Anthracnose

Overview: Anthracnose is a general term for a group of diseases on hardwoods that cause lesions on leaves, twigs, and fruits. Generally, these diseases are cosmetic and cause no serious damage to the tree (but there are some exceptions). There are many species of fungi known to cause anthracnose, but most only infect one or a few specific host species. Therefore, these diseases are named for the tree species on which they may be found (Fig. 1-6). For example, the most common anthracnose diseases in North Carolina include sycamore anthracnose, oak anthracnose, maple anthracnose, and dogwood spot anthracnose. Disease cycles and control strategies are similar regardless of the causal organism; therefore specific identification of the fungal species is not usually necessary. Dogwood anthracnose (not to be confused with dogwood spot anthracnose) is a serious disease of dogwood, and most often results in the death of the host; the following information does not apply to dogwood anthracnose (*see Dogwood Anthracnose*).

Causal Agent: Fungus (*Gloeosporium* spp., *Gnomonia* spp., and *Apiognomonia* spp.)

Hosts: A wide variety of hardwoods. Common hosts include oak, maple, sycamore, ash, walnut, and dogwood.

Symptoms / Signs: Symptoms vary with species, but in general the most obvious symptoms are the leaf lesions produced in the spring and expand throughout the summer. Lesions often begin as pale green or greenish-grey blotches, but then turn yellow, tan, reddish-brown, or brown. Often lesions have a distinct, colorful margin.

Lesions tend to begin along leaf veins (because the depressions along veins hold water for a longer period of time and spores tend to collect there), but often rapidly expand. Expansion often follows the soft tissues adjacent to veins, and may result in the coalescing of many lesions. Severely infected leaves may have a scorched appearance, becoming almost completely brown, wilted, or cupped. (*Note: drought symptoms and/or leaf scorch differ because the browning and wilting of leaf tissue begins at the leaf tips and leaf margins and progresses inward*).

Some trees respond to infection by prematurely shedding leaves (e.g. sycamore and ash), but others retain their leaves until normal leaf drop in the fall (e.g. oak). In sycamore, the fungus is able to grow out of leaves into adjacent twigs where it causes small cankers, shoot dieback, witches brooms, and/or deformed twigs.

Disease Cycle: Most anthracnose fungi infect their hosts during the spring, just as the first new leaves begin to expand; infections continue through the summer while environmental conditions are suitable. Spores are released from last year's diseased tissue (most commonly from fallen leaves). Spores can be spread by wind or rain-splash, and can only infect soft, succulent tissues such as new shoots, flowers, and fruits, but leaves are the most severely infected. The fungus obtains nutrients from plant cells, and in turn the cells are killed creating the leaf lesion. The lesion expands as the fungus spreads. During periods of sustained leaf wetness and cool temperatures, spores are produced from leaf lesions which can re-infect the same leaf or neighboring leaves. New infections usually do not occur after mid-summer. Most anthracnose fungi over-winter on the ground in fallen leaves, but some (e.g. sycamore anthracnose) can also spread from the leaves into adjacent shoots and over-winter in the twigs where they directly infect new leaves in the spring. Anthracnose fungi that spread from leaves into twigs may cause small cankers on woody tissues adjacent to the infected foliage.

Importance: Low. Anthracnose diseases are generally cosmetic and cause no serious damage to the tree. In years of severe disease, trees can become unsightly or even appear to be dying, causing great concern to homeowners.

Management: Management for anthracnose is usually not necessary as it causes no serious harm to the tree; however, aesthetic concerns may warrant an attempt to reduce disease severity. Plant trees on a wide spacing, keep the surrounding area clear of vegetation, and prune properly to improve air circulation within the crown. Raking and destruction of leaves in the fall is often very effective in reducing disease incidence the following spring. Fungicides are available for small ornamentals and shade trees.

Timeline: Infection occurs in early spring and may continue through early summer. Symptoms begin to develop shortly thereafter; some tree species may begin to lose infected leaves during the summer months.

Range: Statewide.

Fig. 1 Sycamore anthracnose

Fig. 2 Sycamore anthracnose

Fig. 3 Maple anthracnose

Fig. 4 Dogwood spot anthracnose

Fig. 5 Ash anthracnose

Fig. 6 Oak anthracnose

Brown Spot Needle Blight

Overview: Brown spot needle blight is the most important disease of longleaf pine in the southeastern United States. The blight kills needles and slows growth, and is particularly important because it reduces seedling survival after planting and prolongs the period of time seedlings spend in the grass stage. Brown spot has little or no impact on trees more than 10 feet tall. Warm and wet weather favors this disease.

Causal Agent: Fungus (*Mycosphaerella dearnessii*; syn. *Scirrhia acicola)*

Hosts: Longleaf pine. Other southern pine species may become infected, but disease symptoms are usually not severe (*see Needle Cast*). Brown spot can cause significant damage to Scots and Ponderosa pines grown for Christmas trees, and on many ornamental and exotic pine species.

Symptoms / Signs: Usually only seedlings (Fig. 1), small saplings, and the lower branches of larger trees are affected by brown spot (Fig. 3). Lesions on needles can appear at almost any time of year, but most commonly between May and October. Lesions start out as a small grayish-green spot; then turn yellow. As the lesion expands, the inside of the spot will turn brown and have a yellow or chestnut brown margin (Fig. 2). Lesions may expand all the way around the needle, or coalesce with other spots. Girdled needles will then turn brown and die from the tip of the needle down to the lesion, but the base of the needles will stay green. Needles with many spots will have a banded or mottled appearance. Eventually infected needles will be killed. Dead needles turn reddish-brown and then fall off. If infections are severe and happen for many consecutive years, seedlings may develop a needle-free stem with only the terminal bud surrounded by a tuft of green needles.

Fruiting bodies that release spores are produced throughout the year and will appear as small black dots within lesions (Fig. 4).

Disease Cycle: The brown spot fungus overwinters in the lesions of both living and dead needles, and produces both sexual and asexual spores in the South. Sexual spore production begins in early spring, peaks in late summer, and is halted during the winter months. Infections resulting from sexual spores are usually very mild. Asexual spores can be produced on both living and dead needles, and may be produced throughout the entire year. Many generations of asexual spores are produced annually, resulting in rapid disease buildup. Fruiting bodies produce and expel spores in warm wet weather, and infection can only occur when needles are wet. Asexual spores are spread by rain-splash, wind-driven rain, and contaminated equipment; only sexual spores are airborne. It generally takes 2-3 consecutive years of warm wet weather for severe disease epidemics to develop.

Importance: Moderate. Brown spot needle blight kills needles, slows growth, weakens seedlings, reduces seedling survival after planting, and prolongs the period of time seedlings spend in the grass stage. Brown spot is a disease of importance and concern in seedling nurseries (Fig. 5).

Management: Seedlings with resistance are available and should be planted when possible. Remove and destroy infected seedlings to reduce inoculum levels. Plant seedlings on a wide spacing and keep the surrounding area clear of vegetation to improve air circulation. Fungicide treatments (root-dip) for seedlings are available and can reduce brown spot incidence in new plantings. Avoid activities in the stand that may spread spores, especially during warm wet periods. Regular, low intensity, prescribed burns during the winter are the most effective way to control brown spot. Seedlings can survive the low intensity fires that destroy dead or infected needles harboring spores (Fig. 6 & 7). Fungicides are available for Christmas tree plantations.

Timeline: Infection occurs mainly during warm wet periods throughout spring, summer, and fall. Symptoms develop within a few weeks of infection.

Range: Longleaf pine in central and eastern North Carolina; a possible concern for Christmas tree growers in the western part of the state.

Fig. 1 Blighted needles

Fig. 2 Needle lesions

Fig. 3 Needle blight

Fig. 4 Fruiting bodies on needle

Fig. 5 Infected seedlings

Fig. 6 Pre-burn

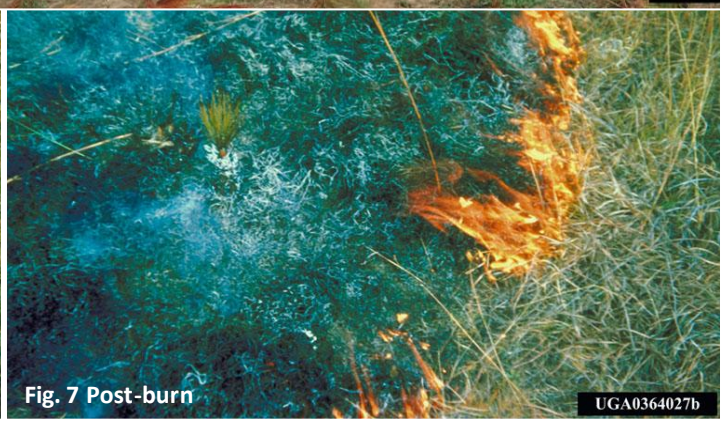

Fig. 7 Post-burn

Pine Needle Cast

Overview: Needle cast is a general term for a group of foliar diseases on pine which cause small spots or lesions on needles, needle browning, needle death, and premature needle drop. For the most part, these diseases are cosmetic and cause no serious damage to the tree. There are more than 30 species of fungi known to cause needle casts, but only a few affect southern pines. Disease cycles and control strategies are similar regardless of the causal organism; therefore specific identification of the fungal species is not usually necessary.

Causal Agent: Fungus (*Lophodermium* spp., *Ploioderma* spp., and others; the most common is *Ploioderma lethale* and *Lophodermium pinastra*).

Hosts: All southern pines are susceptible with the exception of longleaf pine (*see Brown Spot Needle Blight*). Needle cast fungi are often very host-specific.

Symptoms / Signs: Most needle cast fungi infect young, newly formed needles in the late spring or early summer; however, symptoms do not begin to develop until the following winter or early spring. The first symptoms of infection are small yellow spots on needles less than one year old. Yellow spots begin to turn brown and expand to form bands that surround the entire circumference of the needle (Fig. 3). In spring, the bands may turn pale yellow or grayish-brown. Tips of the needles and tissue between multiple bands will then turn brown and die; the base of the needle will often stay green.

Infected needles (especially needles with completely dead tips) will begin to prematurely drop in the late spring and throughout the summer the year after infection occurs. On severely diseased trees, all needles from the previous growing season may be lost, leaving only new growth (Fig. 1 & 2). Heavily defoliated branches and shoots may not grow very much, therefore new needles produced will be very close together around the shoot tip, giving it a tufted appearance.

Damage is most severe in dense plantings, on small trees, and on the lower branches of larger trees. Trees of all sizes can become heavily infected when weather conditions are suitable. Severe infections are often confused with bark beetle infestations. Needle cast is most commonly confused with the normal loss of old needles which typically occurs during the fall.

Disease Cycle: In the late spring (April – May) sticky spores are produced on lesions from the previous year's infections. The spores are spread by splashing rain or may be carried by strong winds for short distances. Infection can only occur during wet and cool weather. The fungus grows slowly, and the first symptoms of infection will not be visible until the following spring. The fungus eventually expands to surround the entire needle, at which time small black fruiting bodies form within the lesion and produce spores (Fig. 4). There is only one disease cycle per year.

Importance: Low. Needle cast diseases are generally cosmetic and cause no serious damage to the tree. In years of severe disease, trees can become unsightly or even appear to be dying, causing great concern to homeowners.

Management: Management for needle cast is usually not necessary as it causes no serious harm to the tree; however aesthetic concerns may warrant an attempt to reduce disease severity. Plant pines on a wide spacing and keep the area clear of vegetation to improve air circulation. Destruction of diseased needles that have fallen is ineffective because these needles have already released their spores before falling to the ground.

Timeline: Infection occurs in late spring or early summer. Symptoms begin to show the following year in late winter or early spring and through the summer. Normal shedding of older, non-diseased needles usually occurs in the fall.

Range: Statewide.

Fig. 1 Needle cast

Fig. 2 Needle cast

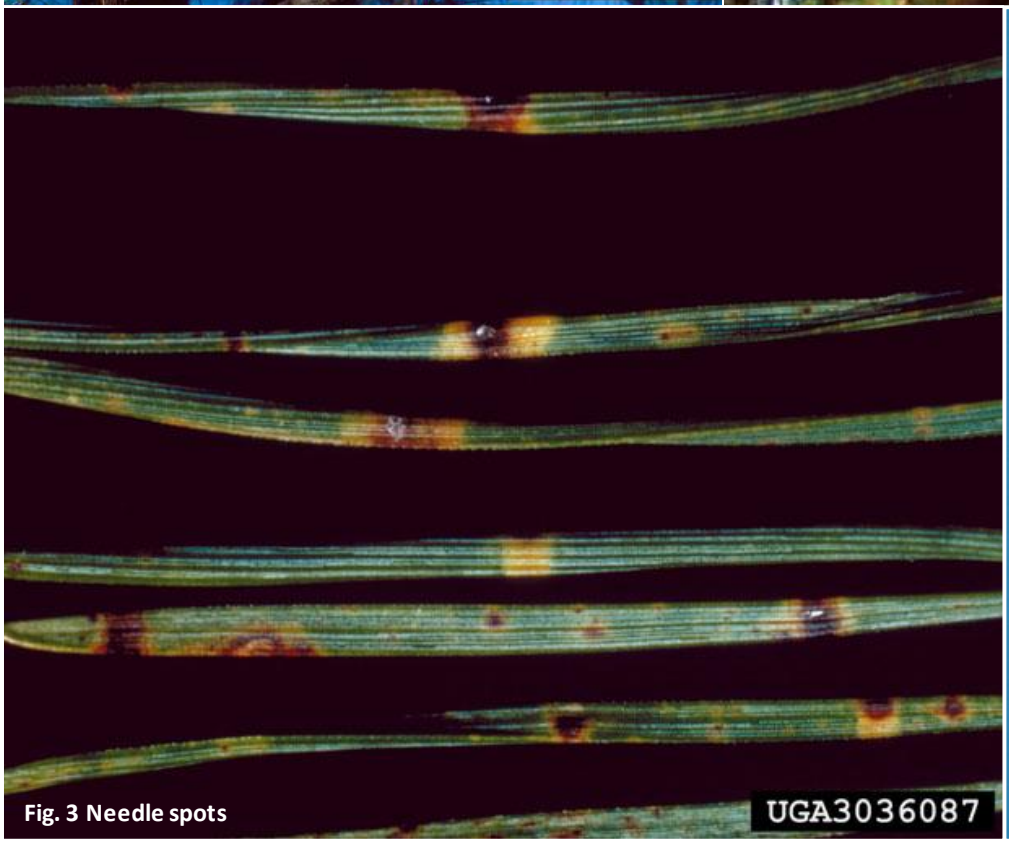

Fig. 3 Needle spots

Fig. 4 Fruiting bodies

Dogwood Anthracnose

Overview: Dogwood anthracnose is caused by a fungus that was introduced from Asia to the eastern United States in the late 1970's. Since its arrival in North Carolina, the disease has spread across much of the western part of the state and threatens to wipe out dogwoods at higher elevations. The disease is similar to many other anthracnose diseases because its airborne spores infect soft succulent tissue (especially leaves), but the dogwood anthracnose fungus can grow out of the leaves and cause girdling cankers (bark lesions) on branches and stems that eventually kill the tree.

