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Forest Nursery Diseases

in the

United States

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Agriculture Handbook No. 470

Forest Service
U.S. Department of Agriculture

Forest Nursery Diseases in the United States

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Discusses hosts, symptoms, damage, life cycles, distribution of pathogens, and methods for control for 31 diseases of seedlings in forest nurseries—9, root; 8, stem and branch; 14, foliage. Includes a short discussion on storage molds, plus selected references, glossary, disease index, and host index.

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Forest Nursery Diseases in the United States

INTRODUCTION

Forest nurseries and the seedlings they grow play an important part in keeping this Nation's forest lands productive. The need for more trees and forests, for a host of purposes, is steadily increasing. Currently, millions of acres of commercial forest land under private, State, or Federal ownership are nonstocked or poorly stocked. More of these nonproductive acres are created each year by fire, pests, and timber harvests. Planting of these understocked forest lands is one means of avoiding or reducing a future timber shortage. At the 1972 National Tree Planting Conference, John McGuire, Chief of the USDA Forest Service, said, "I believe that tree planting should be the main thrust in meeting the challenge of providing for American forests." Our forest tree nurseries must play the key role in this program by providing forest managers with the needed quality and quantity of planting stock.

Factors that influence the quantity and quality of nursery seedlings may also influence our future timber supply. These factors include seedling diseases caused by fungi, bacteria, and nematodes. These diseases, which are active from the time of sowing through outplanting, may kill seedlings directly, or stunt or malform them so they must be rejected. Nursery diseases may also lower field survival of outplanted seedlings. Lastly, nursery diseases may be a threat to our forests when infected seedlings are planted in forested areas where the disease does not and has not existed. The introduction of white pine blister rust into North America on seedlings is the most notable example of this kind of disaster.

The economic losses due to diseases in forest nurseries must include more than just the cost of producing the dead and culled seedlings. They include the cost of a second site preparation of the plantation when plantable seedlings are not available due to disease losses at the nursery, or the cost of a second planting or interplanting when seedling diseases continue to cause mortality in the plantation. The basic land cost must also be included when forest land is held out of production by nursery disease problems. Thus the economic losses resulting from nursery diseases are not restricted to the nursery operation; impacts on the forests may be even greater.

SCOPE

The purpose of this Handbook is to assist the nurseryman, forester, extension pathologist, and others in identifying, evaluating, and controlling the diseases encountered in forest nurseries.

In preparing this Handbook we have tried to bring together all the currently available information on seedling diseases that have been a problem in our forest tree nurseries. In most cases, each disease is treated separately in the following standard format: Distribution, Hosts and Damage, Life History, Control, and Selected References. This information has been gathered and presented by authorities who in most cases have worked with these individual disease problems in the field. Limited coverage of some diseases indicates our lack of knowledge about them. General information on disease diagnosis, disease control, and pesticide handling is included in the introduction. A glossary, a disease index, and a host index are in the back of the Handbook.

We have not attempted to cover the subjects of nutrient deficiency, herbicide injury, or environmental injury (except air pollution). These large subjects could not be adequately treated in this Handbook, and probably deserve separate coverage. There are also numerous minor diseases, such as some of the foliage diseases of hardwoods, about which we have such limited information that we made no attempt to cover them.

If further information or assistance is required, the nurseryman should contact a plant pathologist from one of the agencies listed below in his area:

Forest Pest Control, Forest Service, U.S. Department of Agriculture
Agricultural Extension Service, State university
State Department of Agriculture
State Department of Forestry

DISEASE DIAGNOSIS

Within each disease writeup, the sections on distribution, hosts, and damage will be the most helpful in diagnosing a particular disease. This diagnosis is an investigation to determine the cause of a condition or problem. It involves the art of identifying a disease from its symptoms, signs, and patterns. As in all investigations, it requires a systematic search for clues, and the assembly of these clues into a reasonable explanation of the situation. The symptoms (expressions of the diseased host), signs (evidences of the cause), and patterns of occurrence, are the clues upon which we must base our determinations.

The following guide may be helpful in diagnosing tree disease:

1. Determine as accurately as possible the part of the plant which is actually affected. The death of only the needles indicates a needle disease; of the stem and/or branches—a canker disease; of the whole tree—a root disease, or drought. Note the pattern

of the disease in the seedlings. Is the damage limited to the south side? to the lower or upper crown?

2. Note what species are affected. Are there any individuals which are affected less or which are free of the problem?

3. Note the pattern of occurrence. What areas show the problem most severely? How do these areas differ from those areas free of the problem? Are these problem areas in any particular portion of the bed or related to a particular cultural activity?

4. If the cause of the disease is not immediately evident, look first for the simplest causes, such as animal damage, frost, simple injuries, or other obvious causes of trouble.

5. Look for the presence of fungi, insects, or other parasites. Observe accurately, and try to judge whether the organisms found are the main cause of the trouble or just secondary.

6. If the whole tree is dead or suffering and nothing is found aboveground to indicate the cause of the disease, expose the roots and root crown for examination.

