

## **OAK WILT: *Ceratocystis fagacearum* (Bretz) Hunt**

### **Order - Microascales; Family – Ophiostomataceae**

**DISTRIBUTION** - The oak wilt pathogen has not been found outside of the United States. It ranges from Texas and South Carolina, to Maryland and Pennsylvania, and into eastern Nebraska and central Minnesota. The pathogen is most common in the upper Midwest where it is believed it may have originated.

The pathogen was first identified in Wisconsin in the early 1940's, although disease survey records suggest that it may have been present in Minnesota and Wisconsin as early as 1912.

During the last 35 years the regional distribution of the oak wilt pathogen has changed very little, although its incidence has fluctuated sharply in many localities.

**DESCRIPTION** - Ascomycete. Mycelium produces rectangular endoconidia (3 micron x 6.5 micron average) formed at the tips of the hyphae and extruded one after the other. Conidia can be asexual or behave as spermatia. Heterothallic. Conidia of opposite mating types form perithecia. Ascospores form and are extruded in a drop of liquid from the tip of the long beak of the perithecia. Perithecia are produced on mycelial mats between the wood and the loosened bark. Imperfect stage - *Chalara quercina*.

**HOSTS** – All of the oak species that have been tested (36 species) have been proven to be susceptible to the oak wilt fungus. This includes oaks which were either naturally infected or artificially inoculated. Other susceptible tree species include: the Chinese, American, and Spanish chestnut, Allegheny and bush chinkapin, tanbark oak, and apple. Of these, only the Chinese chestnut was found to be naturally infected.

**DAMAGE** - Species in the red oak group are more susceptible to oak wilt than species in the white oak group. An infected red oak will commonly be completely wilted within a few weeks, whereas, an infected white oak may take two to four years to completely wilt, or even longer. Once a red oak is infected it never recovers, but one third to one half of infected white oaks may recover and grow normally.

Infected red oaks typically begin to show symptoms of wilting and bronzing of leaves and premature defoliation of branch tips in the upper crown. The wilt symptoms progress rapidly down through the crown, turning the outer portion of the leaves a dull green, bronze, or tan color. The affected leaves often show a distinct line of transition between the necrotic and normal green tissues. This discoloration progresses toward the leaf base and the midrib, creating what appears as a "green island" of healthy tissue at the basal end of the leaf.

Infected white oaks typically display symptoms which are more subtle. Early symptoms are often characterized by the wilting of foliage of individual branches. Leaves may turn yellow, but necrosis is usually limited to the margins of the leaf blade. Affected leaves may resemble normal autumn coloration. The death of individual branches over a period of several years can often result in a "stag-headed" appearance of the crown.

Areas located at the southern fringes of the oak wilt range (Texas) display somewhat different symptoms. Infected foliage typically becomes chlorotic or bronze, often with a yellow to brown color following along the veins of the leaves. An infected tree may defoliate and die quickly, but commonly survives for several years, displaying a progressive dieback of twigs and branches, and producing adventitious sprouts with small leaves on the trunk and larger limbs.

The oak wilt fungus invades the water-conducting tissues of the tree and is restricted to the outermost sapwood. An infected oak responds to the attack by developing balloon-like projections (tyloses) from cells lining the water-

conducting vessels. As the tyloses plug the vessels, the movement of water within the tree becomes increasingly restricted. This causes the outer- most ring of sapwood to turn a dark brown or black, appearing as streaks when the bark of an infected branch is peeled or as a ring when the branch is cut in cross-section. It is this blockage that causes the foliage to wilt and die.

The oak wilt fungus has the potential to be one of the most destructive of all tree pathogens; however, the devastation that was once so greatly feared has not occurred. So far the spread of the disease has been slow and erratic.

**LIFE CYCLE** - The oak wilt pathogen is strictly a vascular fungus. It has been isolated from the xylem of roots, trunks, branches, twigs, and even leaf petioles. The fungus can be transmitted to healthy oaks either by root grafts or by insects carrying spores.

It has been estimated that over 90 percent of the diseased trees probably become infected through root grafts. The passive transmission of spores through the root system is very slow, and can take up to several years to occur. The fungus may remain alive in dead trees for up to four years within the roots. Trees as much as 50 feet apart may be grafted together. After the infection of the original tree, trees adjacent may wilt one to six years later.

In the upper Midwest, sap beetles, sometimes referred to as picnic beetles (family Nitidulidae) are the predominant insect vector of the disease, and in the south, oak bark beetles are considered to be an important vector. Neither of these vectors are very efficient in the transmission of oak wilt. However, the European oak bark beetle, *Scolytus intricatus* Ratzeburg, displays the potential of becoming an excellent vector of the oak wilt fungus. Care should be taken that this and other similar exotic insects do not become established in North America.

Infected oaks wilt anytime between late spring and late summer. After the leaves die, the fungus also dies within the twigs and small branches. The fungus can survive as long as one year within the trunk and tends to survive longest on the lowest part of the north side of the trunk. This is because the fungus is unable to tolerate heat above 32 degrees centigrade and requires a high level of sapwood moisture. Trees that wilt in the first half of the summer (up to mid July) tend to produce fungal mats in late August or September, while those that wilt later in the summer produce fungal mats the following spring.

