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MAJOR TREE DISEASES OF THE CENTURY[1]

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Tree diseases may be divided into three types: foliage diseases, canker diseases, and wilt diseases. Most foliage diseases are not fatal and can be controlled by fungicides. Many fungi that cause canker diseases attack mainly trees under stress. Maintaining trees in a vigorous growing condition will prevent these canker diseases such as Cytospora canker of spruce and Melanconium canker of birch. However, some canker-producing fungi are highly virulent and will attack trees regardless of vigor, such as chestnut blight and white pine blister rust. Most wilt diseases are caused by organisms that are highly virulent and for which it is difficult to develop controls. The more destructive and widespread wilt diseases include elm phloem necrosis, Dutch elm disease, Verticillium wilt, and oak wilt. The six major diseases of the past century which I will briefly discuss are dwarf mistletoe, chestnut blight, white pine blister rust, elm phloem necrosis, Dutch elm disease, and oak wilt.

Dwarf Mistletoe

Dwarf mistletoes (*Arceuthobium spp.*), are highly specialized seed-producing plants that parasitize trees in the Pine Family (Pinaceae). They are short (1/4" to 1" long), leafless, brownish green shoots, and produce colored fruits that are forceably shot into the air. There are 28 species in the New World and 4 species in the Old World. In North America, dwarf mistletoe ranges from central Canada and southeastern Alaska to Honduras. However, most species are found in the western United States and Mexico. Only one species occurs in eastern North America.

Although dwarf mistletoes affect many western conifers, it is reported to cause widespread damage to lodgepole and ponderosa pines. It has been estimated that dwarf mistletoe causes an annual loss of 55 to 75 million board-feet of ponderosa pine in Arizona and New Mexico and an annual loss of about 9.6 million cubic feet of lodgepole and jack pine in Alberta.

The plants of dwarf mistletoe are either male or female and both types cause damage. Seeds are produced only on the female plant. The viscous-coated seed is produced in a berry. Pressure builds up in the berry as it ripens during the summer. This pressure forces the seed from its stem and it is shot 20 to 30 feet into the air at a speed of 60 miles per hour. Because of the viscous (gluey) coating, the seed sticks wherever it lands. Many seeds become attached to the needles of the same or of adjacent trees. They can be carried long distances by birds. The seeds are washed by rain onto the twigs in the fall. They germinate in late winter or spring and penetrate the bark tissue of the host during the summer. By the second summer the infected tissues swell and one year later (third summer) shoots of the dwarf mistletoe appear. The mistletoe develops into either male or female plants during the fourth summer. Pollination occurs in the fifth year followed by production of fruit in the sixth year which mature by September when the seed is forceably liberated to start the 6-year life cycle again.

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Most infection by the dwarf mistletoe takes place in segments of branches less than 5 years old. It produces a root-like growth which ramifies throughout the bark. Some parts of the root-like growth may penetrate successive layers of the sapwood. Affected branches swell and become spindle-shaped (fusiform). Excessive twig formation occurs around the swollen areas to form witches'-brooms.

Dwarf mistletoe weakens affected trees by robbing them of water and nutrients. Affected trees gradually die from the top down and may be killed over a period of years. The rate of dying is influenced by the number of mistletoe plants in the tree and the vigor and age of the tree when it first became infected.

Since dwarf mistletoe spreads slowly, cutting all affected trees will eliminate the parasite. The procedure may result in clear-cutting of large areas of timber in some forests. Forests only recently invaded by dwarf mistletoe may be protected by removing diseased branches as trees become affected. To save affected trees of high value, all infected branches are removed by cutting them back flush with the trunk. Trunk infections are not likely to be eliminated by cutting out the diseased tissues, since mistletoe root systems extend several inches inside the affected branch beyond the visible mistletoe shoots. Such trees are removed. Pruning is done in the summer when the mistletoe shoots are more easily seen and before the berries are ripe. Dispersal of seed starts about mid-August. It has been found that some individual trees remain free of mistletoe. Such trees may be resistant and can be utilized in trying to develop host strains resistant to the disease.