Causal Agent: Fungus (*Discula destructiva*)

Hosts: Flowering dogwood and Pacific flowering dogwood. Several exotic dogwood species and cultivars (e.g. hybrids of Kousa x flowering dogwood) are now available that are resistant.

Symptoms / Signs: Shortly after infection in early spring, leaf lesions will appear on the upper leaf surface (Fig. 1). Spots will have a tan center with a purple or reddish margin; black spots (fruiting bodies) often appear on the underside of the leaf directly below leaf lesions. Lesions expand, often killing a large portion of the leaf (Fig. 2). Lesions that occur on leaf margins may expand and give the leaf a scorched appearance (Fig. 3). Leaves often droop and die, and can be retained on the tree through the winter months. Flower bracts can also become infected and may have lesions similar to those found on leaves (Fig. 4).

The fungus spreads out of the leaf causing cankers on stems and branches. Cankers are often centered on a small dead shoot or twig. Split bark and swelling may indicate the location of cankers (Fig. 5); removal of bark around these areas will reveal a tan to dark brown, often elliptical canker (Fig. 6).

Diseased trees will decline over several years; often lower branches will die first. Dogwoods will compensate for the loss of branches by producing lots of epicormic sprouts (suckers) on the stem or large branches, but these shoots are easily infected and are often the pathway for the fungus to enter and kill the main stem.

Dogwood anthracnose is often confused with dogwood spot anthracnose and drought; however, dogwood spot anthracnose does not cause cankers. Dogwood anthracnose is currently not known to be present in central and eastern North Carolina; in these regions dogwoods frequently suffer from drought-related decline or mortality and other common pests such as dogwood spot anthracnose and dogwood borer.

Disease Cycle: Asexual spores are produced from fallen leaves (infected the previous year) and stem cankers in early spring just as new leaves are beginning to expand. The spores are sticky and are easily spread by rain-splash, birds, and insects; spores are not easily spread by the wind. Infection occurs on soft succulent tissue such as leaves, flower bracts, and young shoots, but even thin bark can be directly penetrated under good conditions. Infection can only occur when leaves are wet for prolonged periods of time during cool weather. Additional spores are produced throughout the spring and summer when conditions are suitable. The fungus spreads from leaf tissue into adjacent twigs and branches where it parasitizes the inner bark and causes cankers. Cankers may eventually expand to girdle branches and stems. The pathogen overwinters in fallen leaves and cankers.

Importance: High. Dogwood anthracnose may potentially eliminate native dogwoods from the mountains of North Carolina.

Management: Plant trees on a wide spacing, keep area clear of competing vegetation, and prune trees regularly to improve air circulation. Dogwoods are an understory species, but planting them in partial sunlight will help keep leaves dry. Rake and destroy fallen or diseased leaves in the fall or winter, prune off branches with cankers, and remove epicormic sprouts to reduce disease incidence the following year. Remove severely diseased trees. Proper tree care is critical to keep trees healthy; water during dry periods (apply water to soil only), fertilize (avoid high nitrogen fertilizers), mulch around trees. Prune during hot and dry weather to avoid infection of wounds. Fungicides are available, but must be applied every 7 – 10 days during the spring and summer.

Timeline: Infection occurs in early spring and may continue through the summer during cool wet weather. Leaf symptoms begin to develop within a few weeks of infection; cankers can be seen year-round.

Range: Western North Carolina.

Fig. 1 Early leaf lesions

Fig. 2 Leaf lesions

Fig. 3 Leaf lesions

Fig. 4 Flower lesions

Fig. 5 Canker

Fig. 6 Canker with bark removed

Fusiform Rust

Overview: Fusiform rust is a very common and potentially serious disease, especially of slash and loblolly pines. The disease causes swellings (galls) on stems and/or branches which deform trees, reduce growth, and weaken wood making the trees more susceptible to breakage in high winds or bark beetle attack. Fusiform rust is also a problem in seedling nurseries; seedlings can be killed by the disease or the disease can be transported from the nursery to new plantings. The fusiform rust fungus requires an alternate host (oak) to complete its life cycle.

Causal Agent: Rust fungus (*Cronartium quercum* F. sp. *fusiforme*)

Hosts: Slash and loblolly pines are highly susceptible. Pitch and pond pine are also common hosts. Longleaf pine is moderately resistant and shortleaf pine is highly resistant. The alternate host of *C. quercum* include many species of oaks; most commonly water, willow, laurel, blackjack, southern red, and turkey oaks. The disease only causes small leaf spots (lesions) on oak foliage.

Symptoms / Signs: On pine, the most obvious symptom of infection is the formation of a spherical or spindle-shaped gall on a branch or main stem (Fig. 1). Galls are caused by chemicals released by the fungus that trigger abnormal and excessive wood growth. Pitch canker is commonly found in association with fusiform rust galls, so the gall may be pitch-soaked and/or exude sap (Fig. 6). Galls are often produced at the base of infected seedlings, though the swelling may be minor and often occurs at or below the soil line; these infections are frequently overlooked (Fig. 3). Fusiform rust will frequently kill the lower needles of seedlings in nursery beds.

During cool spring months, bright orange spores are often produced on the gall surface (Fig. 1 & 7). These aeciospores are blown off by the wind and serve to infect oak leaves. On oak, symptoms are limited to small leaf spots that may be chlorotic or necrotic. Often leaf spots are not noticed. Easier to observe are bright orange spores (urediospores) produced on the underside of the leaf (Fig. 2).

Disease Cycle: *C. quercum* has five different types of spores, and requires two or more years to fully complete its life cycle (Fig. 5). Bright orange aeciospores are produced on the surface of galls during the cool spring months (February-April). Aeciospores are blown on the wind and infect the leaves of susceptible oaks, where they cause a small leaf spot. After one week, bright orange urediospores are produced on the underside of the leaf, which serve to re-infect the same leaf or nearby leaves to build up inoculum levels. A week later, small brown teliospores are also produced on the underside of the leaf. Teliospores do not directly infect any plants, rather they remain attached to the leaf surface or may be blown off into leaf litter. These spores can survive for several months, until conditions are just right (60-80ºF, 95-100% RH), at which time they germinate and produce basidiospores. The basidiospores are also blown by the wind, back to nearby pines where they can infect needles, young shoots, and thin bark. The fungus grows a short distance into the branch or stem, where it produces plant growth-regulating compounds that cause the surrounding plant cells to rapidly divide and enlarge, forming a gall. If conditions are right during the first year of infection, pycniospores in orange droplets are produced on the gall surface (September - February) and allow sexual reproduction of the fungus.

Importance: High. Fusiform rust can be devastating in plantation and seedling nursery settings.

Management: Avoid planting susceptible species in areas with a historically high incidence of fusiform rust. Relatively resistant loblolly pine seedlings are available, but disease may still occur and/or be less severe. Close spacing in pine plantations will allow for some mortality while maintaining adequate stocking levels, and encourage infected branches to break off before the fungus reaches the main stem. Branches with galls within 8 inches of the main stem should be pruned off (Fig. 4). Avoid practices that over-stimulate growth such as fertilization, as this has been shown to increase the incidence of rust.

Timeline: Orange aeciospores are produced on the gall surface in early spring; followed by orange urediospores on oak leaves. Infection of pines by basidiospores occurs during the late spring / early summer months.

Range: Statewide, particularly central and eastern North Carolina.

Fig. 1 Gall with aeciospores

Fig. 2 Urediospores on oak

Fig. 3 Infected seedlings

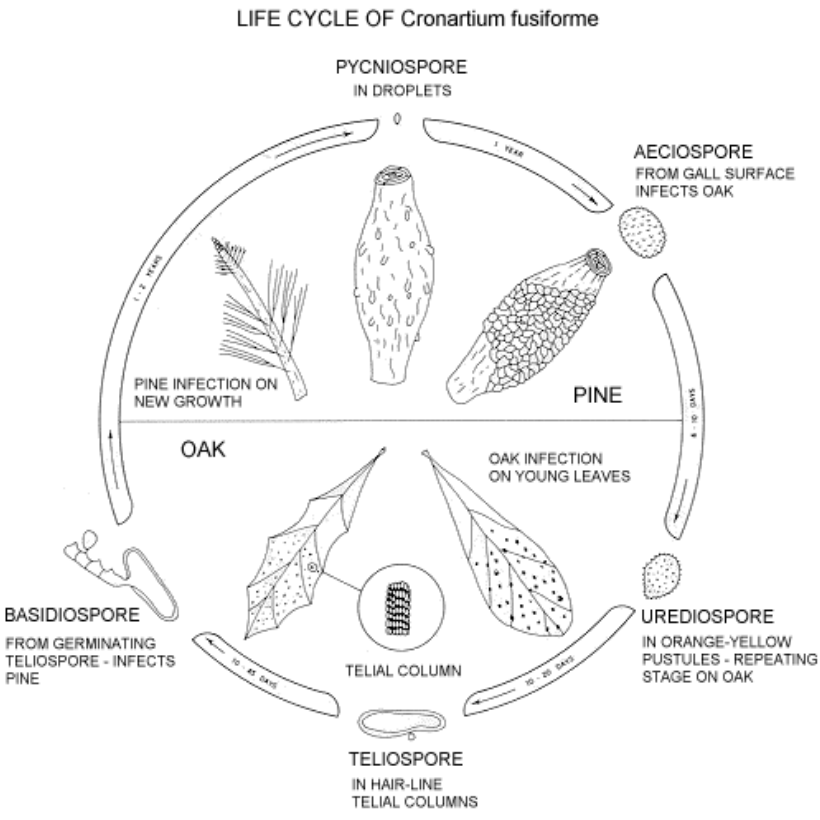

Fig. 5 Fusiform rust disease cycle

Fig. 4 Branch gall

Fig. 6 Stem Gall

Fig. 7 Stem Gall

Hypoxylon Canker

Overview: Hypoxylon canker is a secondary disease of many hardwood species that affects trees that are already severely stressed by some other cause. There are many *Hypoxylon* fungi, most of which are strictly wood-rotters, but some species can become pathogenic and kill stressed trees. The disease is most common in large mature trees, especially after prolonged periods of severe drought. Hypoxylon canker is a common contributor to the oak decline disease complex. (*Note: there is a separate, unrelated disease of aspen also called Hypoxylon Canker*).

Causal Agent: Fungus (*Hypoxylon* spp.; most commonly *H. atropunctatum* and *H. mediterranea*)

Hosts: Most common in oaks (especially red oaks, but white oaks are also susceptible). Also found in hickory, maple, beech, sycamore, birch, elm, walnut, and many others.

Symptoms / Signs: Symptoms may initially resemble those of oak decline: delayed bud break; undersized leaves; chlorotic, scorched, or wilted foliage; and branch dieback progressing from the top of the tree downward.

The easiest way to identify Hypoxylon canker is by the large spore-bearing mats (stromata) produced beneath the bark of diseased trees. These stromata usually appear the year following drought (or other severe stress), but may appear within a few months. Stromata will grow in size and eventually rupture the bark; patches of sloughed-off bark range from a few inches to several feet in long. In severe cases, almost the entire tree will lose its bark and be covered in the fungal mats. Stromata are initially tan, brown, or black (Fig. 1 & 2); after all of the spores are released the mats turn grey (Fig. 3 & 4). Trees die quickly, if not already dead, shortly after stromata production.

Disease Cycle: Spores of *Hypoxylon* fungi are airborne and are very common. Even when little or no disease is present in the forest, the fungus is present as a wood-rotter and feeds on dead wood. Trees are most often infected at a very early age through small wounds and natural openings. It is believed that most oaks and other susceptible species have *Hypoxylon* infections, but disease does not develop until trees become severely stressed. In healthy trees, the fungus forms small colonies beneath the bark and cause very little (if any) observable damage. The natural defenses of healthy trees easily keep the infection contained. However, when trees become water stressed, the *Hypoxylon* colonies begin to grow rapidly in the water-depleted sapwood and inner bark. The fungus attacks these tissues, forms stromata, and the bark is sloughed off. With little or no remaining functional vascular system, trees die quickly once the invasion begins. The fungus then invades the remaining wood; initially turning sapwood brown, it causes a yellowish-white decay with black lines.

Importance: Moderate. Following drought, Hypoxylon canker may be responsible for the death of many large mature trees. Valuable shade trees, if not properly cared for, are commonly lost due to Hypoxylon canker.

Management: Few management options are available. Proper tree care is critical. Provide trees with adequate water (at least 1-2 inches per week below the entire drip line), fertilizer if necessary (avoid nitrogen-rich fertilizers), and room to grow with little competition from neighboring trees or understory. Provide a thin layer of mulch (1-3 inches) below as much of the drip line as possible. Avoid planting susceptible species on dry sites.

Timeline: Symptoms usually develop in the year after a drought, but may develop within a few months if conditions are severe. Symptoms most often first appear during late spring and throughout the summer.

Range: Statewide.

Fig. 1 Stromata with spores

Fig. 2 Stromata with spores

Fig. 3 Stromata after spore release

Fig. 4 Stromata after spore release

Pitch Canker

Overview: Pitch canker is caused by a fungus that creates a resin-soaked lesion in the inner bark and outer sapwood of southern pine species. Most often a nuisance, the disease can deform trees, suppress growth, and kill branches or occasionally entire trees. Contrary to popular belief, the pitch canker fungus is not carried or transmitted by insects; rather the fungus infects trees through wounds including but not limited to insect feeding sites.

Causal Agent: Fungus (*Fusarium circinatum*)

Hosts: All southern pine species are susceptible. It is most common in loblolly, slash, and shortleaf pines.

Symptoms / Signs: The primary symptom of pitch canker is a resin-soaked canker (lesion) (Fig. 2). Small branches or twigs that are infected are often completely girdled and killed (Fig. 3); killing of the terminal or uppermost branches is also very common (Fig. 1 & 6). The fungus can also infect and cause perennial cankers on large branches, main stems (Fig. 4), and even exposed roots. Flowers, cones, and seeds can also be killed.

Diseased bark will turn tan to chocolate-brown, and the underlying sapwood becomes yellowish in color and heavily pitch-soaked. Resin-soaking may extend all of the way to the pith in severe cases. Bark remains on the canker and sap will frequently flow out of the lesion and dry on the branch surface or on other understory plants. Pitch canker commonly occurs in close-association with fusiform rust galls.

Needles around cankers usually turn yellow, then brown and are killed; they are retained on the tree by dried sap. Green shoots may wilt and die, giving the appearance of a shepherd's crook (Fig. 5).

Disease Cycle: Asexual spores are usually produced from an individual plant only once a year, but the timing varies widely based on climate, host species, and host age. Therefore spores are usually present year-round (particularly during cool and wet weather). The spores are produced on small pink fruiting bodies on and adjacent to cankers. The spores are easily carried by the wind and may travel for many miles, but must land on a fresh wound (less than a few days old) for infection to occur. Wounds are most often produced by hail, wind, falling cones, equipment, and insects such as twig and cone feeders. The fungus feeds on the inner bark and the tree responds by flooding the tissue with sap to prevent the fungus from spreading further.