About two to three months after the defoliation of an infected tree, the mycelium of the fungus begins to grow extensively in the xylem vessels. It grows both into the xylem and outward towards the cambium. At the cambium, the fungus may form a sporulating mat characterized by a layer of mycelia and conidiophores surrounding a raised pressure "pad" or "cushion". As the fungal mat cracks the bark from the sapwood, a fruity odor is emitted. This odor can be highly attractive to sap-feeding insects, especially sap beetles. The sticky spores of the fungus then become attached to the bodies of the beetles. When the beetles fly to healthy oak trees and feed on the sap flow from fresh wounds (as a result of pruning, iron climbing cleets, insect damage, etc.), the spores of the fungus are transmitted. It also has been suggested that sap beetles may act as agents of fertilization in that they could transport conidia of type A mats to type B, and visa versa. This would then allow for the development of perithecia and the production of ascospores.

The development of fungal mats is much more common in the northern part of the oak wilt range than in the south. Also it is important to recognize that fungal mats are rarely produced on tree species in the white oak group.

Oak bark beetles (considered to be important vectors in the south) behave as tree wounding vectors. These beetles breed hi the wilt killed trees and feed on the twigs of healthy oak.

**INSPECTION TIPS** - For species in the red oak group, look for the rapid wilting of the entire crown of the tree (within a few weeks). Wilted leaves typically show distinct lines of transition between the necrotic distal ends and the healthy green tissue ("green islands") at the bases. Fungal mats may be visible beneath bark areas which have been split open.

For species in the white oak group, look for the wilting of individual branches and a "stag-like" appearance of the crown. The vascular discoloration is usually very easily seen in cross- section cuttings of infected branches.

For both white and red oaks, look for dead trees or stumps nearby (within 50 feet) and areas on the tree which might represent fresh wounding.

Since diagnosis may be difficult at times, it is helpful to submit samples for laboratory testing. Samples should consist of several branch segments one-half to one inch in diameter and six to ten inches long. Branch segments should show oak wilt symptoms and be living (the fungus cannot be isolated from dead wood). Samples should be sealed in plastic bags and kept cool and dry prior to shipment.

**CONTROL TIPS** - There are no fungicides available to prevent the spread of the oak wilt fungus. The use of insecticides to control vector populations is not feasible.

The best method to prevent the spread of the fungus is by breaking the root grafts. A vibratory plow with a 5-foot blade can effectively cut the root systems of the oak trees, segregating the infected roots from the roots not infected. Often two lines are recommended: a primary line outside of apparently healthy trees and a secondary barrier outside of every obviously infected tree. If buried utilities are a factor, the soil sterilant Vapam can be used, but it is not nearly as effective as the mechanical barrier.

Tree wounding (pruning) should be avoided from April 15 to July 1. If wounding is unavoidable, a nontoxic tree wound dressing should be applied immediately after wounding. Tree climbing irons should never be used on living oak trees.

Deep girdling red oak trees at the base speeds up bark drying and can prevent fungal mat formation. Since white oak species rarely produce fungal mats, girdling is not necessary on white oaks.

The selective pruning of diseased branches may aid the recovery or prolong the survival of highly valued trees in the white oak group.

#### **REFERENCES -**

**Alexopoulos, C. John.** (1962) *Introductory Mycology*. Second Edition. John Wiley and Sons, New York. 613 pp.

**French, D. W..** Oak Wilt in Minnesota. Minnesota Extension Service, University of Minnesota AG-MI-3174. 6 pp.

**Gibbs, J.N., and D. W. French.** (1980) The transmission of oak wilt. U.S. Forestry Service Research Paper NC-185. 17 pp.

**Gleason, M. L.** (1987) Oak wilt. Cooperative Extension Service, Iowa State University. PM-482. 6pp.

**Gleason, M. L.** Oak wilt. Cooperative Extension Service, Iowa State University. Meeting handout. 2 pp.

**Riffle, J. W., and G. W. Peterson.** (1986) Tech. coords. Diseases of trees in the Great Plains. U.S. Forestry Service General Tech. Rep. RM-129. 149 pp.

**Sinclair, W. A., H. H. Lyon, and W. T. Johnson.** (1987) Diseases of trees and shrubs. Cornell University Press. 574 pp.

**PREPARED BY** - William R. McAdams, Iowa. 1989.

**PHOTOGRAPHS -**



**Top left:** Oak tree succumbing to oak wilt. –

Joseph O'Brien, USDA Forest Service, Bugwood.org



**Top right:** Ceratocystis fungal mat exposed after the bark is peeled away.

Fred Baker, Utah State University, Bugwood.org

**Bottom right:** Pine wilt infected branch sliced to show the discolored vascular tissue.

Robert F. Bassett, USDA Forest Service, Bugwood.org