Chestnut Blight

Chestnut blight, also called Endothia canker, is caused by the fungus *Endothia parasitica*. It is believed that this fungus was brought to North America on Asiatic chestnut planting stock. Chestnut blight is one of the most destructive tree diseases in North America. It has eliminated the American chestnut which was a major forest tree in the eastern United States. Trees susceptible to chestnut blight in addition to the American chestnut (*Castanea dentata*), include European chestnut (*C. sativa*), Eastern chinquapin (*C. pumila*), and post oak (*Quercus stellata*). The Asiatic species, especially the Chinese chestnut (*C. mollissima*), are resistant to blight. The fungus has been observed growing saprophytically on various woody hosts including red maple, shagbark hickory, staghorn sumac, and various species of oak.

Chestnut blight was first observed by H. W. Merkel of the New York Zoological Park in 1904. He noticed that large numbers of chestnut trees were dying in the City of New York. Even at that time it was evident that the disease was spread over Nassau County and in adjacent counties in Connecticut and New Jersey. By August of 1907, the disease was known to occur as far south as Trenton, New Jersey and as far north as Poughkeepsie, New York. The disease continued to spread throughout the natural range of the chestnut and into the Midwest and West. By 1965 it had been reported on chestnut trees planted in California, Oregon, and Washington, as well as in Illinois, Iowa, Michigan, Missouri, and Wisconsin.

Spores of the fungus are carried by wind, rain, insects, and birds. Wind and rain are important in local spread while insects and birds can carry the fungus spores for long distances. Also, the fungus can be distributed long distances by the transporting of diseased nursery stock. Infection occurs through wounds in the bark.

Development of chestnut blight is indicated by yellowing and browning of leaves on affected twigs and branches. Dead leaves and seed burs cling to diseased branches and are conspicuous during the dormant period. Young cankers develop as yellowish-brown oval to irregular areas on smooth-barked, vigorous-growing young stems. They appear as brown, circular to irregular dead areas with slightly depressed or raised margins on slow-growing or old stems with smooth bark. Girdling of stems by these cankers result in death of the parts beyond the affected regions. Usually trees die within 2 to 4 years after they have become diseased.

Efforts to control chestnut blight were started soon after discovery of the disease. It was recommended that diseased trees be removed

and destroyed and that infected chestnut products, mainly nursery stock, be quarantined. It soon became evident that efforts to prevent spread of the disease were ineffective. Starting in 1913, a search was made in Asia for resistant chestnuts that might be satisfactory substitutes for the American chestnut. Also, an effort was made to find American chestnut trees and sprouts that might be resistant to the disease. So far there is no evidence of resistance in the American chestnut.

Of the Asiatic species tested, the Chinese chestnut is most resistant to the disease and it withstands climatic conditions of the Midwest. Crosses of the Chinese chestnut with the American chestnut are resistant to the disease. However, these trees are more suitable for ornamental plantings and nut production than for forest trees.

White Pine Blister Rust

This disease, caused by the fungus *Cronartium ribicola*, is believed to have originated in Asia where it affected Swiss pine and to have spread gradually to Europe. It has been reported in central China and Japan. The eastern white pine of North America was introduced into Europe in 1705 and white pine blister rust was not discovered there until 1854 when it was found in the Baltic provinces of Russia. By 1890 it had spread over northern and most of western Europe. It was found in England in 1892. For all practical purposes, white pine blister rust eliminated the white pine in England within a period of 20 years.

In North America, blister rust was present in small quantities in New England as early as 1898. The fungus was found affecting *Ribes* the alternate host, at Geneva, New York in 1906 and affecting eastern white pine in 1909. Pines found affected in 1909 had been obtained from a German nursery and they were widely distributed throughout the Northeast. Additional diseased trees were found in 1910 in eastern white pine stock obtained from several French nurseries.

The disease was discovered on eastern white pines in Vancouver, British Columbia, in 1921 on trees imported from Ussy, France in 1910. From this one introduction it has spread throughout the range of the western white pine in British Columbia, Washington, Oregon, and northern California. In the United States, white pine blister rust is widespread in the north-central, northeastern, and northwestern states.

