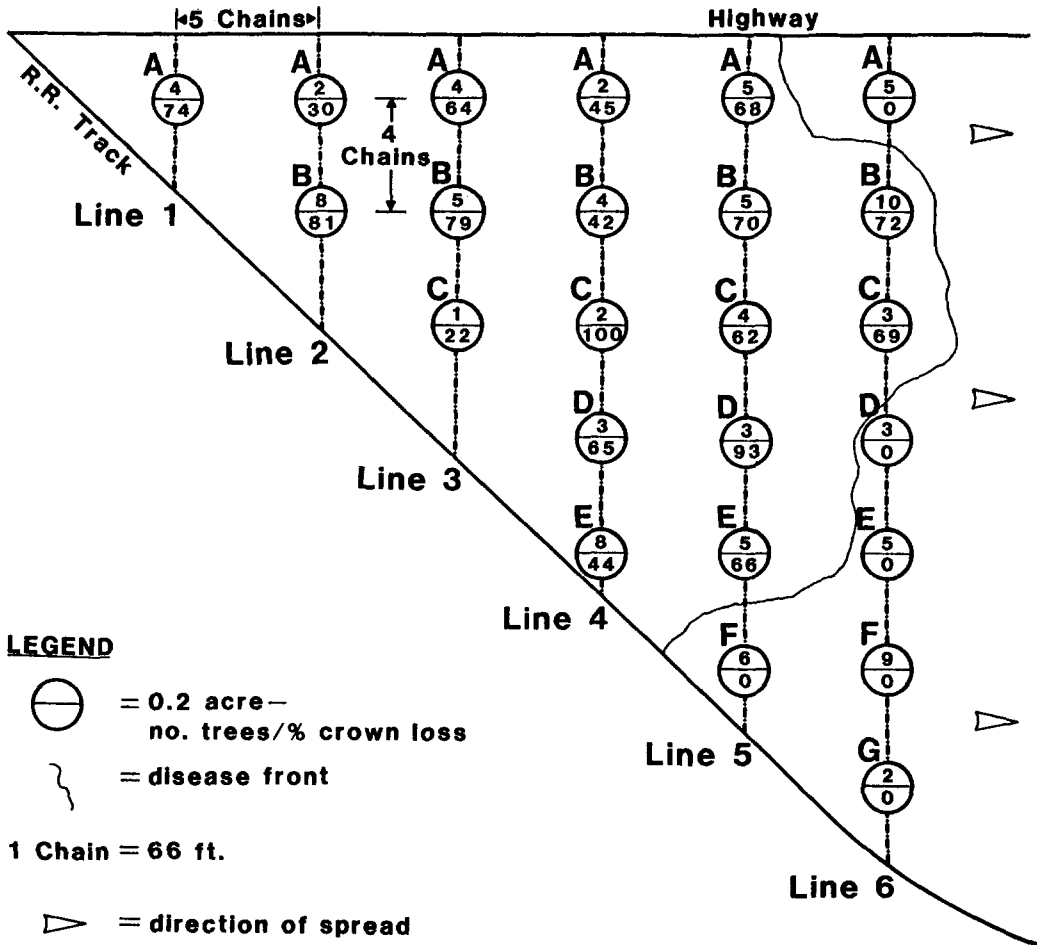


Figure 1. Ten percent line-plot cruise of an oak wilt center depicting survey lines, plots, numbers of trees per plot, and the average percentage crown loss of trees within a plot.

usefulness of traditional control recommendations (Appel and Lewis, 1985). For example, where disease incidence is high, disease centers are extremely large, and soils are shallow, measures such as trenching to disrupt root connections are not practical (Appel and Lewis, 1985).

There are also opportunities in Texas to develop a better understanding of oak wilt and formulate new methods of control. For example, the susceptibility and rapid death of live oaks resembles the typical response of red oaks to the pathogen. Yet, the slow deterioration and survival

of some trees is typical of resistant white oaks (MacDonald and Hindal, 1981). The reasons for these contrasting responses are unknown, but if clarified may eventually be used to increase tree survival.

Oak wilt is now considered to be the most serious disease of live oaks in Texas. Rapid and accurate diagnosis of the disease is the first step in developing suitable control recommendations. By carefully considering symptoms, tree location, and sample collection, diagnostic problems experienced in the past can be avoided and appropriate measures can be taken to reduce losses of valuable trees.

Table 2. Symptoms observed on diseased oaks located in suspected oak wilt centers.

Symptom type	Description
1	Chlorosis and necrosis of the mid-vein and smaller lateral veins.
2	Necrotic scorch of leaf tips and margins.
3	Chlorosis and necrosis of the interveinal regions of leaves.
4 ^a	Browning and bronzing of leaf margins, necrosis of entire leaf; wilting.
5	General, non-specific chlorotic and necrotic mottling of leaves; crown dieback.

^a These symptoms were observed only on deciduous Spanish oaks and shin oaks.