Importance: Moderate. Usually pitch canker is of little concern. Trees are easily able to overcome minor infections of small branches and twigs. Occasionally large epidemics of pitch canker occur, resulting in deformed, stunted, highly stressed, or even dead pines. It can be a serious disease in seedling nurseries. There are quarantines and regulations in effect to prevent movement of this disease to other countries.

Management: Little can be done to prevent or treat pitch canker. Avoid wounding trees, especially during cool wet weather. Proper tree care is important to help trees overcome infection. Use of fertilizers rich in nitrogen is often accompanied by a large increase in pitch canker incidence. Excessive nitrogen is responsible for the high incidence of pitch canker near exhaust fans of chicken and hog houses.

Timeline: Symptoms most often appear between fall and spring. Infections occur year-round during cool wet weather.

Range: Statewide.

Fig. 1 Killed terminal shoots

Fig. 2 Resin-soaked canker

Fig. 3 Severe infection

Fig. 4 Stem canker

Fig. 5 Killed shoot

Fig. 6 Top kill

Wetwood / Slime Flux

Overview: Wetwood is not a serious disease of trees, but is very common and often of great concern to homeowners. Wetwood is a general term for a water-soaked condition of the wood caused by bacteria that occurs naturally in many tree species. Occasionally when pressure builds up in wetwood, wetwood fluids and gases may be released onto the surface of affected stems and limbs. This is known as slime flux. A separate condition known as alcoholic slime flux is also common in many trees, but is unrelated to wetwood. Alcoholic slime flux occurs when fermentation (due to a variety of microorganisms) occurs in sealed cracks and wounds; occasionally gas and ethanol can be released from these as well.

Causal Agent: Bacteria (mostly anaerobic)

Hosts: Found very commonly in elm. Also found in maple, oak, sweetgum, sycamore, willow, hemlock, and fir.

Symptoms / Signs: Wetwood is mostly an internal condition that cannot be detected from the surface. Wood is heavily water-soaked and usually discolored (color varies from pink, yellow, green, brown, red, and black). Pockets of wetwood are usually irregular in shape; the wood often has a sour smell. The condition usually starts in the main stem, but may spread to larger limbs. It is often associated with old wounds, but this is not always the case, and can be found in both heartwood and sapwood. Occasionally wetwood can cause leaf chlorosis, wilting, minor defoliation, and even dieback.

Wetwood is usually noticed when fluid pressure builds up and breaks through the outer sapwood and bark (wetwood slime flux). The fluid will have a sour smell, and leave grey, brown, or black streaks down the bark (Fig. 1 & 4). Alcoholic slime flux will always be associated with an old crack, wound, or cavity (Fig. 3). The flux is colorless (though may be frothy white under pressure) and has a fermented odor. It will also discolor bark. Both wetwood slime flux and alcoholic slime flux are highly attractive to insects such as bees, wasps, ants, butterflies, and moths (Fig. 2). Fluid darkens when exposed to air and may be produced so excessively as to flow or pool on the ground below. Various fungi and bacteria colonize the fluids once exposed to oxygen, and therefore the ooze may become slimy and foul smelling.

Disease Cycle: Wetwood develops due to a bacterial infection and is promoted by a tree's natural physiology. Bacteria enter trees (often when young) through wounds and natural openings and spread through the sapwood. It is unknown what causes the formation of wetwood; initially the accumulation of organic acids, ions, and alcohol draw large amounts of water into the wood, but whether these products are produced by the bacteria, the tree, or a combination of the two is not known. Wood becomes heavily water-soaked (the surrounding wood will be unusually dry) and anaerobic bacteria frequently colonize the area and feed on the cells of the sapwood. The by-products of bacterial activity cause the wood to discolor, have an elevated pH, and smell; pressure of these by-products may cause fluid and gas to escape through the surface.

Alcoholic slime flux occurs when microorganisms (fungi and bacteria) invade cracks, wounds, or cavities and begin to ferment sap and dead wood. Ethanol and gases are produced during the fermentation process, which causes pressure to build. Excess pressure is relieved when liquid and gas is expelled through the surface.

Importance: Low. Wetwood and slime flux do little if any harm to affected trees. Minimal living tissue is destroyed in wetwood; none in alcoholic slime flux. Wood is not rotted or structurally weakened in any way; in fact, wetwood has antibiotic properties and is highly resistant to rot/decay. The disease is mainly a nuisance to homeowners, but does reduce lumber value because it requires more time and energy to dry adequately.

Management: There are no preventative measures for wetwood / slime flux. Fluids can be washed from the bark surface with a mild soap solution. Do not attempt to cut into or drain pockets of wetwood or alcoholic slime flux; introduction of oxygen into these cavities will allow wood-rotting fungi to invade and severe decay can occur.

Timeline: Symptoms usually appear during the spring or summer, but can occur in the fall.

Range: Statewide.

Fig. 1 Bark staining

Fig. 2 Attracted insects

Fig. 3 Bark crack

Fig. 4 Slime flux

White Pine Blister Rust

Overview: White pine blister rust is the most important disease of eastern white pine (and other five-needled pines) in the United States. The white pine blister rust fungus was introduced from Asia in the early 1900's, and causes swollen cankers that eventually girdle entire branches or stems resulting in tree death. The disease is most severe in seedlings and saplings, but mature trees are also susceptible. The white pine blister rust fungus requires an alternate host (*Ribes* spp. in including gooseberry and currant) to complete its life cycle.

Causal Agent: Rust fungus (*Cronartium ribicola*)

Hosts: All five-needled pines (five needles per fascicle) are susceptible. In North Carolina, eastern white pine is the only native susceptible pine species. Alternate hosts are in the genus *Ribes* and include gooseberry and currant.

Symptoms / Signs: Small yellow or red needle spots are usually present on needles at the point of infection (Fig. 3) but are difficult to see. Roughly diamond-shaped cankers (bark lesions) develop a few years after infection. Cankers may be found on the branches or main stem, and are often centered on a small dead twig through which the fungus spreads after infection (Fig. 4). The first symptoms observed are usually individual flagging branches killed by cankers. Cankers that do not completely girdle the stem may not be swollen, rather these lesions tend to be elliptical or elongated with rough bark and a resinous center. Bark around lesions may be brownish-red (Fig. 8). Cankers that girdle the stem completely may become slightly swollen and are usually very resinous (Fig 2). Swelling may develop on the apical (branch tip) side of the lesion because the products of photosynthesis are blocked by girdling cankers and cannot be translocated from the branch to the stem or roots. In late spring or early summer, aeciospores may be visible in yellow-orange blisters on the canker surface (Fig. 6).

Girdling cankers will eventually kill the branch, or if on the stem, the entire tree. Infection in seedlings or small trees usually results in death because the fungus can easily travel the distance from needle to main stem. Infections in mature trees may only result in dead or dying tree tops or large branches; the bottom of the tree occasionally survives the infection (Fig. 7).

Disease Cycle: *C. ribicola* has five different types of spores, and requires three to six years to fully complete its life cycle (Fig. 5). Infection of pine by basidiospores occurs in late summer or early fall during extended periods of cool wet weather. Basidiospores can only travel a couple of miles (usually much less) from the alternate host. Within a few weeks of infection, a small yellow or red spot will appear on the needle surface at the point at which the fungus entered the plant. Unless the tree responds to the attack by shedding infected needles, the fungus will grow out of the needle and into the twig or branch within one year of infection. The fungus continues to spread through the inner bark and living cells of the sapwood at a rate of approximately three inches per year. Infected bark begins to swell due to rapid cell division and growth induced by chemicals released by the fungus. Spermagonia (which produce spores called spermatia) allow the fungus to sexually reproduce and are usually formed on the canker surface (late summer or early fall) with one or two years of infection. Yellow-orange aeciospores (Fig. 1) are also produce on the lesion (in spring) and can be blown many miles to infect the alternate host *Ribes*. Yellow-orange urediospores are produced on the underside of *Ribes* leaves within two weeks, and re-infect the same leaf or neighboring leaves to build up inoculum levels. In the late summer or early autumn, teliospores are produced on the underside of *Ribes* adjacent to urediospores infections. During wet weather, teliospores germinate to form basidiospores which re-infect pine.

Importance: High. This disease wiped out most of the mature white pines in the U.S. during the early – mid 20th century.

Management: Resistant white pine seedlings are becoming available. Eradication of *Ribes* is usually not effective and is no longer practiced. Avoid regenerating pines in small openings more prone to dew/condensation on needles. Prune lower branches (more prone to infection) and infected branches to prevent spread to main stem.

Timeline: Infection of pines occurs in late summer / early fall. Cankers can be seen year round, but produce yellow-orange aeciospores in the spring.

Range: Mountains of North Carolina; within the natural range of white pine where cool and wet conditions are prevalent in the fall.

Fig. 1 Urediospores on *Ribes*

Fig. 2 Dried sap

Fig. 3 Needle lesion

Fig. 4 Canker

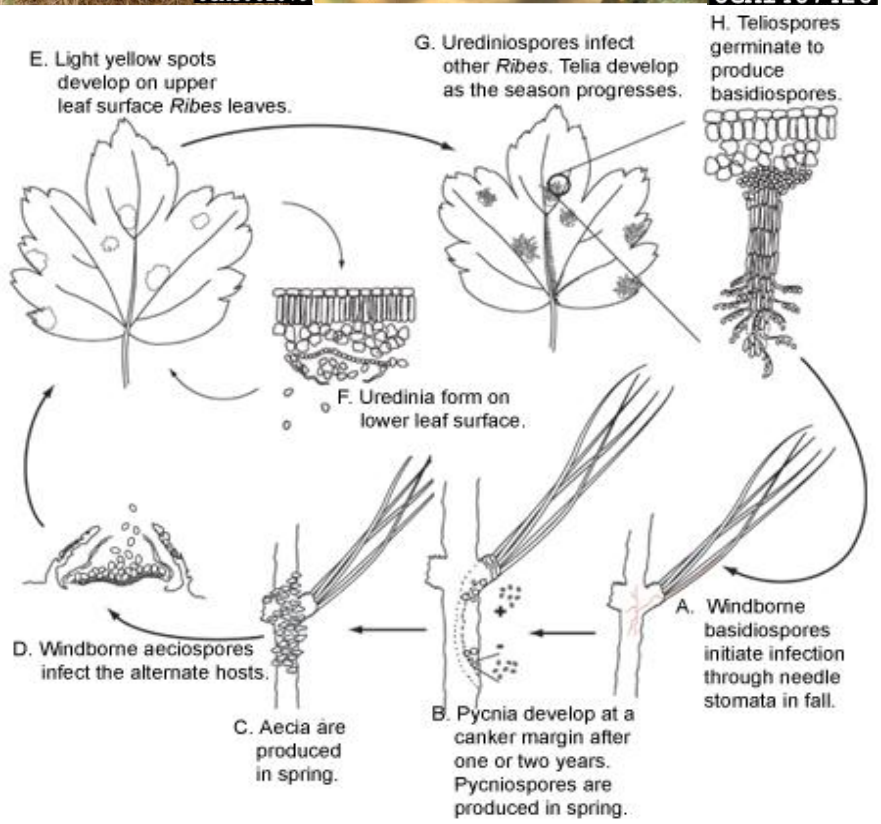

Fig. 5 White pine blister rust disease cycle

Fig. 6 Aeciospores

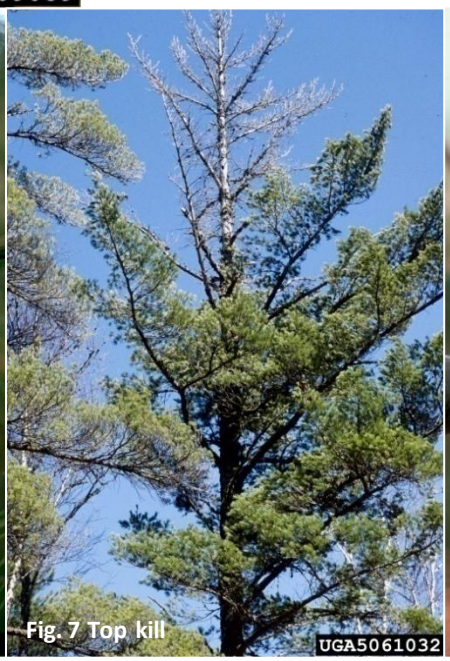

Fig. 7 Top kill

Fig. 8 Resinous canker

Beech Bark Disease

Overview: Beech bark disease was first reported in Canada in the 1920's; since that time it has spread south to some of the western-most counties of North Carolina. It is caused by a fungus and spread by a scale insect; both of which were introduced from Europe. The scale insect itself causes serious damage to the tree, and the fungus causes cankers which eventually coalesce to cause severe decline and death. The disease poses a serious threat to mature beech trees, but young trees and stump sprouts are not affected.

Causal Agent: Fungus (*Neonectria* spp. including the native species *Nectria galligena* and the exotic species *Nectria coccinea var. faginata*) and beech scale (*Cryptococcus fagisuga*)

Hosts: American and European beech.

Symptoms / Signs: Beech scale attacks and forms colonies on large branches and the stem (small braches and twigs are not attacked). The adult is less than 1/16 of an inch long, yellow, round, and slightly elongated, and soft-bodied. Woolly wax filaments are produced by sedentary adults and cover the colonies which begin as scattered white tufts in cracks and crevices. Colonies frequently start on the north side of trees; as the colonies grow they form broad white sheets on the trunk (Fig. 1 & 2). Minor bark necrosis is directly caused by scale infestations, and colonies disappear from killed bark.

After scales become established on the tree, the beech bark fungi begin to attack the tree through the wounds caused by the feeding insects. The fungus kills the inner bark tissue causing an expanding canker with an orange margin (Fig. 5); the edges of the canker will be rough-barked due to the production of callus tissue. Slimy red-brown fluid frequently leaks from cankers (Fig. 4). Dead bark falls off of lesions after a few years. Trees may decline and die over a period of several years; occasionally decay may become so severe in girdled trees that stem breakage occurs before the tree dies completely (Fig. 3). Clusters of small red fruiting bodies are produced on and around cankers in the fall (Fig. 7). Some trees possess various degrees of resistance; cankers on these trees may be limited in size (Fig. 6).

Disease Cycle: Scale insects reproduce once a year; male beech scales are not present in North America, therefore the scales reproduce parthenogenetically. Eggs are laid on the surface of branches and trunks; shortly thereafter the small mobile crawlers emerge. Crawlers may be blown on the wind for several miles; they are mobile for a few days before permanently attaching themselves to the bark and feeding on the nutrient-rich sap of the phloem. As they age, they produce a white waxy protective coating. The scales weaken the tree and kill localized sections of bark allowing infection by *Neonectria* species. Sexual spores of the fungus are produced in the fall through spring on clusters of small red fruiting bodies during wet weather; asexual spores are also produced in late summer and fall. Spores infect the tree through scale feeding sites and cracked dead bark. The pathogen kills the inner bark and outer sapwood; cankers expand most rapidly during cool fall weather. The native *Nectria galligena* usually attacks scaleinfested trees first, but is then displaced by the exotic *Nectria coccinea var. faginata*.

Importance: High. Beech bark disease is a serious threat to mature beeches. After the disease passes through an area, sprouts and seedlings begin to grow again and are not attacked by the scales. Some large trees, which may possess various levels of resistance, may also survive but are usually disfigured. The long term impact of the disease is not known.