White pine blister rust affects the majority of the 5-needle or white pines. The western species are more susceptible than the eastern species of white pine.

The blister rust fungus invades and kills the needles. It grows from the needles into the bark of twigs and branches, where it produces swollen, oval cankers. As these cankers enlarge, they girdle and kill infected stems. These cankers may be conspicuous because of the outflow of resin that flows down over the smooth bark below the canker. In time the resin turns white. In late spring, orange to yellow blisters or fruiting bodies of the fungus break through fissures in the diseased bark. Spores produced in these fruiting bodies infect leaves of currants and gooseberries (*Ribes*). Small orange to yellow pustules are produced on the leaves of these shrubs during the summer. The spores produced in these pustules cause new infections on white pine. The life cycle of the blister rust fungus requires 3 to 5 years, usually 4, to be completed. It is on the *Ribes* during only one growing season.

During the 1920's, it was observed that secondary fungi invaded bark cankers. They grew rapidly throughout the diseased bark and crowded out or often killed the blister rust fungus. Also, rodents including squirrels and field mice were observed to eat the diseased living bark more or less completely in the cankered areas. These activities reduced the production of spores by the blister rust fungus but did not eliminate the disease.

Removal of infected branches will eliminate individual infections but will not prevent new infections. The control practice vigorously followed in many areas was the systematic removal of the *Ribes*. In the early years this was done by digging out the plants. By 1930 various chemicals had been tested and it was found that *Ribes* were killed by sprays of sodium chloride and by sodium hydroxide. Present control of blister rust should include consideration of the following procedures: 1) microclimate rela-

tions, 2) chemical control with antibiotics or other effective materials, 3) pruning to eliminate infected branches, especially on pines in ornamental plantings, and 4) eradication of *Ribes* where feasible. It was found that a basal spray of 2,4,5-T in No. 1 fuel oil was effective in eradicating *Ribes missouriense* during any season of the year. Blister rust does not cause serious losses and can be largely ignored in most sites in the low-rust-hazard zones. Avoid planting in sites characterized by a microclimate favorable for rust. Maintain an overstory over juvenile white pine.

Elm Phloem Necrosis

Elm phloem necrosis, described as a virus disease in 1938, was reported to be caused by a mycoplasma-like organism in 1972. It is a native disease. Dying of elms in conspicuous numbers in Kentucky was reported as early as 1893. Later research showed that seed obtained from elms in Kentucky that survived the early dying gave seedlings that were resistant to phloem necrosis. A similar dying of elms was reported in southern Illinois in 1912. In Ohio, the dying of elms at Ironton in 1918, at Dayton in 1927, and at Chillicothe in 1935 was believed to be caused by phloem necrosis. In 1942 phloem necrosis was widespread in Kentucky, the southern halves of Ohio, Indiana, and Illinois, southeastern Missouri, northwestern Tennessee, and western West Virginia. By 1946 it had spread into southeastern and southwestern Iowa, southeastern Nebraska, eastern Kansas, and northeastern Oklahoma and Arkansas. It was present in a few limited areas in Mississippi, extending as far south as Jacksonville. The northern limits of the disease was roughly along a line extending from Columbus, Ohio through Indianapolis, Indiana, and Peoria, Illinois to Burlington, Iowa. Phloem necrosis was not found in other states until 1971 when it was discovered at Ithaca, New York and in Lycoming County, Pennsylvania. It was found in New Jersey in 1973. Phloem necrosis is now known to occur in 19 states.

Phloem necrosis is a destructive vascular disease of American elm, including the Augustine ascending, Moline, vase, and holly-leaf varieties. It can affect the winged elm. Many slippery elms (*Ulmus rubra*) in the State of New York displayed foliar symptoms and fibrous root necrosis typical of phloem necrosis. Cedar elm (*U. crassifolia*) and September or red elm (*U. serotina*) were listed susceptible to phloem necrosis in Mississippi.