Literature Cited

1. Appel, D. N. and R. L. Lewis. 1985. Prospects for oak wilt control in Texas. Pages 60-68. In, *Insects and Diseases of Southern Forests*, R. A. Goyer and J. P. Jones, ed. Proc. 34th Ann. For. Symp., Louisiana State University, Baton Rouge. 135 pp.
2. Appel, D. N. and R. C. Maggio. 1984. Aerial survey for oak wilt incidence at three locations in central Texas. *Plant Disease* 68:661-664.
3. Appel, D. N., C. F. Drees and J. Johnson. 1985. *An extended range for oak wilt and Ceratocystis fagacearum compatibility types in the United States*. *Can. J. Bot.* 63:1325-1328.
4. Barnes, B. V. 1975. *Phenotypic variation of trembling aspen of western North America*. *For. Sci.* 21:319-328.
5. Buechner, H. K. 1944. *The range vegetation of Kerr County, Texas, in relation to livestock and white-tailed deer*. *Am. Midl. Nat.* 31:697-743.
6. Dooling, O. J. 1961. *Oak wilt identified in Texas*. *Plant Dis. Rep.* 45:749.
7. Gibbs, J. N. and D. W. French. 1980. The transmission of oak wilt. *U.S.D.A. For. Serv. Pap. NC-185*. 17 pp.

Table 3. Isolation of *Ceratocystis fagacearum* from diseased oak trees with various symptom types.

Result	Symptom type ^a								
	1	2	3	4 ^b	5	1+2	2+3	1+2+3	1+3
Positive	18	6	2	10	7	11	2	0	0
Negative	19	7	4	7	29	10	3	2	1
Total	37	13	6	17	36	21	5	2	1

^a Symptoms include; 1. veinal necrosis; 2. tipburn; 3. interveinal chlorosis; 4. wilting; 5. non-specific, decline symptoms.

^b Wilting occurred only on diseased Spanish oaks and shin oaks. The remaining symptoms were found on live oaks.

8. Hepting, G. H. 1971. Diseases of forest and shade trees of the United States. U.S.D.A., For. Serv., Agricultural Handbook No. 386. 658 pp.
9. Houston, D. R., C. R. Drake and J. E. Kuntz. 1965. *Effects of environment on oak wilt development*. *Phytopathology* 55:1114-1121.
10. Lewis, R. 1985. *Temperature tolerance and survival of Ceratocystis fagacearum in Texas*. *Plant Disease* 69:443-444.
11. Lewis, R., Jr. and F. L. Oliveria. 1979. *Live oak decline in Texas*. *J. Arboric.* 5:241-244.
12. MacDonald, W., and D. Hindal. 1981. Life cycle and epidemiology of *Ceratocystis*. Pages 113-144. In, M. E. Mace, A. A. Bell, and C. H. Beckman, ed., *Fungal Wilt Diseases of Plants*. Academic Press, N. Y. 640 pp.
13. Muller, C. H. 1951. *The significance of vegetative reproduction in Quercus*. *Madrono* 11:129-137.
14. Scifres, C. J. 1985. IBMS: Ecological basis and evolution of concepts. Chapter 1. In, *Integrated Brush Management Systems for South Texas: Development and Implementation*. C. J. Scifres, ed. Texas Ag. Exp. St. B-1439. 71 pp.

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Abstract

FUECHT, J. R. 1985. **Diagnosing damage from landscape chemical use**. *Am. Nurseryman* 162(5):92-94, 96.

Damage to trees from chemicals applied for control of pests, weeds, and diseases is a relatively common problem. Sources of chemical injury include incompatible or incompletely mixed ingredients, spray drift from herbicides, and residues from previous sprayings in the spray tank or discharge hose. Other less frequently recognized sources are the leaching of soil-applied herbicides and the application of herbicides within the root zones of trees. Perhaps the most common tree injury results from herbicide drift. Phenoxy types, such as 2,4-D, can cause considerable foliage distortion on some trees a long distance downwind from a spray area. Careless mixing of two or more pesticides or using an old pesticide that has frozen and separated can result in tree injuries. Most, if not all, of these damages are the result of "carriers," such as xylene and other petroleum distillates. When improperly dispersed on plant foliage, these materials will concentrate and damage the natural wax protection (cuticle) of a leaf. Sometimes spray burn resembles drought stress. This is particularly true when the spray is applied on a hot day. Many herbicides are applied to the soil, where they act upon the roots of weeds. Dicamba (Banvel) is a common chemical that acts in this way. Unfortunately, dicamba also acts on roots of trees and shrubs. It has a relatively long life and is subject to leaching. Soil sterilants have no useful place in any landscape, residential or commercial. Soil sterilants are intended only for industrial locations. Soil sterilants sterilize the soil. They kill not only trees, but most other forms of life in the soil as well, rendering the soil unfit for plant growth.