LIVE OAK DECLINE IN TEXAS

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Live oak, Quercus virginiana L., is the predominant oak in much of central Texas and along the Gulf and southern Atlantic coasts of the United States. Live oak is the predominant tree in many central Texas parks, including the Lyndon B. Johnson National Historic Site. It has adapted to various soil types and can survive periodic droughts. The live oak protects rangeland and livestock, is a habitat and food source for wildlife, and is sometimes used as fuel. Moreover, the live oak is highly prized for aesthetic reasons and is planted as an ornamental, even outside its natural range.

The live oak is threatened by a disease complex commonly known as live oak decline. This disease has destroyed thousands of acres of nearly pure live oak stands, and it continues to spread.

Taubenhaus (1934, 1935) first reported the apparently new disease, which he found affecting about 200 live oaks near Austin, Texas, but he did not learn its causes.

Dunlap and Harrison (1949) found the disease in 20 central Texas counties. Although they also failed to learn its causes, they established that the disease was unrelated to soil conditions and was probably caused by a biological agent.

Halliwell (1965-1966) found the disease throughout most of the natural live oak habitat in Texas. He named the disease live oak decline. He associated Cephalosporium sp. with slow decline and Hyalodonton sp. with rapid decline resembling oak wilt (1965). Van Arsdale (1970) identified live oak decline as a vascular wilt that he believed was caused by Cephalosporium diospyri. Van Arsdale et al. (1974) believed this fungus to be a highly evolved pathogen requiring 10 or more years to kill live oaks. Taubenhaus (1935), Dunlap and Harrison (1949), and Halliwell (1965), however, all reported that the disease could kill trees within a few weeks.

To obtain more information about the causes of live oak decline, we began to study the disease in 1976. This paper reports the causes of live oak decline in Texas and how adverse environmental conditions and insect defoliations contribute to the disease complex.

Live oak wilt

Live oak decline is essentially a vascular wilt. The initial wilt symptoms are leaf chlorosis, leaf browning, and defoliation (Fig. 1). Advanced symptoms are twig and branch dieback, adventitious sprouts on trunks and large limbs, small new leaves, and thin crowns. Initial symptoms develop rapidly in healthy trees during spring and fall and are very conspicuous; they resemble what Halliwell (1965) referred to as “fast decline.” The advanced symptoms are evident within 3 months after wilt begins. Except for dieback development and adventitious sprouts, there is little change in the appearance of surviving trees with advanced wilt symptoms; the advanced symptoms resemble what Halliwell (1965) described as “slow decline.” Many trees were killed by wilt within a few weeks; some developed advanced symptoms but survived several years before dying from other complications, and others developed advanced symptoms but slowly recovered. The wilting mechanism appeared to become static after trees developed advanced symptoms.

During 1977-78, in areas between Johnson City and Kerrville, Texas, we were able to con-
consistently isolate *Ceratocystis fagacearum* (oak wilt) from live oaks with incipient wilt. Rarely were we able to isolate it from some of the same trees after advanced symptoms developed. Because other fungi later colonized these trees, we concluded that such colonization might prevent frequent isolation of *C. fagacearum* from trees with advanced symptoms.

The fungi most frequently isolated from trees with advanced symptoms were: *Botryodiplodia theobromae*, *Cephalosporium* sp., *Coryneum* sp., *Dendrophoma* sp., *Dothiorella* sp., *Endothia* sp., *Hypoxylon* sp., *Penicillium* sp., *Phialophora* sp., and *Trichoderma* sp. Though some of these fungi are known pathogens, we could not associate them with initial wilt symptoms. Only *C. fagacearum* was consistently associated with initial symptoms.

To test the pathogenicity of suspect fungi, we conducted inoculation experiments in growth chambers at controlled temperatures. Two- and three-year-old pot-planted live oaks inoculated with *C. fagacearum* at 26° C consistently developed wilt similar to that observed in the field. They did not develop symptoms when they were inoculated at 32° C. *Cephalosporium diospyri* did not produce wilt at either temperature, nor did it produce wilt in a greenhouse inoculation experiment with temperatures ranging from 22-35° C. In similar experiments, Kaufman (1978) was also unable to produce wilt from *C. diospyri* in non-stressed trees. Inoculations with different isolates of each fungus yielded the same results. Only *C. fagacearum* produced wilt in our 1977-1978 inoculation experiments.

*Ceratocystis fagacearum*, therefore, is the primary cause of live oak decline. Other fungi isolated from trees with advanced symptoms and inactive wilt are secondary invaders that follow the primary pathogen. Some of these secondary invaders are weakly pathogenic and capable of causing additional dieback in trees already stressed by oak wilt. The canker fungi are perhaps the most important among these secondary invaders.

**Canker fungi associated with decline**

We frequently isolated canker fungi from dieback in trees with advanced but inactive wilt symptoms. *Botryodiplodia theobromae* was the most frequently isolated canker fungus, but we also found *Dothiorella* sp., *Endothia* sp., and *Hypoxylon* sp. During 1977-78 we isolated *B. theobromae* from more than 100 live oaks with inactive wilt but active dieback (Lewis 1978). Inconspicuous cankers sometimes extended from dieback and could be detected only after we removed bark to expose dead cambium flanked by living tissues. The fungus sometimes killed large limbs and even boles but only in trees that had been stressed by wilt. Repeated attacks by *B. theobromae* sometimes killed these trees after 2 or more years.