The first symptom of phloem necrosis is the dying of fibrous roots. This is followed by foliage symptoms which appear as drooping of leaf blades or curling upward of leaf margins. In time the leaves turn yellow then brown and drop. Some leaves drop when they become yellow. These symptoms may occur over one or more growing seasons. On some trees the leaves wilt within a period of a few weeks, turn brown and many leaves remain attached to the branches. Many elms which show leaf symptoms after early August live over winter, produce a sparce crop of leaves the next spring, and die in late June or July. Since these leaf symptoms frequently can be confused with those caused by other elm diseases, field diagnosis of phloem necrosis is based on the color and odor of the inner bark. In an affected tree, the thin layer of inner bark (current season growth) in contact with the sapwood, especially that at the base of the trunk and in the buttress roots, becomes yellow to butterscotch in color. Occasionally, dark brown to black flecks are evident in the butterscotch-colored bark. The butterscotch color can be detected only in freshly cut samples of bark, since the inner bark from both diseased and healthy trees turns brown within a few mintues after being exposed to air. The odor of wintergreen emanates from the butterscotch-colored bark of trees affected with phloem necrosis.

Healthy elms may be protected from infection by spraying with methoxychlor to control the elm leafhopper which carries the phloem necrosis causal organism from diseased to healthy trees. Spray trees twice during the growing season to obtain maximum protection. Apply the first spray as soon as the spring leaf crop is fully mature, usually during June in the Midwest. Apply the second spray immediately after the midsummer or second growth of elm leaves has occurred, usually after July 15. Each spray should contain 6 per cent insecticide if applied

by a mist blower or 1 per cent insecticide if applied by a hydraulic sprayer.

The effectiveness of spraying to prevent the spread of phloem necrosis will not be known for a year after the spray program has been started, as trees are infected with the causal organism a year or more before they show the disease. No tree already infected when the sprays are applied will be benefited by the sprays. For continuous protection, trees must be sprayed each year. Success in preventing phloem necrosis depends on spraying with such thoroughness that no leafhopper that fed on a diseased tree is left alive long enough to feed on a healthy tree.

Dutch Elm Disease

Dutch elm disease, caused by the fungus *Ceratocystis ulmi*, was first observed in Rotterdam, the Netherlands in 1919. The origin of the disease is not known. However, circumstantial evidence indicates that it most likely originated in Asia since the Asiatic species of elm are resistant to the disease. The disease soon became widespread throughout Europe. It had spread over Belgium and into Germany and northern France by 1921. It was reported in Norway in 1926 and in England in 1927.

In the United States the first diseased elms were discovered at Cleveland and Cincinnati, Ohio in 1930. Identification of the fungus was made independently by Christine Buisman, a Dutch plant pathologist, and by Curtis May, a plant pathologist at the Ohio Agricultural Experiment Station. The fungus was probably brought to the United States in elm burl logs imported from Europe. Logs infested with the fungus entered the United States at New York City, Baltimore, Norfolk, and New Orleans. They were shipped to or through Cleveland, Cincinnati, Dayton, Indianapolis, Chicago, Kansas City, Louisville, Knoxville, and other cities. Many of the logs intercepted were infested with elm bark beetles. An infestation of the smaller European elm bark beetle (*Scolytus multistriatus*) was reported near Boston in 1909. To prevent additional diseased logs being shipped into the United States, a quarantine was established October 1933.

Following the discovery of diseased trees in Connecticut, Maryland, New Jersey, and New York in 1933 and at Indianapolis, Indiana in 1934, federal, state, and local agencies initiated programs to control and ultimately eradicate the disease. These programs slowed the spread of the disease but they did not eradicate it. By 1940, the disease was present in seven eastern states in addition to Ohio and Indiana. During World War II, funds for these programs were no longer available. During and following the war years the disease spread into additional states and by 1950 it was present in 19 states and the District of Columbia. By 1960 it had spread to six additional states and by 1973 it was present in 40 states. The ten states in which it had not been reported by 1973 were Alaska, Arizona, California, Florida, Hawaii, Louisiana, Nevada, New Mexico, Utah, and Washington.