Management: Insecticides are available to kill scales and prevent infection by the beech bark pathogens. Promote overall tree health by using proper tree care techniques. Please report suspected cases of beech bark disease to NCFS Forest Health staff.

Timeline: Red fruiting bodies are produced in the fall, and infections occur from fall through spring. Crawlers are active in mid-summer. Cankers expand most rapidly in the fall.

Range: Western North Carolina; potentially statewide.

Fig. 1 Adult scales

Fig. 2 Adult scales

Fig. 3 Diseased beech

Fig. 4 Bleeding canker

Fig. 5 Canker

Fig. 6 Deformed tree

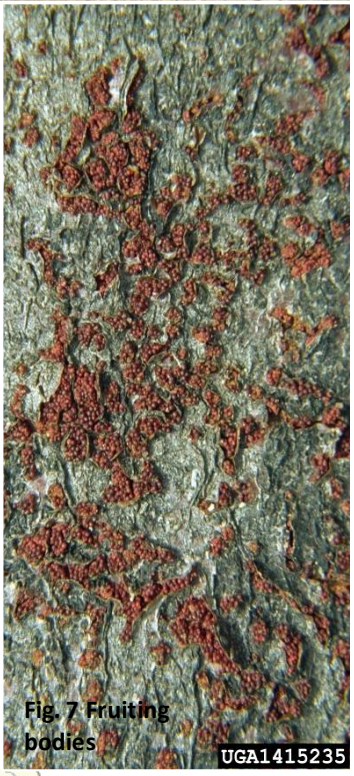

Fig. 7 Fruiting bodies

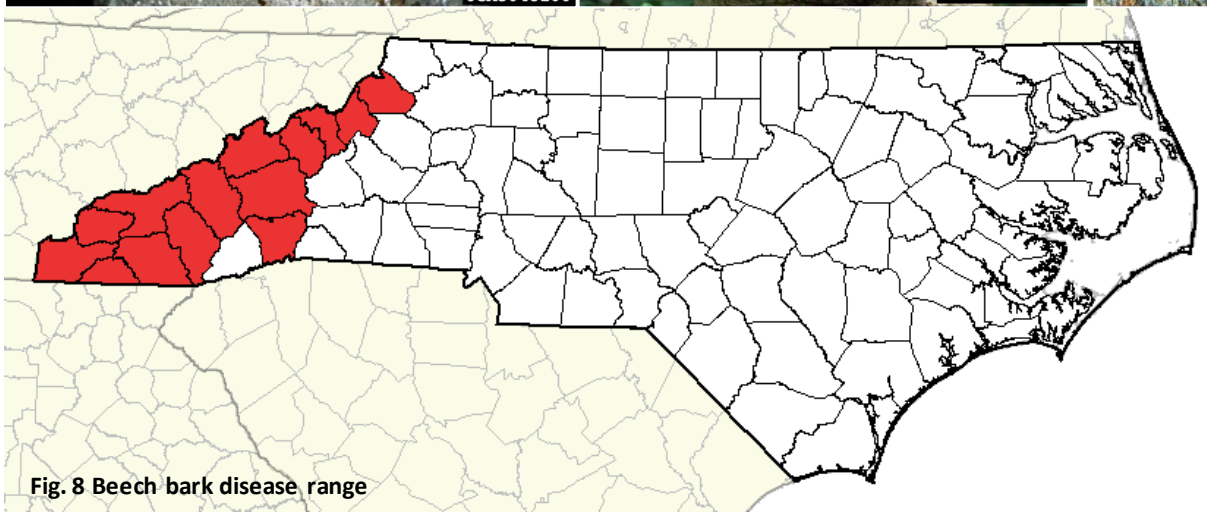
Fig. 8 Beech bark disease range

Sudden Oak Death

Overview: First discovered in 1995, sudden oak death has become a serious problem in northern California and Oregon. The pathogen is currently killing tanoaks, coast live oaks, and California black oaks in these western states (Fig. 4) and threatens the vast oak forests of the eastern United States. The name "sudden oak death" is somewhat of a misnomer, as trees often die over a period of several years. The disease causes severe girdling cankers on the main stem and large branches. The pathogen is known to infect hundreds of plant species, most of which show no obvious symptoms. Therefore, there is a serious risk that the pathogen will be introduced to new areas on infected landscape plants shipped from nurseries in the Pacific Northwest.

Causal Agent: Stramenopile; a fungus-like organism also known as a water mold (*Phytophthora ramorum*). Origin is unknown.

Hosts: Oak species are the most severely affected by the pathogen. In the east, red oaks are the most susceptible. The pathogen can infect and survive in hundreds of other woody and herbaceous plant species, many of which show few symptoms. Rhododendron is an important understory host that is frequently shipped from the west coast for ornamental purposes. The name "sudden oak death" is only used to refer to the disease caused in oaks.

Symptoms / Signs: On oaks and tanoak, cankers develop in the inner bark and outer sapwood (Fig. 1). Cankers rapidly expand to girdle infected trees, cutting off the tree's supply of water and nutrients. Black or reddish ooze is often observed from stem cankers. Bleeding cankers will stain the surface of the bark (Fig. 2); ooze may be difficult to see if it has dried or has been washed off by rain, but the dark staining will remain (Fig. 3). Black "fungal" lines may be visible in the sapwood beneath cankers, especially in dead trees. Leaves will turn from green to pale yellow to brown within a few weeks of girdling; succulent shoots may also wilt (Fig. 7). Crown dieback begins in the upper branches shortly thereafter (Fig. 6). Infected trees may survive for one to several years. Declining or dead trees may be attacked by secondary insects or pathogens such as ambrosia beetles, bark beetles, twolined chestnut borer, Hypoxylon canker, and Armillaria root rot.

Symptoms on other woody and herbaceous plant species vary, but infection occurs mostly on leaves and shoots. Small or large leaf lesions may be present (Fig. 5); premature leaf drop, shoot dieback, wilting, or death may occur.

Disease Cycle: Spores of *P. ramorum* are rarely produced on trees with cankers. Instead, it appears that most inoculum for the disease is produced from leaf infections on hosts other than oak. Therefore, although infections in foliar hosts do not often result in severe disease, inoculum from these plants is responsible for infecting and killing oak trees. Spores produced on foliar hosts are long-lived, can survive drying and cold temperatures, and are transported in rain splash, in flowing water, on the feet of animals and people, and on contaminated equipment, soil, and nursery plants. Spores that splash onto trees can infect the stem by directly penetrating the bark.

Importance: High. Sudden oak death is a serious threat to eastern oak forests. The implications of the disease being introduced are unknown. Several nurseries in North Carolina have tested positive for the disease on infected nursery plants shipped from areas where the disease occurs. The pathogen has not been detected on plants outside of these nurseries as of 2011.

Management: There are no known cures or preventative measures for sudden oak death. Large scale eradication of susceptible hosts in areas where the disease occurs has only been partially successful in slowing the spread. It is critical to prevent the introduction of the disease on infected nursery plants. Purchase plants from local sources when possible; do not purchase or plant diseased or unhealthy plants. Report any suspected cases of sudden oak death to the NCFS Forest Health staff immediately.

Timeline: Unknown. Foliar symptoms will only be observed during the growing season (with the exception of evergreens such as rhododendron). *P. ramorum* appears to prefer cooler temperatures than other *Phytophthora* species; infections may occur more frequently in spring or fall.

Range: Statewide risk. The disease of oaks has not been confirmed in North Carolina as of 2010, but the pathogen has been detected in several nurseries within the state on other plant species.


Fig. 1 Canker UGA5041047


Fig. 2 Bleeding canker UGA5297081


Fig. 3 Stained canker UGA5041037


Fig. 4 Sudden oak death epidemic in Oregon UGA5046053


Fig. 5 Leaf lesion UGA1427111

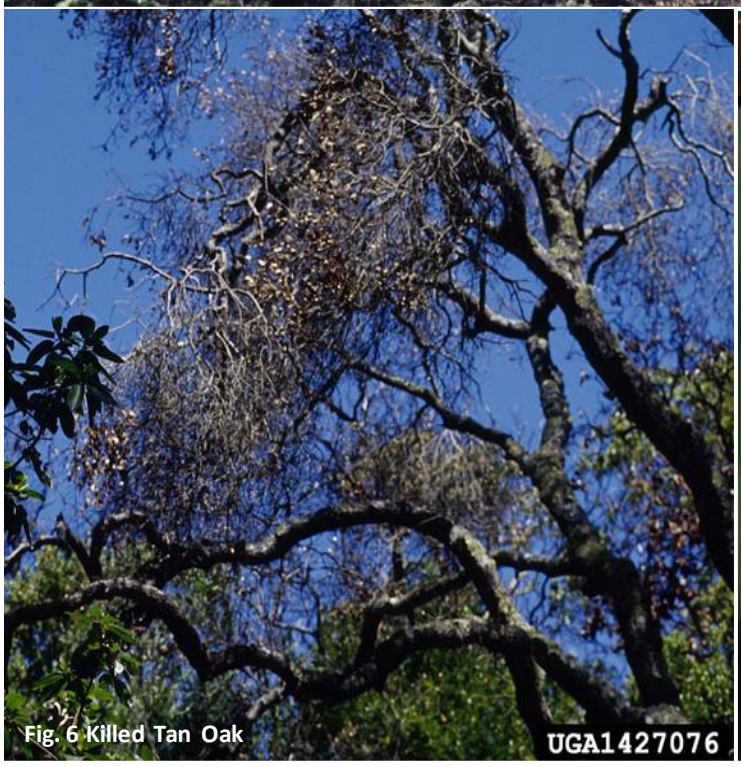
Fig. 6 Killed Tan Oak UGA1427076


Fig. 7 Wilted shoot UGA1427103

Thousand Cankers Disease

Overview: Thousand cankers disease is a newly recognized disease of black walnut caused by the fungus *Geosmithia morbida*. The disease is so named because it is characterized by large numbers of small cankers on the branches and stem that eventually coalesce. This results in widespread necrosis of inner bark tissue, dieback, and eventually death. *G. morbida* is vectored by the walnut twig beetle (*Pityophthorus juglandis*). Both the beetle and fungus are thought to be native to the southwestern U.S. and Mexico. In 2010, the disease was discovered in the eastern U.S.: first in Knoxville, TN and then subsequently in several additional states. The disease could potentially spread through the entire native range of black walnut. Suspected infestations of the thousand cankers disease should be reported immediately to NCFS Forest Health staff.

Causal Agent: Fungus (*Geosmithia morbida*) vectored by the walnut twig beetle (*Pityophthorus juglandis*)

Hosts: Black walnut and butternut; also other *Juglans* spp. native to the western U.S.

Symptoms / Signs: Symptoms usually appear during the summer months (June-August). Early symptoms include thinning crowns, undersized or stunted foliage, branch flagging (Fig. 8), yellowing or wilting leaves (Fig. 7), and brown wilted leaves that remain attached to branches (Fig. 9). Symptoms in individual branches may develop over a period of several weeks; often rapid wilting of leaves soon follows branch flagging. Epicormic sprouting is common. Eventually, individual branches are killed; dieback tends to develop in the upper crown and spreads downward (Fig. 10). Cankers can only be observed if the outer bark is carefully stripped away from infested branches; cankers are not visible on the bark surface. Cankers are diffuse, chocolate brown, usually less than one inch in diameter, and can only be found in the inner bark (periderm), phloem, and cambium tissues adjacent to beetle galleries (Fig 1 & 3). Cankers do not affect the sapwood. Eventually, cankers coalesce and individual cankers can no longer be distinguished (Fig. 2). Trees usually die within 3 years of initial symptom development, but it may take more than 10-15 years after the tree is first attacked for obvious symptoms to develop.

Symptomatic branches will be riddled with many pinhole sized exit holes often spaced closely together (Fig. 6). Walnut twig beetles, which are dark brown and less than 1/16 inch long (Fig. 4&5), initially attack branches greater than 1½ inches in diameter in the upper crown. The stem and larger branches in the lower crown are attacked later as the tree declines. Beetles tend to favor the undersides of branches on the south or west-facing side of the tree. Beetle galleries in the inner bark are winding and usually less than a few inches long (Fig. 3). There may be thousands of beetles in every linear foot of infested branches in the later stages of the disease.

Disease Cycle: The pathogen produces small circular or oblong cankers in the branches and stem. Cankers are often initially restricted to the inner layers of the cork cambium. Over time, the cankers may expand into the phloem and cambium; they become more diffuse and cause affected tissues to soften and turn dark brown or black. Eventually, the cankers coalesce and girdle the branches and stem. The pathogen is thought to require the walnut twig beetle for transmission and infection; it is not known if other methods of spread are possible or if other insects could serve as vectors. Relatively high populations of the walnut twig beetle are required before thousand cankers disease can cause severe decline or mortality. Beetles are likely initially attracted to stressed trees; both males and females emit aggregation pheromones that attract additional beetles when a suitable host is found. Individual beetles can travel up to two miles in search of a host tree. It is not known how many generations of the walnut twig beetle will occur annually in the eastern U.S.

Importance: High. While little is known about how this disease will affect black walnut in the eastern U.S., it has the potential to devastate the host population in its native range. Regulations to limit the spread of this disease are in effect and will significantly limit the sale and transport of walnut material out of quarantined zones.

Management: There are no known cures or preventative measures for this disease. Limit movement of infested wood; without human-assisted movement disease spread will be significantly limited. Report any suspected cases of thousand cankers disease to the NCFS Forest Health staff immediately.

Timeline: Unknown. Symptoms are most visible during the summer months.

Range: Statewide risk, especially western North Carolina. This disease was confirmed in the state in late 2012.


Fig. 1 Cankers around beetle galleries
UGA5024084


Fig. 2 Coalescing cankers
5406089


Fig. 3 Canker forming around beetle gallery
5406094

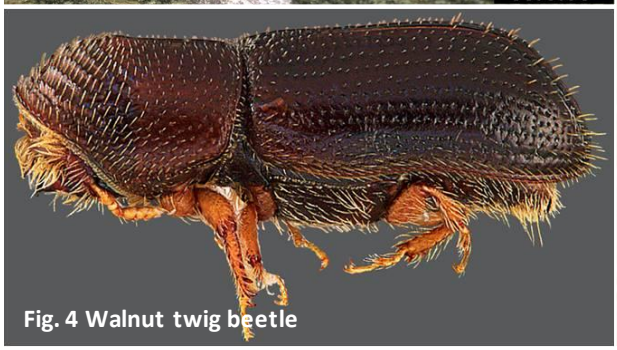
Fig. 4 Walnut twig beetle


Fig. 5 Walnut twig beetles


Fig. 6 Exit holes and exposed galleries
5406084


Fig. 7 Wilting leaves


Fig. 8 Thinning in crown and flagging
5406047


Fig. 9 Persistent wilted leaves
UGA5024087


Fig. 10 Decline
5406045

Fire Blight

Overview: Fire blight is a very important disease of many fruit-bearing trees in the Rose family. It is caused by bacteria that infect leaves, flowers, and fruits; but may also cause cankers, dieback, and even tree death in severe cases. The disease is easily spread by rain-splash, a number of insect vectors, and even on infected pruning tools. Fire blight is usually most severe in years with wet warm springs, especially during the blooming period.