We also isolated *B. theobromae* from trees that had not been stressed by wilt. During the summers of 1976-78 on the Lyndon B. Johnson Ranch, we isolated *B. theobromae* from terminals of recently killed small twigs on otherwise healthy live oaks. This twig loss has recurred annually in...
the same trees at the Ranch over the past 5
years. Slow thinning of healthy tree crowns by B.
theobromae can be considered a form of "slow
live oak decline," but it does not result in death of
large limbs or the tree itself.

We tested pathogenicity of B. theobromae in
healthy live oaks. When they were inoculated with
B. theobromae at 32° C, 3-year-old pot-planted
live oaks consistently developed cankers. No
cankers developed when the trees were in-
oculated at 26° C. In the trees inoculated at 32°
C, cankers sometimes girdled stems, killing them
above the inoculation wounds. There was,
however, little downward canker advancement.

Dothiorella sp., Hypoxylon spp. and Endothia
spp. were occasionally isolated from some of the
advanced dieback in declining live oaks. These
fungi did not produce cankers when healthy live
oaks were inoculated with them at 26° and 32°
C. It is possible, however, that they may cause
cankers, dieback, and even death in severely
stressed trees. If so, then they are part of the oak
decline complex.

Stresses that can be confused
with oak decline

Live oaks in central Texas are in an area of
relatively low rainfall. Though drought tolerant,
they are sometimes stressed by unusually long
dry periods. A slow rate of tree growth is the most
noticeable effect of the droughts. Comparatively
small leaves have also been observed in some
drought-affected live oaks. Low vigor due to
droughts might resemble decline, but vigor can be
restored by adequate water supply.

Leaf scorch is generally associated with
droughts, but we did not observe it in Texas live
oaks during 1976-1978. We did observe its
characteristic symptoms in post oak, Quercus
stellata, in central and east Texas and in Shumard
oak, Q. shumardii; southern red oak; Q. falcata;
and butternut hickory, Carya cordiformis, in east
Texas during the 1978 summer drought. The
symptoms were browning of leaf margins and in-
tervinal areas, with veins remaining green; bronzing
of leaves; and sometimes leaf curling. All
leaves were uniformly scorched on some trees,
but twigs remained green for a few weeks after
symptoms began. Later, we did observe twig
dieback in some of the drought-affected trees.
The new leaves that developed on the drought-
affected trees during late summer and early fall
were scattered and much smaller than normal.

Live oaks in central Texas are sometimes
defoliated and wounded by hail. Some live oaks
defoliated by hail at Stonewall, Texas, in April
1976 showed very thin crowns in August of the
same year but slowly recovered to near normal by
May 1978. Defoliation following a new flush of
leaves in the spring will stress trees, especially
during dry and hot years. Hail wounds on stressed
live oaks are potential infection courts for
pathogenic fungi.

Live oaks in central Texas are also affected by
nutrient deficiencies, whose symptoms might be
confused with decline. Van Arsdale (1977) has
describes these symptoms.

Other factors cause symptoms like those of oak
decline. Urban construction projects can prune
roots severely or produce earth fill and lower
grade around tree trunks. This damage may cause
decline similar to wilt. Some trees die; others
develop thin crowns and dieback but survive many
years.

Associated Insects

During 1977-78 we studied defoliating insects
to see if they contribute to the decline of Texas live
oaks. The forest tent caterpillar, Malacosoma
distria, sonoran tent caterpillar, M. tigris, and the
buck moth, Hemileuca maia, were the primary
defoliators in central Texas during spring.
Because only a few defoliators were present,
defoliation by these insects was light. Since the
live oak disease continued its rapid spread in trees
unaffected by insect defoliation, we could not
associate insect defoliations with the primary
decline of live oaks. Some years may be favorable
for defoliators, and defoliation might be heavy.
Like hail defoliation, insect defoliation will probably
stress trees, and they will appear to be declining
from an infectious disease during the summer.

Live oaks in Texas are also affected by several
species of leaf miners. Leaf miner, Phyllonorycter
basistrigella, damage is sometimes conspicuous
during summer and fall. At a distance the brown
and yellow blotches on leaves of heavily infested trees resemble wilt symptoms. Close examination of miner-infested leaves, however, revealed subcuticular tunnels and void spaces where mesophyll had been consumed by the insects. Also, small frass pellets could be seen in affected areas.

We have observed pruning and twig-girdling insects in live oaks. Girdled twigs in the crowns of healthy trees develop brown leaves before they break off. Individual twigs with wilting or brown leaves may be thought to have vascular wilt. Close examination of affected twigs revealed borer tunnels around the twig. Damage from these girders was light during 1976-78. If the same trees are repeatedly attacked by girders over several years, thin crowns resembling decline will become evident. Girdled twigs make excellent infection courts for canker fungi and may contribute to some infectious diseases of live oaks.

Summary and conclusion
Recent studies of live oak decline have suggested its main cause to be *Cephalosporium diospyri*. We were unable, however, to produce disease symptoms when we inoculated healthy live oaks with the fungus. *Ceratocystis fagacearum* (oak wilt) was the only pathogenic fungus that we consistently isolated from trees with active wilt, and it always produced symptoms in inoculated healthy trees. We concluded that it is the main cause of live oak decline in Texas. Some trees are killed by the wilt. Others are severely stressed by it and may be colonized and later killed by other fungi, especially canker fungi such as *Botryodiplodia theobromae*.

### Literature Cited