7. If still unsure, learn about the recent history of the problem and the area. Is the problem of recent origin? When was it first noted? What cultural practices have been used in the area, such as the use of herbicides, fertilizers, irrigation, or flooding? Can these be related through pattern or time of appearance to the injuries?

DISEASE CONTROL

There are two general types of disease control available to the nurseryman: cultural and chemical.

In cultural control methods, the nurseryman maintains control over a disease problem by performing routine nursery operations in certain prescribed ways. Such prescriptions may dictate specific seedbed densities, time of planting, watering schedule, and so forth. Most of these cultural methods control a disease problem by modifying the environment so that it becomes unfavorable for the disease or its causal organism.

Chemical control methods rely mainly on treatment before the disease becomes established, either by eradicating the pest from the area (soil fumigation) or by protecting the plant with a chemical barrier (foliage sprays). Once a plant has been infected it is very difficult to cure the plant with chemicals. This means that the nurseryman must be prepared to apply his chemical controls either just before or at the first appearance of the disease.

In some cases the information in this Handbook on control measures—particularly on fungicides—is very generalized, because pesticide regulations prohibit the recommendation of non-registered fungicides for plant disease control. As information required for pesticide registration becomes available, more specific recommendations can be made.

USE AND SAFE HANDLING OF PESTICIDES

Some States have restrictions on the use of certain pesticides. Check your State and local regulations. Also, because registrations of pesticides are under constant review by the Federal Environmental Protection Agency, consult your county agricultural agent or State extension specialist to be sure the intended use is still registered.

The following rules should be followed when handling pesticides. These rules should be read by all persons involved in pesticide use. Copies of these rules should be posted in several places, particularly in the areas of pesticide storage.

Pesticides are poisonous and always should be used with caution. If used properly, they will not cause injury. The dangers associated with mishandling and misapplication of pesticides, however, include possible injury to the operator and handler, and damage to the seedling crop, to the equipment, and to the environment. Read the Health and Safety codes of your organization pertaining to use of toxic chemicals prior to use of pesticides.

1. *Read the label.*—Handlers should read, understand, and follow all instructions on the label. Notice warnings and cautions before opening the container. Repeat the procedure every time, no matter how familiar you think you are with the directions. Apply the material only in the amounts and at the times specified.

2. *Avoid contact.*—Avoid inhaling sprays and dusts. Avoid contact with skin and eyes. When directed by the label, wear the proper protective clothing and a mask. Do not eat or chew while spraying or dusting. Wash thoroughly before eating.

3. *Apply safely.*—Use only the specified dosages and mix as directed. Do not use your mouth to siphon liquids from containers or blow out clogged lines or nozzles. Use clean, well-functioning equipment to apply the pesticides. Do not spray with leaking hoses or connections. Do not work or allow others to work in the drift of the spray or dust.

4. *Wash immediately.*—Stop and wash off any pesticide spilled on the body. Remove contaminated clothing. Wash and change to clean clothing after spraying or dusting. Also, wash clothing each day before re-use.

5. *Dispose of containers properly.*—Always dispose of empty containers so that they pose no hazard to humans, animals, or plants (either terrestrial or aquatic). When in doubt on proper disposal procedures, contact the nearest agricultural authority.

6. *Store safely.*—Keep pesticides stored together outside the home or office away from food and usual working areas. Keep them under lock and key. Label and sign the area well and do not store other chemicals among the pesticides. Always keep the pesticides in the original containers, and keep them tightly closed.

7. *Report illness.*—If symptoms of illness occur during or shortly after dusting or spraying, call a physician or get the patient to a hospital immediately.

I. ROOT AND SOILBORNE DISEASES

Root and soilborne diseases are those diseases of the below-ground portions of seedlings which may occur from the time of planting until lifting. Included in this group are pre- and post-emergence damping-off, and diseases of the root crown, taproot, lateral roots, and tips. The diseases in this group are caused by bacteria, fungi, and nematodes. A section on mycorrhizae also is included in this group because they involve an infection of the root tips, even though beneficial to the host.

Root diseases are some of the most difficult disease problems to diagnose. The above-ground symptoms of most root diseases are very similar to one another and also to symptoms caused by unfavorable soil conditions such as high plow pan, excessive soil moisture, soil toxicity, and some nutrient deficiences.

The following diseases are considered to be the most important and common root and soilborne diseases found in North American forest tree nurseries.

1. DAMPING-OFF

T. H. FILER, JR. AND GLENN W. PETERSON*

Damping-off is one of the first diseases the nurseryman encounters in his newly sown beds. Many fungi cause damping-off, but those most commonly encountered are *Phytophthora* spp., *Pythium* spp., *Fusarium* spp., and *Rhizoctonia solani.* The disease sometimes causes 15 percent or greater mortality of the viable seed, and must therefore be considered of major importance.

Only very young seedlings are attacked; after the stems begin to develop woody tissue—perhaps in 4 to 6 weeks after seed germination—susceptibility declines rapidly.

Distribution and Hosts

Damping-off is widespread in nurseries throughout North America. Most conifer species are susceptible, with the notable exception of the junipers. Among the hardwoods, American and Siberian elms, black locust, Russian mulberry, sweetgum, sycamore, yellow-poplar, and Russian-olive are very susceptible. Green ash, northern catalpa, hackberry, honeylocust, and bur oak have considerable resistance.