Causal Agent: Bacteria (*Erwinia amylovora*)

Hosts: There are more than 100 species of plants known to be susceptible to fire blight; all are in the Rose family. Trees susceptible include apples, hawthorns, pears, and mountain-ash. The disease can also occur (but is far less common) in the stone fruits: peach, cherry, and plum.

Symptoms / Signs: Blighting of flowers is usually the first symptom to appear in spring. Flowers will darken, droop, shrivel, and turn black. Lesions on fruits, leaves, and green shoots will follow shortly thereafter (Fig. 1 & 2). The tips of infected shoots (and associated leaves) will also droop and turn black (Fig. 5). Wilted shoots will often resemble a shepherd's crook (Fig. 3 & 4). If many shoots are infected, trees will appear to be burned or scorched, hence the name "fire blight" (Fig. 6 & 7).

Cankers may form when branches and stems are infected by the bacteria. Bark on cankers may appear raised and slightly blistered, especially when cankers are actively expanding during the growing season. During the winter months, cankers may appear sunken and bark may be cracked. Cankers can eventually girdle and kill branches or the entire tree. Orange drops of slimy liquid filled with bacteria may be visible oozing on or near infected plant parts during warm and humid weather.

Disease Cycle: Bacteria overwinter in diseased plant parts including twigs and buds with cankers. When warm humid weather returns, small drops of orange fluid ooze from infected plant parts and can be transmitted to new growth via rain-splash, wind-driven rain, irrigation water, insects, birds, and pruning tools. There are many insects known to vector the fire blight bacteria, but the most commonly implicated are cicadas, leaf hoppers, and pollinators such as bees and flies. The bacteria can enter susceptible tissues through small wounds or insect feeding sites, or they may multiply on the plant surface and eventually penetrate through stomata and lenticels. Bacteria populations can double once every hour and spread through plant tissues at rates of up to 10 inches a day. As bacteria spread, they release enzymes that kill and dissolve plant cells; symptoms begin to appear a few weeks after infection.

Importance: Moderate. Fire blight can be of great concern to homeowners and fruit tree orchards. Many fruit trees and ornamentals are highly susceptible and symptoms can be severe in years with warm wet springs. Bradford pears (a popular ornamental) were originally thought to be resistant, but the disease has become prevalent in this pear cultivar.

Management: Prune off infected plant tissues as soon as they are observed; be sure to prune 8 inches or more away from the nearest symptomatic tissue. Prune trees regularly to increase air circulation in the crown. Make sure to sanitize pruning equipment after use to prevent spreading the disease. Most new infections start on flowers; removal of flowers on small trees can prevent infection. Bactericides are available, but proper timing of applications is critical and is difficult to accomplish effectively. Examine trees thoroughly 1-3 weeks after warm wet periods in the spring for any symptomatic tissue and remove.

Timeline: Most infections occur during wet/humid warm weather in the spring. Strong winds and rains during this period dramatically increase disease incidence.

Range: Statewide.

Fig. 1 Leaf lesion

Fig. 2 Fruit lesions

Fig. 3 Blighted shoot

Fig. 4 Blighted shoot

Fig. 5 Blighted shoot

Fig. 6 Severe blight

Fig. 7 Blighted shoots

Bacterial Leaf Scorch

Overview: Bacterial leaf scorch is caused by a bacterial infection of a tree's water conducting tissue. The bacteria are vectored by a number of insects. This disease can be very difficult to diagnose; symptoms of bacterial leaf scorch closely resemble those of other vascular diseases, declines, and abiotic stresses such as drought. Symptomatic tissue samples from trees suspected to have bacterial leaf scorch should be submitted to NCFS Forest Health staff or a diagnostic lab for confirmation.

Causal Agent: Bacteria (*Xylella fastidiosa*)

Hosts: There are hundreds of known hosts of bacterial leaf scorch. Trees include maple, buckeye, hackberry, dogwood, sweetgum, sycamore, plum, oak, and elm. There are many plants in which the bacteria grow and multiply without symptom development. These plants may serve as inoculum reservoirs.

Symptoms / Signs: Infection is perennial; bacteria are able to survive from year to year in the vascular system. Bacteria interfere with water transport in the xylem, therefore symptoms closely resemble those of drought and other vascular diseases. Leaf margins turn red or yellow; then leaves will wilt and turn brown especially during the summer months (Fig. 1 & 2). A red or yellow band often separates brown from green tissue. Older leaves are usually scorched first, with symptoms progressing towards shoot tips. Scorched leaves are retained on the tree into the fall.

Trees may have decreased fruit production, delayed bud break, reduced growth, stunting, branch dieback, and eventually death. Leaves usually expand normally each year; then symptoms begin to appear in late spring and progress throughout the summer. Hot droughty weather makes symptoms worse. Symptoms may initially appear in isolated branches or sections of the crown, but eventually spread throughout the tree (Fig 3, 4, & 5). Symptoms can fluctuate in severity from year to year.

Disease Cycle: Bacteria must be introduced into a tree's vascular system by insects in order for infection to occur. Common insect vectors include spittlebugs, sharpshooters, and leafhoppers; insects pick up the bacteria from infected trees and transmit it during feeding to healthy trees. Bacteria multiply rapidly in the xylem and are carried upward rapidly in the transpiration stream; downward spread occurs more slowly. Symptoms develop within a few weeks to a year depending on tree health and species. The bacteria overwinter in roots and stems and each year they spread to new xylem vessels. The bacteria produce enzymes that dissolve cell wall components and plug the vascular system.

Importance: Moderate. The disease can be severe, but is not commonly observed in North Carolina. The disease can stress trees and predispose them to attack by other insects and pathogens.

Management: There are currently no effective treatments for bacterial leaf scorch.

Timeline: Infections occur during the spring and summer. Symptoms begin to develop within a few weeks of infection. In subsequent years, symptoms begin to develop in late spring and are worst during the summer.

Range: Statewide.

Fig. 1 Scorched leaf

Fig. 2 Scorched leaves

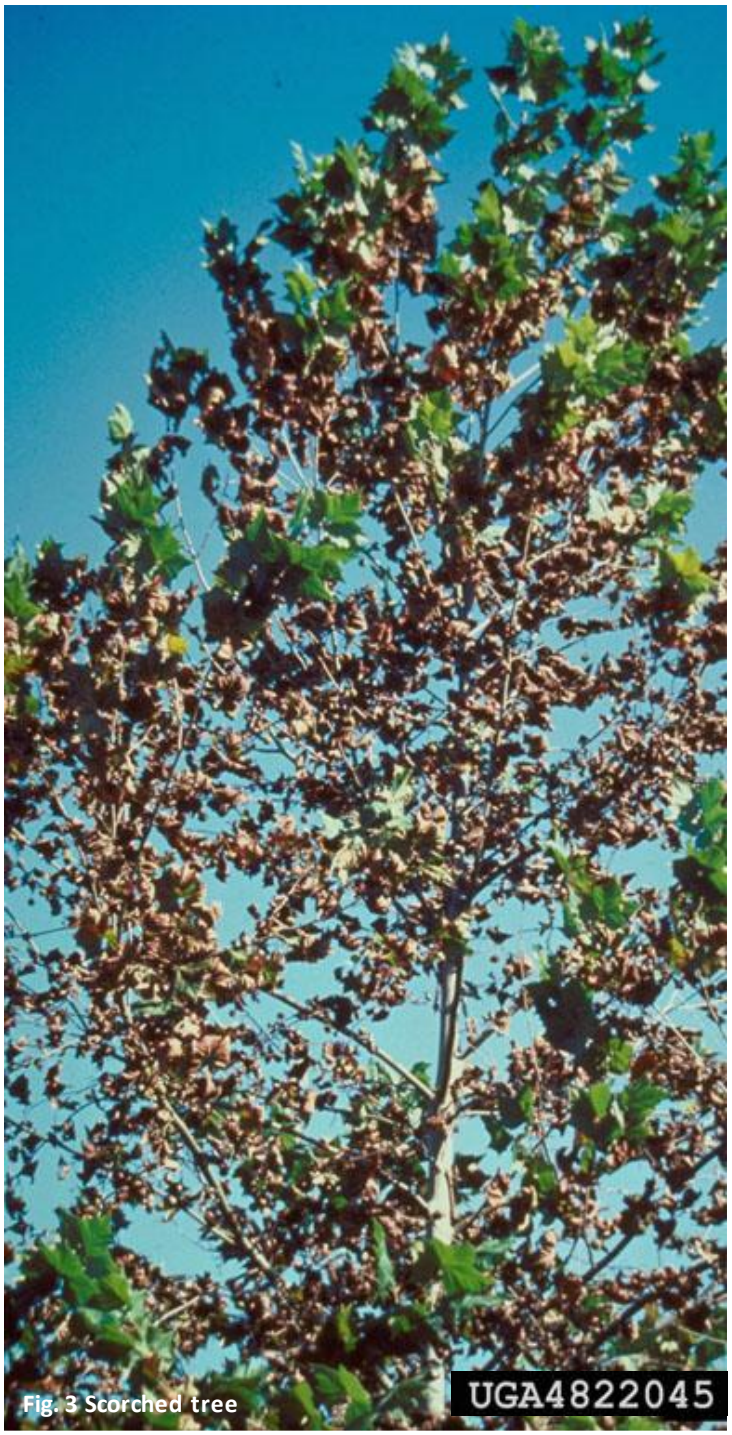

Fig. 3 Scorched tree

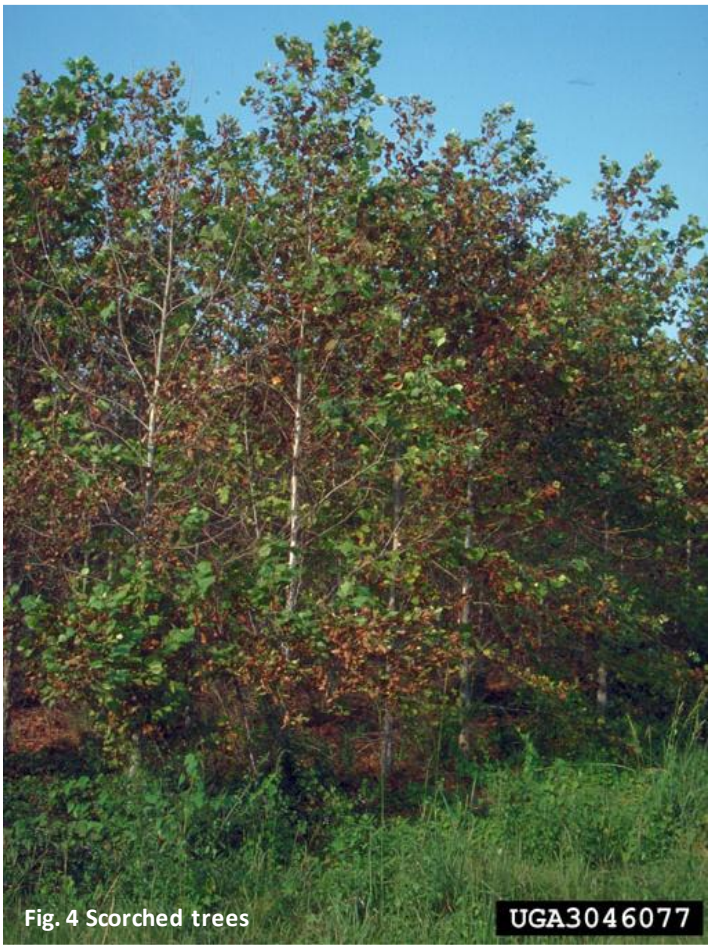

Fig. 4 Scorched trees

Fig. 5 Severely scorched tree

Oak Decline

Overview: Oak decline is known as a disease complex: a disease that has many contributing stress agents with no single agent playing a dominant role. Oak decline has been a major concern since the 1950's when it was first noticed that mature and otherwise healthy oaks would begin to decline and slowly die over the course of several years. The disease complex affects oaks in both forested and urban settings. Drought, defoliation, and secondary stress agents are common contributors.

Causal Agent: A combination of biotic and abiotic stress agents; most commonly drought, twolined chestnut borer, Armillaria root rot, late frost, soil compaction, root damage, and mechanical injury.

Hosts: Oak species. More common in red oaks.

Symptoms / Signs: Early symptoms include late bud-break, small and/or chlorotic leaves, and scattered dead twigs. Leaf scorch, premature leaf-drop, early fall coloration, epicormic sprouts (suckers), and reduced diameter and shoot growth may also be observed. The most obvious symptoms occur during the later stages of the disease. Large branches or even entire sections of the crown will begin to die. Dieback most often proceeds from the top of the crown downward. This creates a "stag-head" effect: large antler-like branches (with no leaves) sticking up out of the remaining green crown below (Fig. 1 & 5).

Oaks affected by decline are often host to a number of secondary insects and diseases, especially *Armillaria* root rot (Fig. 2) and the twolined chestnut borer (Fig. 3 & 4). Signs of many wood borers, secondary fungi, and cankers are often visible.

Disease Cycle: Decline is usually initiated by a prolonged or reoccurring stress. For instance, while trees can survive the activity of defoliating insects periodically, repeated and severe defoliation by late frost, gypsy moth, or leaf rollers can reduce tree health. Prolonged drought, root compaction, root damage, and mechanical injuries also weaken the tree. These predisposing factors weaken normal defense responses, making trees susceptible to attack by secondary insects and pathogens such as Armillaria root rot, Hypoxylon canker, and the twolined chestnut borer. Attack by these stress agents further decreases the tree's ability to get water and nutrients, and the tree is weakened further. Eventually the oak will succumb to this cycle of accumulating stresses

Importance: Moderate. Following drought, a significant percentage of mature oaks may begin to decline and die in susceptible forest stands. Trees are particularly vulnerable in urban settings where abiotic stresses are prevalent.

Management: Proper tree care is critical. Provide trees adequate water (at least 1-2 inches per week below the entire drip line), fertilizer if necessary (avoid nitrogen rich fertilizers), and room to grow with little competition from neighboring trees or understory. Provide a thin layer of mulch (1-3 inches) below as much of the drip line as possible. Avoid planting susceptible species on dry sites. Planting the right species on the right site is the best way to lower the risk of oak decline.

Timeline: Symptoms are most visible during the summer months when trees are water-stressed. Symptoms commonly appear in the years following a severe drought.

Range: Statewide.

Fig. 1 Crown dieback

Fig. 2 *Armillaria* fruiting bodies

Fig. 3 Twolined chestnut borer

Fig. 4 Twolined chestnut borer larva

Fig. 5 Advanced decline

Oak Wilt

Overview: Oak wilt is considered to be the most important disease of oaks in the eastern United States. Few other diseases encountered in forestry can kill large mature trees as quickly as oak wilt. The disease is caused by a fungus that spreads through a tree's vascular system. In response, the tree plugs up its vascular system to stop the fungus from spreading, but this also inhibits water movement, so the tree wilts and dies.

Causal Agent: Fungus (*Ceratocystis fagacearum*)

Hosts: All species of oak are susceptible to the disease; however, some species are more resistant than others. Oaks in the white oak group can sometimes live for many years with the disease or even recover completely. In contrast, members of the red oak and live oak groups are highly susceptible and usually die within several weeks to several months after infection occurs.

