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LIVE OAK DECLINE IN TEXAS¹

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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 *Hyalodendron* sp. with rapid decline resembling oak wilt (1965). Van Arsdel (1970) identified live oak decline as a vascular wilt that he believed was caused by *Cephalosporium diospyri*. Van Arsdel 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-

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sistently 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.



Figure 1. Live oak defoliated by oak wilt in November 1977 at Kerrville, Texas.

The fungi most frequently isolated from trees with advanced symptoms were: *Botryodiplodia theobromae*, *Cephalosporium* sp., *Coryneum*, sp., *Dendrophoma* sp., *Dothiorella* sp., *Endothia* sp., *Hypoxyton* 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 *Hypoxyton* 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 inoculated 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 an 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 interveinal 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 Arsdel (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 disstria*, 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 girdlers was light during 1976-78. If the same trees are repeatedly attacked by girdlers 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*.

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