In Canada, the first trees affected with Dutch elm disease were found in St. Ours, Quebec, in August 1944. It is believed that the fungus was brought from Europe on crates made of diseased elm wood that were shipped to Sorel. The disease apparently spread from Quebec to Ontario where it was found at St. Isidone in 1946 and from Maine to New Brunswick where it was found at Woodstock on the St. John River in 1957. It was reported in Nova Scotia in 1969.

No species or variety of elm is known to be immune to Dutch elm disease. Trees in the related genera of *Planera* and *Zelkova* have become diseased when inoculated with the Dutch elm disease fungus. It is unfortunate that the American elm, a long-time favorite for both shade and ornamental use, is the most susceptible of all elms. Although Chinese and Siberian elms are highly resistant to the disease, a few trees of these species have succumbed to natural infection.

The first noticeable symptoms of Dutch elm disease are wilting, curling, and yellowing of leaves on one or more branches, a condition often called "flagging." These symptoms are followed by dying and browning or premature falling of leaves, and death of affected branches, followed by wilting of leaves on additional branches and finally death of the affected trees. Elms affected in this manner in early summer

may die during the same growing season. Those affected in late summer may die during the winter, soon after leaves appear in the spring, or slowly over a period of a year or more. Other elms may show foliage wilt on most or all of their branches at one time and die within a few weeks. Trees that wilt and die rapidly probably became infected during the previous growing season, at which time they would have shown no wilt symptoms or only limited, relatively inconspicuous symptoms.

Brown streaking develops in the sapwood of diseased branches. It appears mostly in the spring wood of the current-season growth. In a cross-section of a branch, browning may appear as a series of dots in a single wood ring or the dots may be so abundant that the entire wood ring appears brown. In branches on which leaves wilt before summer wood is produced, the discoloration is usually conspicuous as fine streaks on the surface of the wood when the bark is carefully peeled from the diseased branch. The outer surface of sapwood on trunks may also be brown.

In North America, the fungus is transmitted by the smaller European elm bark beetle, *Scolytus multistriatus*, and the native elm bark beetle, *Hylurgopinus rufipes*, and through grafted roots between diseased and healthy elms. The smaller European elm bark beetle, the principal carrier of the fungus, feeds mainly in crotches of 1- and 2-year-old twigs. The fungus spores, deposited in the feeding wounds, grow and spread in the vessels of the young sapwood. Infection occurs mainly in May and early June, when the spring wood vessels are adjacent to or near the inner surface of the bark. In time the leaves on the infected branches wilt and die.

Methods of trying to control Dutch elm disease may be divided into four categories—1) preventing spread of the fungus from diseased trees, 2) protecting healthy trees, 3) curing diseased trees, and 4) producing elms resistant to the disease. Since discovery of the disease in the United States in 1930, numerous procedures and thousands of chemicals have been tested by federal and state personnel. The chemicals tested include fungicides, insecticides, attractants, repellants, antibiotics, and growth regulators. The procedures include applying materials to the foliage or to the soil, and implanting chemicals in the trunk. The present recommendations which have resulted from the various phases of research include—1) planting resistant elms wherever feasible, 2) employing thorough and effective sanitation to suppress the bark beetle population, 3) spraying with an insecticide to protect healthy elms, and 4) using a soil sterilant to prevent transmission of the fungus through grafted roots between diseased and healthy trees.

There is intense interest at present on chemicals that can be introduced into trees which will prevent healthy elms from becoming diseased and which will cure the disease in elms that show the very early symptoms of foliage wilt.