Symptoms / Signs: Early symptoms are often confused with drought. Leaves in the upper crown usually show symptoms first. Leaves will begin to turn greenish-grey or olive-green, and will have a wilted, limp, or water-soaked appearance (Fig. 1). The leaves will begin to brown at the leaf tips and edges, and then progress inward (Fig. 2). If symptoms begin in a single branch or isolated part of the crown in red oaks, they will rapidly spread through the rest of the tree within a few months (Fig. 3). Symptoms may be halted or only spread slowly in white oaks. Red oaks will often shed their leaves rapidly once infection starts; leaves on the ground may only be partially wilted. In cases where the tree wilts very rapidly, leaves may be retained. White oaks tend to retain wilted leaves for several months. The fungus often spreads through root grafts to neighboring trees, creating a "disease center" with recently infected and wilting trees surrounding dead trees (Fig. 6).

The outermost rings of sapwood will almost always be discolored in the wilting branches of white oaks; discoloration may or may not be present in red oaks. Vascular discoloration can be observed by looking at the branch in cross-section, or stripping away the bark to reveal the outer sapwood. Discoloration will look like dark (grayish, purplish, or black) streaking in the outermost ring of sapwood (Fig. 4).

Under the right environmental conditions, the oak wilt fungus will produce a black or grayish fungal mat that produces spores (Fig. 7). The mat is produced beneath the bark; enlargement of the mat causes the bark to rupture and allows insects to enter (Fig. 8). Mats are difficult to observe, usually produced in the spring, and have a fruity aroma like bananas or bubble gum. Mats can be just a few inches wide or almost a foot across.

Disease Cycle: *C. fagacearum* spreads to trees in two different ways: it can be carried by certain oak bark beetles or sap beetles resulting in new disease centers, or it can spread to neighboring trees through root grafts (Fig. 5) resulting in the expansion of disease centers. A beetle carrying the fungus must land on a fresh tree wound in order for infection to occur. Root graft transmission may take one or more years to occur. Oaks respond to infection by plugging up water-conducting vessels of the xylem to prevent further spread of the fungus. However this inhibits water movement in the tree and causes death. Spore mats produced beneath the bark attract sap beetles which can carry spores several miles.

Importance: High. Quarantines are in effect for counties where oak wilt is present.

Management: Over-land transmission by insect vectors is managed by removal of diseased trees to eliminate production of spore mats. Firewood from diseased trees should be tightly covered in plastic for at least one year or immediately burned. Avoid pruning oaks during months when spore-vectoring beetles are most active (March-July). Any pruning wounds or injuries during this time should be immediately treated with pruning paint. Root graft transmission is managed by severing root grafts between trees with a vibratory plow or trencher. Systemic fungicides are available for high-value trees and can provide protection for one to two years.

Timeline: Insect vectors are most active March-July. Spore mats are produced March-May. Most trees begin to wilt between June and September. Trees that are not completely wilted before winter may leaf out in the spring, but quickly wilt thereafter.

Range: Currently only known to occur in Swain, Jackson, Haywood, Buncombe, and Madison counties.

Fig. 1 Early wilt symptoms

Fig. 2 Wilted leaves

Fig. 3 Wilting oak

Fig. 4 Vascular streaking

Fig. 5 Root grafts

Fig. 6 Disease Center

Fig. 7 Spore mat

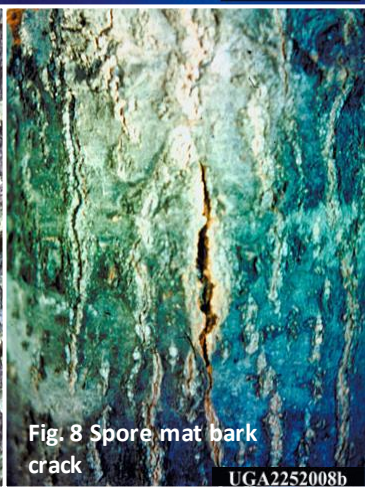

Fig. 8 Spore mat bark crack

Dutch Elm Disease

Overview: Dutch elm disease is a vascular wilt of elms caused by a fungus that is transmitted by bark beetles and root grafts. The fungus originated in Asia; the name "Dutch" refers to the identification of this disease and causal agent in the Netherlands by a Dutch scientist. The story of Dutch elm disease is long and interesting: it spans more than a century and involves complicated international politics, multiple continents, symbiotic relationships between several insects and pathogens, inter-species hybridization of fungi, and in the end, the catastrophic loss of an American icon. The disease has killed millions of elms in both Europe and the U.S. since the early 1900's. Two waves of the disease, first in the 1930's and then again in the 1970's, all but eliminated the American elm from the streets of U.S. cities (Fig. 2). Elms are beginning to make a comeback however, due to integrated management programs, systemic fungicides, and disease resistant cultivars.

Causal Agent: Fungus (*Ophiostoma ulmi* and *Ophiostoma novo-ulmi*)

Hosts: Elms. American and European elm species are highly susceptible; many Asian elm species have partial or complete resistance.

Symptoms / Signs: Early symptoms are often confused with drought. Usually one branch or an isolated area of the crown will "flag": leaves will wilt and turn yellow; then reddish-brown and die (Fig. 3). As the pathogen spreads out of the infected branch, larger areas of the crown begin to rapidly wilt resulting in crown dieback (Fig. 4). Complete wilting can occur within a few months of infection, but it may take a year or more (Fig. 1). Removal of bark on symptomatic branches or the main stem will reveal dark brownish-purple streaking in the outer sapwood (Fig. 5 & 6). Pathogen spread through root grafts may result in wilt development in nearby trees in subsequent years; trees infected through root grafts usually wilt more quickly than bark beetle-related infections.

The fungus produces microscopic fruiting bodies that are not visible in the field. Diagnosis based on vascular streaking/discoloration is usually reliable, but the diagnosis can be confirmed in a laboratory.

Disease Cycle: The fungus spreads to trees in two different ways: it can be carried by elm bark beetles for up to several miles, and it can spread to neighboring trees through root grafts. Adult beetles carry the fungus on their bodies or in their mouthparts, and transmit the pathogen into the tree's vascular system when feeding on tender bark, usually in branch crotches. The fungus kills the branch, then grows downward and spreads through the rest of the crown, and eventually, the root system. Root graft-transmission may take one or more years to occur because the pathogen must grow out of the crown and into the root system. However, when the pathogen spreads to a neighboring tree through roots, it is rapidly carried upward throughout the entire tree. Elms respond to infection by plugging up water-conducting vessels of the xylem to prevent further spread of the fungus, however, this inhibits water movement in the tree and causes death. Beetles use dead elms for breeding habitat.

Importance: High. This disease can be devastating for high-value landscape trees. Large specimens of American and European elms have become increasingly rare.

Management: Transmission by insect vectors is managed by rapid removal of diseased trees to eliminate breeding habitat for elm bark beetles. Firewood from diseased trees should be tightly covered in plastic for at least one year or burned immediately. Do not transport elm firewood. Root graft transmission is managed by severing root grafts between trees with a vibratory plow or trencher. Systemic fungicides are available that can protect high-value trees for one to three years; if the disease is caught early enough and has not spread into the root system, these fungicides can be used to successfully eradicate the fungus and save a diseased tree. Likewise, experienced arborists or tree surgeons can prune out diseased branches, or even infected sections of the stem, to save a tree in the early stages of disease. Disease resistant cultivars of American elm are now available.

Timeline: Insect vectors are most active throughout the growing season (*see elm bark beetles*). Wilt symptoms generally begin to develop during the summer months and early fall.

Range: Statewide.

Fig. 1 Wilted American elm

Fig. 2 Street lined with American elms

Fig. 3 Branch flagging

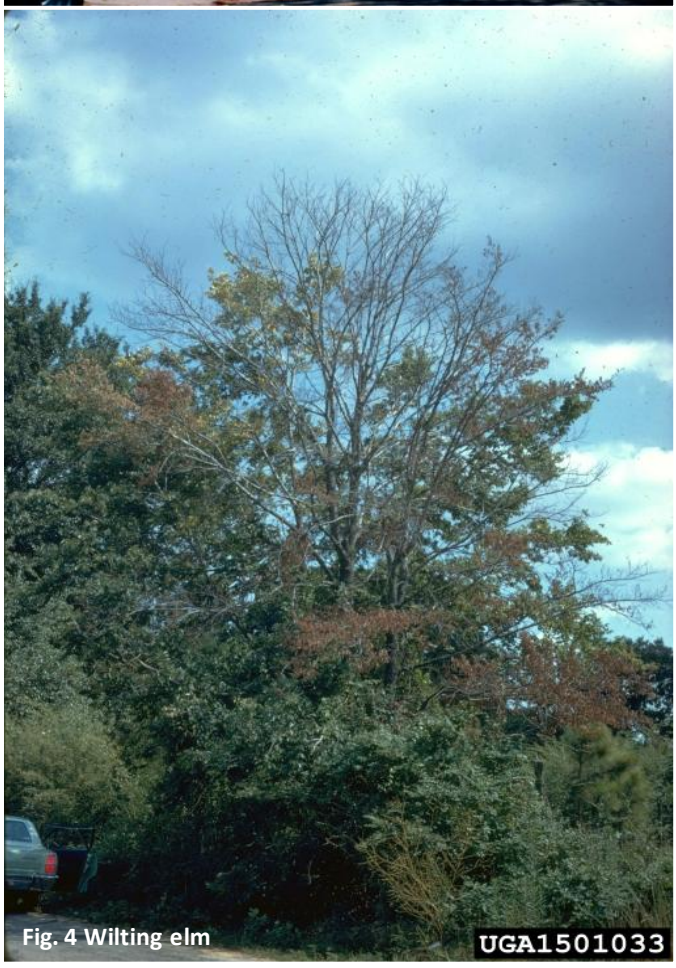

Fig. 4 Wilting elm

Fig. 5 Vascular discoloration in cross section

Fig. 6 Vascular streaking in sapwood

Laurel Wilt

Overview: First discovered in 2002, laurel wilt has rapidly become a disease of serious concern in the southeastern U.S. The laurel wilt fungus can kill mature trees very quickly, is vectored by a small ambrosia beetle from Asia, and is spreading through the southeast at approximately 20 miles per year. It affects plants of the Lauraceae family; most commonly redbay. The disease has become a serious concern because avocado (an economically important crop in Florida) is also susceptible, and the disease threatens the survival of several plant and animal species. Laurel Wilt was discovered in North Carolina in 2011.

Causal Agent: Fungus (*Raffaelea lauricola*) vectored by the redbay ambrosia beetle (*Xyleborus glabratus*)

Hosts: Plants of the laurel family; most commonly redbay, sassafras, and avocado. Also susceptible are swamp bay, silkbay, pondberry (endangered-U.S.), pondspice (threatened-NC), northern spicebush, and California laurel.

Symptoms / Signs: Symptoms of the disease may be similar to drought. Leaves begin to turn olive-grey, then reddish-brown, and begin to droop before wilting completely and turning brown (Fig. 1). Leaves are retained on dead trees for a year or more. Symptoms may start in an isolated branch or in the top of the tree, but will spread throughout the entire tree rapidly (Fig. 2 & 3). Most trees wilt and die a few weeks or months after symptoms first appear.

Discoloration in the outer sapwood is clearly visible in dead or dying trees. Staining will be dark purplish or black; vascular discoloration and be seen by looking at wilted branches in cross section (Fig. 8) or removing the bark to expose the xylem (Fig. 7). Staining in dead trees extends through the roots, stem, and all branches.

Redbay ambrosia beetles are very difficult to detect, especially prior to the tree's death. Small round entrance holes (< 1/32 inch diameter) may be visible on the stems and branches of severely wilted or dead trees. Toothpick-like tubes of sawdust may be expelled from beetle galleries and stick out of dead trees (Fig. 6); sawdust tubes are easily destroyed by wind or washed away by rain. Sawdust may be visible at the base of the tree. The beetle is 1/16 inch long, dark brown or black, and shiny (Fig. 4 & 5). There are many species of ambrosia beetles (native and exotic), many of which attack redbay and can be easily confused with the redbay ambrosia beetle. Suspected redbay ambrosia beetles should be collected and submitted for proper identification.

Disease Cycle: The redbay ambrosia beetle is a "fungus farmer." It creates galleries in dead or dying trees to grow the fungus on which it feeds. The beetle carries the fungus with it from tree to tree, and coincidentally, the fungus that the redbay ambrosia beetle farms is highly pathogenic on the beetle's preferred host tree. This creates a symbiotic relationship in which the fungus kills trees for the beetles to invade, and the beetles farm and carry the fungus to new trees. It only takes one beetle to infect a tree. Beetles cannot successfully attack healthy trees, so it is likely that the fungus is introduced to the tree during failed attempts by beetles to enter. Only after the tree is dead or dying can beetles return to infest the tree and farm the fungus.

Once introduced into the tree's vascular system, the fungus rapidly spreads in the sapwood throughout the entire tree. The tree responds by plugging its vascular system to prevent spread of the fungus, but this limits water movement in the sapwood. Therefore, trees wilt and die rapidly after infection.

Importance: High. The disease threatens the survival of many laurel species including the endangered pondberry. The Palmedes swallowtail butterfly and spicebush swallowtail butterfly require specific laurel species to complete their life cycle. Redbay and sassafras are important wildlife and landscape trees and are an important mid-story species.

Management: There are currently no effective control options. Research is being conducted on systemic insecticides and fungicides. Human movement of infested wood is responsible for the disease's rapid spread through the southeast; without human assistance the disease can only spread approximately 20 miles per year. If laurel wilt is suspected contact the NCFS Forest Health staff immediately.

Timeline: Trees may become infected when temperatures are warm enough for beetle activity. Symptoms usually develop during the growing season, but the tree is evergreen and the disease can be detected year-round.

Range: Eastern North Carolina; potentially statewide in sassafras. It was discovered in North Carolina in 2011.

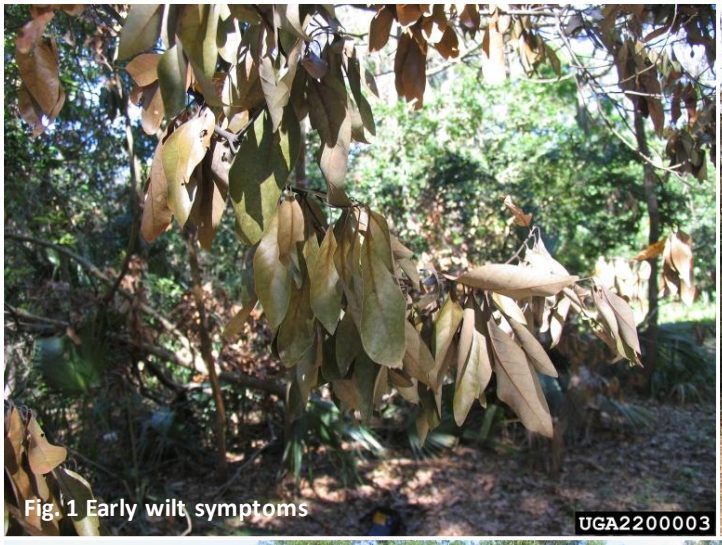
Fig. 1 Early wilt symptoms
UGA2200003

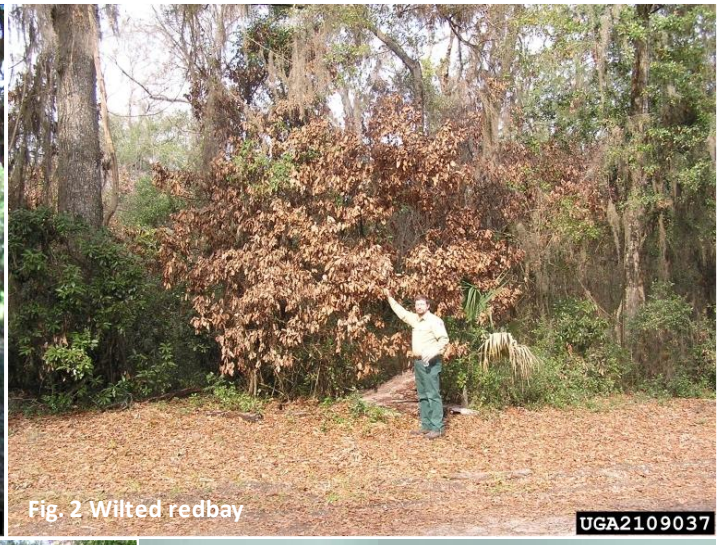
Fig. 2 Wilted redbay
UGA2109037


Fig. 3 Wilted redbays
UGA2200002


Fig. 4 & 5 Redbay ambrosia beetle
UGA5198081
UGA1413003


Fig. 6 Sawdust tubes
UGA2199082


Fig. 7 Vascular streaking
5383210


Fig. 8 Vascular streaking
UGA5005056

Heterobasidion Root Disease / Annosus Root Rot

Overview: Previously called Annosus root rot, Heterobasidion root disease is a serious disease in pine plantations that have been recently thinned. All species of conifers are susceptible. The fungus that causes Heterobasidion root disease infects freshly cut stumps and spreads to living trees through interconnected root systems (root grafts). The fungus attacks the large roots near the base of the tree, limiting the tree's supply of water and nutrients. The rotted roots of diseased trees make them highly susceptible to wind-throw.

Causal Agent: Fungus (*Heterobasidion annosum*)

Hosts: All conifers are considered susceptible, including all species of southern pines. Most common in loblolly, slash, and white pines. Longleaf and shortleaf pines, as well as hemlock, are also commonly infected. Annosus root rot is less common in spruces, firs, and cedar.

Symptoms / Signs: Symptoms first develop 1-3 years after thinning. Crowns may appear thin; needles may be chlorotic, stunted, and tufted at shoot tips (Fig. 1). Trees that have been killed may stay green through the winter, but needles will turn brown the following spring/summer.

Wind-throw is commonly observed in stands where Annosus root rot is present (Fig. 2). Rotted roots are unable to provide the necessary structural support. Windthrow may also be the first indication of infection; green and otherwise healthy looking trees have been known to fall over prior to the development of needle symptoms.

Roots may be resin-soaked (Fig. 3); white and stringy rot (Fig. 7) may be present in wind-thrown trees.

In late winter or early spring, fungal fruiting bodies (conks) may be produced at the base of the tree (possibly under the litter layer) (Fig. 5). The conks are brown on top, white and porous underneath, and have a creamy-white edge (Fig. 6).

Disease Cycle: Airborne spores are produced by conks in cool weather and germinate on freshly cut stumps and tree wounds. The fungus grows down into the root system and spreads to neighboring trees through interconnected root systems (root grafts). The fungus degrades the cellulose and lignin in large roots, especially around the base of the tree, cutting off the tree's supply of water and nutrients in addition to making the tree susceptible to wind-throw. Spores may be present almost year-round depending on the climate, but are most common during cooler months. Spores can travel for many miles. The disease usually dissipates 5-10 years after thinning.

Importance: Moderate. This disease is relatively common in North Carolina at low to moderate levels. Severe cases have been observed, and can devastate recently thinned pine stands. In severe cases, landowners may need to decide whether to harvest timber (possibly before rotation age) before maximum losses are sustained, or to allow the disease to progress and allow remaining trees to reach rotation age while accepting some losses.

Management: Thin stands during summer months when temperature is above 85ºF; spores can only germinate on stumps in cooler weather. If thinning during cool weather, stumps should be treated immediately after cutting with granular borax (or alternative product) to prevent infection (Fig. 4). Treatment with borax is ineffective once infection has occurred. Clearcut stands with greater than 50 percent infection; removal of diseased trees will result in an under-stocked stand. Wide spacing is recommended when planting to increase time to first-thinning, and to reduce the number of thinnings necessary in the stand. Wounding of roots and lower stems should be avoided during logging, fire break installation, and road building because these wounds are also suitable infection courts.

Timeline: Infection occurs during cooler months (< 85ºF). Conks are generally produced February through May. Symptoms can develop year-round.

Range: Statewide (Fig. 8). High hazard: sandy or sandy loam soils with at least 65 percent sand in the upper 12 or more inches above a clay layer and with no high seasonal water table. Intermediate hazard: silt and silt loam soils 12 or more inches deep. Low hazard: poorly drained clay and clay loam soils or those with high water tables.

Fig. 1 Declining trees

Fig. 2 Windthrow

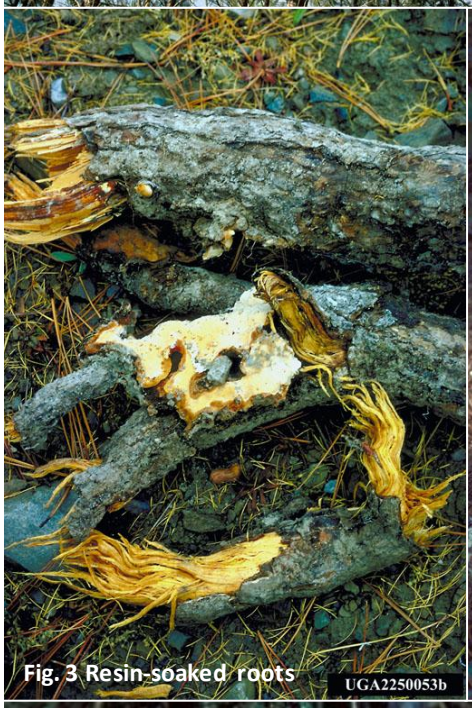

Fig. 3 Resin-soaked roots

Fig. 4 Borax treatment

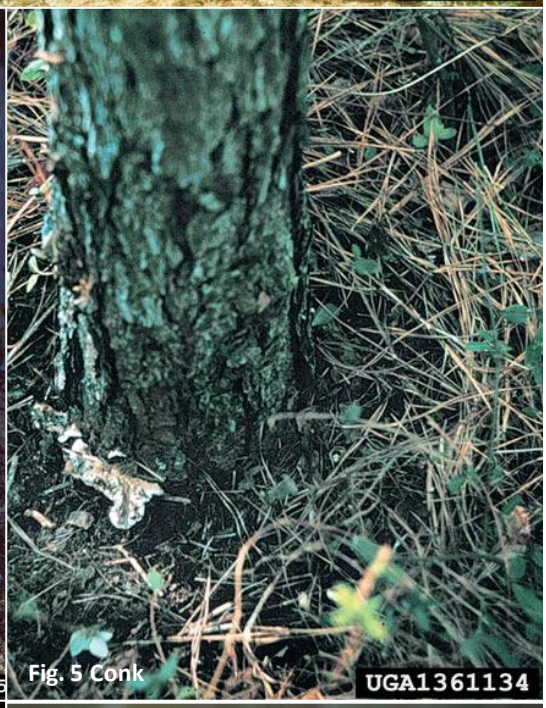

Fig. 5 Conk

Fig. 6 Conk

Fig. 7 Stringy, rotted roots

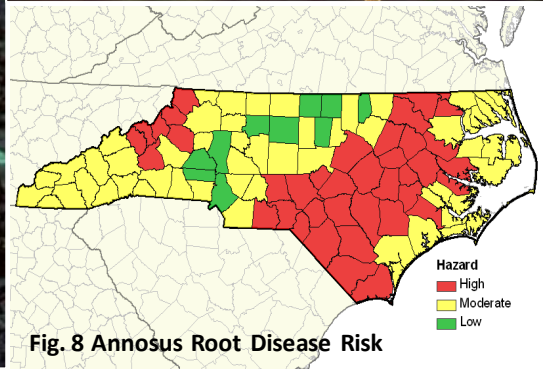

Fig. 8 Annosus Root Disease Risk

Armillaria Root Rot

Overview: Armillaria root rot is a general name for a group of diseases caused by fungi of the genus *Armillaria*. There are many species of *Armillaria* (many of which have only recently been described), but in general they are pathogens of the roots and lower stems of both hardwoods and conifers and are important decomposers of wood. Armillaria root rot is also called shoestring rot because the fungus produces black stringy rhizomorphs below the bark of infected trees. *Armillaria* fungi are commonly referred to as honey mushrooms because they produce large, golden-colored mushrooms around infected trees and stumps. Armillaria root rot is most often a secondary disease of stressed trees, but occasionally the pathogen can attack healthy trees as well. *Armillaria ostoyae* holds the record as the world's largest organism.

Causal Agent: Fungus (*Armillaria* spp.; most commonly *A. mellea* and *A. ostoyae*)

Hosts: Hardwoods and conifers.

Symptoms / Signs: The symptoms of Armillaria root rot often resemble many other diseases and disorders of trees such as drought, decline, Hypoxylon canker, Annosus root rot, and Phytophthora root rot. Growth reduction, chlorotic or scorched leaves, early fall coloration and/or premature leaf drop, branch dieback, wind-throw, and tree death are common above-ground symptoms. Conifers may produce large crops of undersized cones during decline. Trees are often affected in groups.

Armillaria causes cankers (lesions) on the inner-bark and outer-sapwood on the root-crown and lower stem. Cankers may expand slowly and eventually kill large roots; entire stems are not usually completely girdled, but large lesions may cause dieback or death. In conifers, cankers are often pitch-soaked, and resin may ooze and dry on the canker surface. Some cankers do not expand at all if the tree's defense responses are adequate; healthy trees may eventually compartmentalize infections. Callus / wound wood or scars may be visible at the site of old cankers for several years. After a tree dies, the fungus colonizes and causes decay in sapwood.

White mycelial fans (sheets of white fungal tissue) are often visible beneath the bark of cankers of rotted wood (Fig. 1 & 2). Black or brown branched rhizomorphs (also fungal tissue) that resemble fine roots or shoe strings may also be visible beneath bark, on root surfaces, and may even extend into the soil (Fig. 5 & 6). Rhizomorphs may be flattened when found beneath bark, but are cylindrical (< 1/32 inches in diameter) when found on the bark surface or in the soil. Golden-yellow mushrooms may be produced around dead or diseased trees in the fall (Fig. 3 & 4). Many species of *Armillaria* are bioluminescent. Presence of *Armillaria* signs does not necessarily mean the fungus is the cause of death or disease; it is a common wood rotter.

Disease Cycle: Stressed trees are highly predisposed to *Armillaria* root rot. *Armillaria* spreads through a forest via rhizomorphs and airborne spores. Rhizomorphs are made up of densely packed fungal hyphae to form fine root-like structures. Rhizomorphs can grow through the soil (up to 8 feet per year) feeding on organic matter as they go, until they reach the roots of susceptible trees. Rhizomorphs attach to tree roots and penetrate the bark by mechanical force and enzymatic degradation. Airborne spores are produced from golden-yellow mushrooms (honey mushrooms) that grow around the base of diseased trees. Spores infect stumps or wounds on lower stems and exposed root tissue. The hyphae of a single fungus can spread great distances through the soil; the largest organism in the world is an *Armillaria* in Oregon that is over 2500 hundred acres in size.

Importance: Moderate. Armillaria root rot kills many stressed trees; young conifers are frequently attacked on sites previously occupied by hardwoods.

Management: Prevention is difficult; no practical treatment options are available. It is critical to maintain proper tree health. Select the proper tree species for the site; provide adequate water and fertilization if necessary. Avoid mechanical damage and soil compaction. Remove diseased trees and infected root systems if possible.

Timeline: Honey mushrooms are produced in the fall. Other signs may be visible year-round. Symptoms occur during the growing season.

Range: Statewide.


Fig. 1 Mycelial fans
UGA1473102


Fig. 2 Mycelial fans
UGA4822097


Fig. 3 Honey mushrooms
UGA1371018


Fig. 5 Rhizomorphs
UGA5027093


Fig. 4 Honey mushrooms
5366752


Fig. 6 Rhizomorphs
UGA0364052

Littleleaf Disease

Overview: A type of *Phytophthora* root rot, littleleaf disease is considered to be the most important disease of shortleaf pine in the United States. The pathogen attacks the fine roots of pines, inhibiting their ability to absorb nutrients. The disease is often found in coordination with low soil nitrogen, poor soil drainage, nematodes, and species of *Pythium*. Littleleaf disease usually only affects trees greater than twenty years old; it takes several years for a tree to be killed after symptoms first appear.

Causal Agent: Stramenopile; a fungus-like organism also known as a water mold (*Phytophthora cinnamomi*)

Hosts: Shortleaf pine is highly susceptible; loblolly pine moderately susceptible. Littleleaf disease has also been reported in other southern pine species including Virginia, slash, pitch, and longleaf pines.

Symptoms / Signs: First symptoms are caused by nutrient deficiency due to loss of fine roots. These symptoms include thin crowns, needle chlorosis, stunted needles, and reduced shoot growth (Fig. 1).

As symptoms progress, the crown will look increasingly thin. Needles will become increasingly yellow, then turn brown. Eventually, the tree will lose almost all of its needles except the new growth, giving the shoots a "tufted" appearance (Fig. 2). Prolific production of cones (often stunted) may occur in later stages of disease (Fig. 3 & 4). Branches will begin to die starting in the bottom of the crown, progressing upward. Width of annual growth increments / diameter growth is greatly reduced.

Disease Cycle: *Phytophthora cinnamomi* rarely affects trees less than twenty years old. The pathogen attacks the fine roots that absorb most of the tree's supply of essential mineral nutrients and water. Usually, diseased trees are found in soils that are nutrient deficient and/or poorly drained, or in trees being attacked by nematodes or other pathogenic soil fungi which can weaken the tree's defenses. The pathogen produces motile spores which can swim for short distances, therefore rate of disease spread and risk of infection is highest in soils with poor drainage or that frequently exhibit high soil moisture. Trees are killed slowly, generally over the course of a 1 to 12 year period. On average, trees die 6 years after the first symptoms are observed.

Importance: Moderate. This disease is usually present in North Carolina at low to moderate levels. Trees declining due to littleleaf disease may be more susceptible to attack by bark beetles. The disease is very important for shortleaf pine management.