Oak Wilt

It is believed that oak wilt, caused by the fungus *Ceratocystis fagacearum*, originated in the upper Mississippi Valley of the United States. Although the cause of oak wilt was not reported until 1942, dying of black oaks in Madison, Wisconsin was reported as early as 1881. By 1912 oaks were reported wilting and dying in Minnesota as well as Wisconsin. During the early 1920's many foresters and farmers observed the dying of oaks in a manner similar to the way oaks wilt and die in active oak wilt areas. By 1940 extensive wilting of oaks was conspicuous in southern, southwestern, and western Wisconsin, northeastern Iowa, and northwestern Illinois. It was reported in Pennsylvania in 1950. In 1951, numerous centers of oak wilt were observed in Ohio and the disease was discovered in Maryland, Michigan, Virginia, and West Virginia. The disease continued to spread and is now present throughout much of the oak range in the eastern United States. Hosts of the oak wilt fungus include 36 species of oak and 6 closely related species. These 6 species are American, Chinese, and Spanish chestnut, bush and Allegheny chinquapin, and tanbark oak.

Research on oak wilt was stimulated greatly by funds made available through the National Oak Wilt Research Committee. This committee, composed of members of the hardwood industry, was formed in 1950. This group of private

operators gave $50,000 annually for 5 years to support research on the disease. This money was made available to state universities or other state agencies in the following 6 states: Illinois, Iowa, Minnesota, Missouri, Ohio, and Wisconsin. When funds were no longer available from the hardwood industries, federal funds were made available in 1955.

Oak wilt appears first as wilting of leaves on branches in the upper portion of the crown of an affected tree. The wilt progresses downward and inward until all of the foliage is affected. Leaves on trees in the red oak group become dull or pale green in color, and the margins may curl upward. These symptoms are followed by yellowing or bronzing of the leaf tissues; the discoloration spreads from the margins toward the midribs of affected leaves, which may fall at any stage of wilt. Mature leaves usually remain stiff and fully expanded during the different stages of wilt and for some time after death. Immature leaves curl, droop, turn dark brown to black, and remain attached to the branches. Leaves on wilting bur oak and white oak usually turn light brown to straw color, curl, and remain attached to the branches.

In trees of the red oak group, brown to black discoloration usually develops in the current-season sapwood of wilting branches. This discoloration may appear as streaks or as diffused browning of individual wood rings. Similar sapwood discoloration has been observed occasionally in wilting bur oak trees.

Affected trees in the red oak group may wilt and die in 4 to 6 weeks, or during a single growing season. Trees of the white oak group usually die slowly over a period of years.

The oak wilt fungus is transmitted over long distances (overland spread) mainly by sap-feeding insects. Underground spread occurs through grafted roots between diseased and healthy trees. This type of spread occurs mostly between trees that are spaced 30 feet or less apart.

Various approaches have been tried in efforts to control oak wilt. Hundreds of chemicals have been tested in attempts to protect healthy trees and to cure diseased trees. Also, various procedures have been utilized to control the disease including 1) felling and spraying diseased trees with a mixture of persistent insecticides to reduce insect invasion and to kill fungus-carrying insects as they emerge, 2) the same procedure plus removal of all oaks of the same species within 50 feet of diseased trees, 3) deep girdling into the heartwood of each diseased tree and removing all bark below the girdle to the groundline to prevent or reduce fungus-mat formation, 4) mechanically breaking grafted roots between diseased and adjacent healthy trees within root grafting distance, and 5) killing all healthy trees within root grafting distance of diseased trees by poisoning. Usually, this means killing all living oaks within 30 feet of a diseased oak.

Before utilizing any of these procedures it is necessary to locate the diseased trees. This can be accomplished by a combination of aerial and ground surveys. Two or more such combined surveys annually are necessary to locate the wilting oaks during the growing season. A continuous survey and eradication program should prevent local spread of the fungus.

Conclusions

Of the hundreds of diseases that cause damage to trees the six described here are among the most destructive and widespread. They have caused extensive losses in forest, shade, and ornamental plantings. Although a tremendous amount of research has been carried on by scientists in federal and state agencies, there is much more to learn about each disease and especially about how to economically and effectively control them. This research is continuing and there is hope that new methods and materials will be effective and economically feasible for commercial use.

In addition to working to control the present diseases we must realize that new diseases may appear in the future. We must be alert for such diseases in the hope that effective controls can be developed quickly to prevent the great losses that have occurred in the past.

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