Management: Avoid planting susceptible species on high-risk sites. Shorten lengths of rotation to harvest trees before they become susceptible. Remove diseased trees. Increase soil drainage through site preparation techniques including break-up of hardpan layers. Inter-plant legumes in susceptible stands to increase available soil nitrogen. Alternatively, fertilize with one ton of 5-10-5 fertilizer + one-half ton of ammonium sulfate per acre for high value trees. Fertilization will delay symptom development for 3-4 years.

Timeline: Year-round. Pathogen is most active during periods of high soil moisture. Symptom progression may be accelerated by periods of drought.

Range: Potentially statewide. It is most common in the piedmont region of North Carolina (Fig. 5).

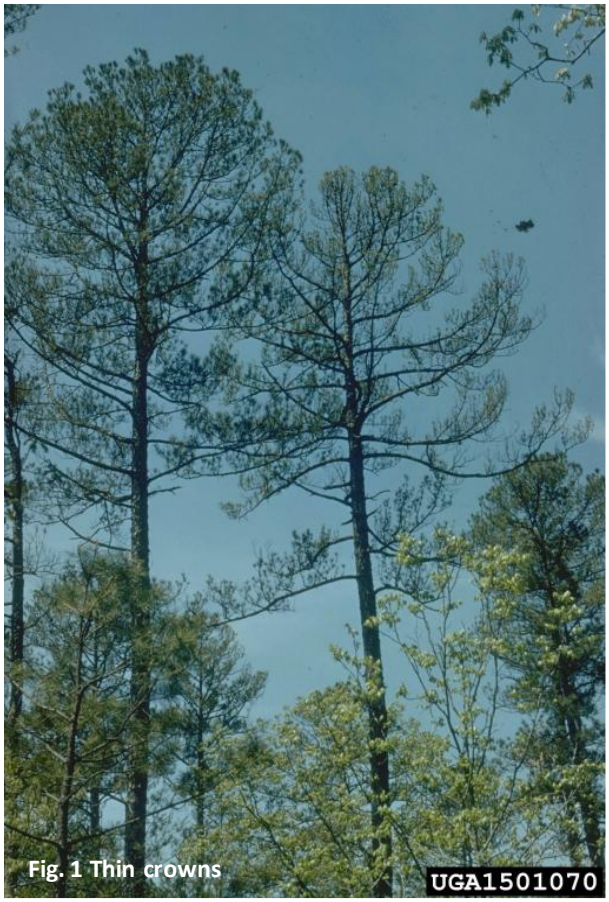

Fig. 1 Thin crowns

Fig. 2 Thin crown and tufts of foliage

Fig. 3 Stunted cones

Fig. 4 Stunted cones

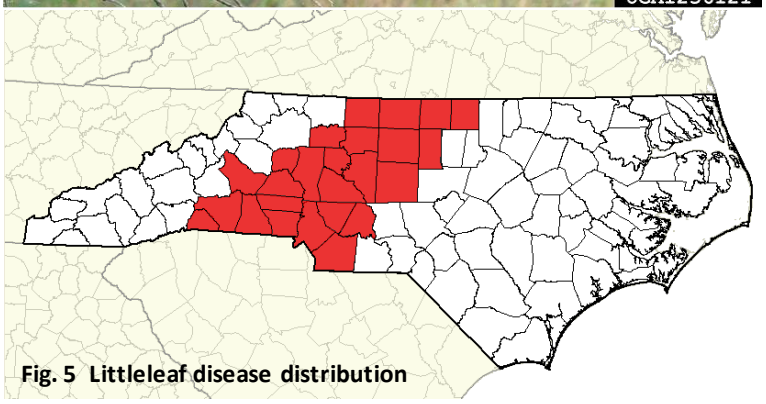

Fig. 5 Littleleaf disease distribution

Phytophthora Root Rot

Overview: Phytophthora root rot is a general name for a group of diseases caused by species of *Phytophthora* belonging to a group of fungus-like organisms. The pathogen attacks the roots of many trees and shrubs, particularly the fine roots that absorb mineral nutrients from the soil. The pathogen prefers poorly drained soil and produces motile spores that can swim through saturated soil for short distances. In North Carolina, the disease is particularly important in Christmas tree production (Fraser firs, Fig. 1), fruit trees, and many ornamentals. (*See also Littleleaf Disease*).

Causal Agent: Stramenopile; a fungus-like organism also known as a water mold (*Phytophthora* spp.)

Hosts: A wide variety of trees an ornamental shrubs including: azalea, rhododendron, dogwood, camellias, yews, deodar cedar, mountain-laurel, heather, juniper, blueberries, Fraser fir, white pine, shortleaf pine, fruit trees, and others.

Symptoms / Signs: Phytophthora root rot can often be difficult to diagnose and laboratory tests are usually required to confirm the presence of the pathogen.

Above-ground, symptoms mimic nutrient deficiency, drought, or decline such as leaf chlorosis, leaf necrosis, wilting, dieback, or death. In Fraser fir, needles will turn yellow-green and may wilt before turning brown. Symptoms often begin in lowest branches progressing upward, or may only affect one side or section of the tree. Plants may decline over several months or several years.

The bark on large roots of infected trees may only be loosely attached; root tissue beneath bark is often discolored red, brown, grey, or black (Fig. 6 & 7). Feeder roots are often fewer in number or even completely absent (Fig. 2). The bark on small roots is easily pulled off the center core; small roots may be mushy, reddish-brown, or black (Fig. 3). The lower stem or root crown of severely diseased plants may be sunken in, swollen, pitch-soaked, or have roughened bark (Fig. 4). On Fraser fir, removal of bark from the lower stem may reveal butterscotch, brown, or black colored sapwood (Fig. 5). Dead or dying trees often occur in groups or in low-lying areas. Seedlings of many trees and shrubs can be killed rapidly by *Phytophthora* under the right conditions; this is known as "damping off." Phytophthora root rot is most often confused with damage caused by soil inhabiting grubs and abiotic disorders.

Disease Cycle: *Phytophthora* overwinters in the soil, infected roots, and other plant debris. The pathogen can lie dormant in the soil for many years. Spore production and subsequent infection occurs in warm and saturated soils. Motile spores can swim through saturated soils for short distances to infect new plants. The pathogen is also easily spread in irrigation water, water runoff, rain-splash, contaminated equipment, and on contaminated plants. The pathogen attacks the fine roots and nutrient-conducting tissue of larger roots and root crowns.

Importance: High. *Phytophthora* species can cause significant and costly damage to landscape trees and shrubs. It is the only serious disease of Fraser fir, and can be responsible for major losses in Christmas tree plantations if soil becomes contaminated and conditions are conducive to spread and infection.

Management: No single practice will prevent Phytophthora root rot; an integrated management approach must be taken. Do not plant susceptible species on sites where the disease has been known to occur; utilize resistant and/or disease-free plants. Obtain plants and soil from reputable sources. Remove and destroy diseased plants (including the root system). Plant in well drained soil; utilize site preparation techniques to improve soil drainage. Promote overall plant health by providing adequate water, fertilization, and maintaining proper soil pH. Avoid over-watering and use of nitrogen rich fertilizers. Crop rotation with resistant plant species is often a successful way to reduce or eliminate soil inoculum. Pesticides are available for specialized applications.

Timeline: Spore production and infection occurs from late spring through fall during saturated soil conditions. Symptoms are most visible during the growing season.

Range: Statewide.

Fig. 1 *Phytophthora*-killed fir

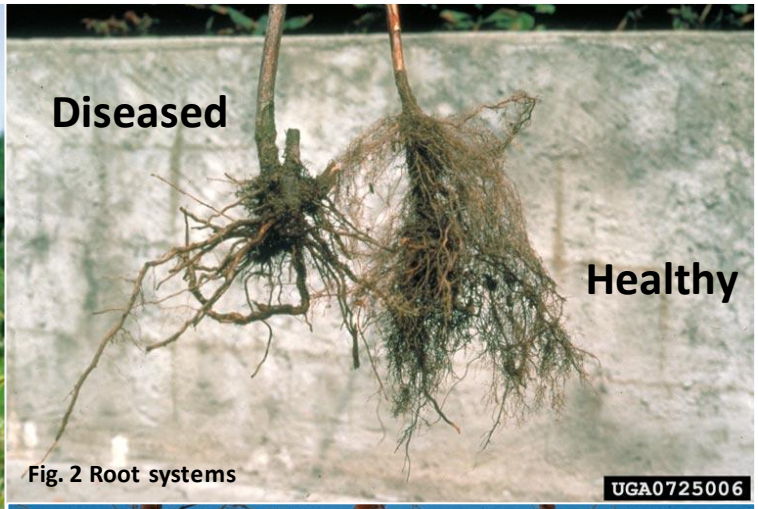

Fig. 2 Root systems

Fig. 3 Rotted roots

Fig. 4 Sunken stem canker

Fig. 5 Vascular discoloration

Fig. 6 Infected root crown

Fig. 7 Discolored sapwood

Procera Root Disease

Overview: Relatively little is known about Procera root disease, mostly because it was not recognized until the mid-1980s; little research has been conducted on it since. In the past, it was a concern to Christmas tree growers, but over the past two decades it has been found more commonly in stands of white pine. The disease tends to affect trees 3-15 years old; therefore it is becoming a concern for sawtimber production and the establishment of mature white pine stands. The pathogen causes cankers on large roots, the root crown, and lower stem, but smaller roots can also be affected. Trees may die quickly, or over several years.

Causal Agent: Fungus (*Leptographium procerum*)

Hosts: Eastern white pine. Occasionally affects Scots and Austrian pines

Symptoms / Signs: Procera root disease usually affects trees 3-15 years old. Infected trees may exhibit delayed bud-break and reduced candle length (Fig. 1). Trees often begin to die from the top down. Sometimes dieback is slow (one whorl at a time), in other cases (especially younger trees) the entire tree may die suddenly (Fig. 2). In the latter case, trees often begin to turn yellow before turning completely brown.

Resin flow may be visible (sometimes obvious) at the base of the tree (Fig. 3 & 5); the trunk may be flattened on the affected side. Removal of the bark may reveal a chocolate-brown or tan canker on the lower stem (Fig. 4), root crown, or large roots, but cankers may not always be visible. Beetle galleries may be found around or within cankers (Fig. 6).

Disease centers containing more than a dozen diseased or dead trees have been observed, however, the pattern of disease development is often random within a stand.

Disease Cycle: Little is known about the disease cycle of Procera root disease. The pathogen does not appear to survive in the soil for long periods of time; rather it survives in infected root systems where it is vectored to neighboring trees by a variety of root-attacking or bark-infesting insects. Large roots are most often attacked and cankers frequently form in the inner bark and sapwood that eventually girdle major roots or the entire stem. Alternatively, small roots may be attacked, leaving little or no evidence of Procera root disease in dead trees. It is believed that this is a secondary disease, requiring certain pre-disposing factors for a successful attack by the pathogen. Wet sites appear to be conducive to disease development and should be avoided when planting white pine, but the disease seems to appear randomly in a stand, even on dry sites.

Importance: Moderate. Procera root disease may hinder efforts to establish mature white pine stands, kill valuable ornamentals, and prohibit the production of certain Christmas tree species on sites with previous Procera-related problems. In parts of North Carolina, anecdotal evidence suggests most white pines do not live more than 30 years, possibly because of Procera root disease.

Management: Few management options are available. Avoid planting white pines on wet sites as the disease seems to be more common in these areas. Excessively dry or otherwise poor sites should also be avoided as stressed trees appear to be much more susceptible. Remove and destroy diseased trees, including as much of the stump and root system as possible. Do not plant white pine on or near sites where Procera root disease has been known to occur. Provide adequate water and maintain proper soil pH (do not over-water).

Timeline: Unknown.

Range: Statewide.

Fig. 1 Stunted growth

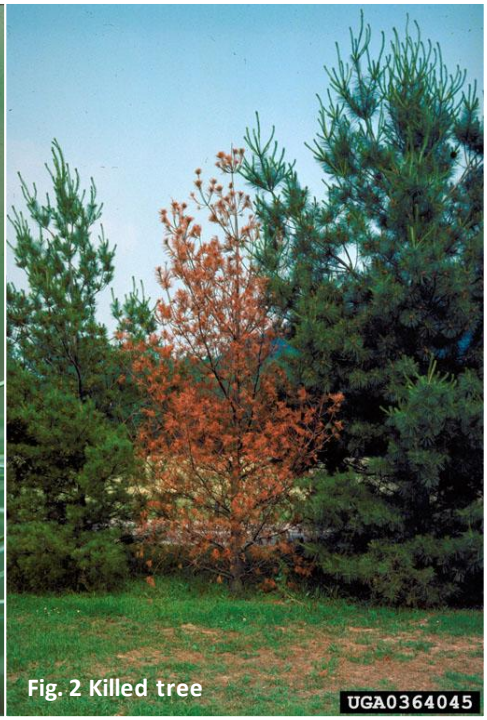

Fig. 2 Killed tree

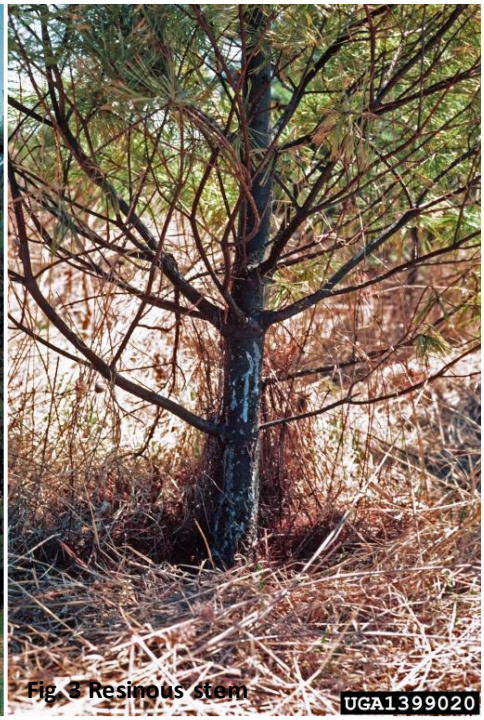

Fig. 3 Resinous stem

Fig. 4 Discolored sapwood

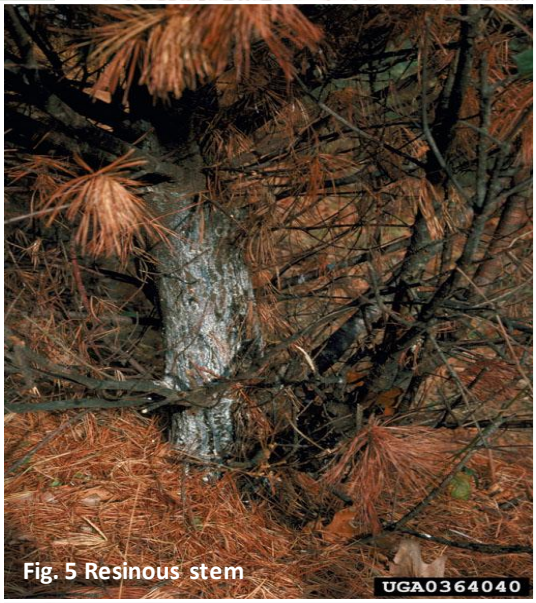

Fig. 5 Resinous stem

Fig. 6 Insect infestation

Disorders and Abiotic Stress Agents