Symptoms

Symptoms vary, depending on when infection occurs. Early attacks on the developing radicle may kill the seedling before it emerges from the soil. Such preemergence damping-off may go undetected or be explained away as "poor seed."

Postemergence infection characteristically occurs at or just below the groundline, resulting in a water-soaked or necrotic area on the succulent stem. In conifers this tissue collapses, and the seedling becomes flaccid and topples over. In contrast, hardwood seedlings usually remain upright, gradually wilt, and break off. Damping-off is often confused with heat lesions, which appear as whitish sunken areas on the stem just above the groundline. Heat lesions, however, usually occur on only one side of the stem.

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Disease Development

The causal fungi as a rule are native to the nursery soils. Some live on dead organic material, while others survive as resting or dormant spores while awaiting a new host to infect. Populations of these pathogenic fungi tend to build up in nursery soils with each successive year of cropping, and average seedling losses tend to increase.

Any condition that reduces seedling growth and vigor usually causes an increase in infection. The disease usually is most severe in nursery soils that are excessively wet. A stand of apparently healthy seedlings may show severe damping-off after just 1 or 2 rainy days. Sideboards and shading frames that restrict airflow may increase infection, since the beds will not dry rapidly. Temperature also influences development of this disease, depending upon the fungus species involved. Some fungi, such as *Pythium* spp., are favored by high temperatures (27°–35°C). Nitrogen fertilizers applied before or during the period of seedling susceptibility may also increase losses. Soil pH is significant; losses are usually greatest at high pH's and minimal near pH 5.5.

Control

Damping-off may be controlled by cultural or by chemical means. Losses have generally been reduced by practices that increase the rate and uniformity of germination and the speed at which the seedlings grow in early weeks. Regulation of soil moisture may reduce losses from certain fungi.

The seed-protectant fungicide thiram is sometimes effective, but also often inconsistent, in reducing losses in conifer beds. Fungicides may reduce the amount and speed of germination if temperatures are high.

Soil treatment prior to sowing is feasible. Applying inorganic acids to reduce pH has been useful on soils with pH range 6–8. Drenching soil with the fungicide captan is also effective, but once symptoms become evident it is usually too late to drench.

Good results may be achieved by soil fumigation prior to sowing. Methyl bromide, chloropicrin, vorlex, or combinations of these and other fumigants give excellent control of damping-off and other soilborne diseases in both hardwood and pine seedlings.

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2. FUSARIUM ROOT DISEASE

Fusarium oxysporum Schlect. emend. Snyd. & Hans.

RICHARD S. SMITH, JR.*

Fusarium root disease is one of the most common diseases of conifer seedlings in the world. Besides causing a root disease, *F. oxysporum* and other species of *Fusarium* are often responsible for damping-off in earlier stages of seedling development. This section is concerned only with the root disease. The subject of damping-off is covered in article 1 of this Handbook.

Distribution

This disease is common and widespread through most of North America. It has been especially important in the southeastern, southern, and western parts of the United States. Although it appears to be favored by the warmer climates, it is not restricted to these areas.

Hosts and Damage

Most conifer seedlings are susceptible to the Fusarium root disease. The pathogen is known to exist in several forms which are host specific. So far as conifer seedlings are concerned, only one specialized form, *F. oxysporum* f. sp. *pini,* has been described.

F. oxysporum attacks and kills the roots of a seedling, causing chlorosis, stunting, and wilting of the top and eventually death. As with many root diseases, the effects are chiefly (a) seedling mortality in the nursery bed; (b) increased number of cull (stunted) seedlings; and (c) increased losses after outplanting because of impaired root systems.

Life History

The pathogenic forms of *F. oxysporum,* like so many other root disease fungi, are inactive in the soil in the absence of a host. They usually remain dormant in the form of chlamydospores—microscopic, thick-walled, single-celled resting spores. When a seedling root grows by a dormant chlamydospore, exudates from the root supply nutrients to the spore and stimulate its germination. The fungus grows over the root surface, penetrates between two epidermal cells, and spreads intracellularly through

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the cortex. The fungus colonizes the cortex and the xylem elements of the infected seedling. The exact cause of seedling death is not clear. Most pathogenic forms of *F. oxysporum* cause a vascular wilt, although many descriptions of the disease in conifers suggest it is a cortical rot. Either or both may be involved in seedling death.

After the pathogen has killed the seedling and has colonized the host tissue, it converts much of its mass into macroconidia and chlamydospores. The macroconidia are large, multicelled spores which may infect other roots, but which usually convert themselves into chlamydospores—when conditions become unfavorable—and await next season's crop.

The *Fusarium* fungus may be carried with the plant when it is outplanted and continue to attack the infected seedling roots, causing unexpectedly high transplant mortality. Although this fungus may continue activity for a year or two, the outplanted seedling may outgrow the infection if the infection is light. Studies in the West have shown that *Fusarium* disappears after 3 or 4 years from the roots of seedlings outplanted into the forest. Thus, although the disease may be the cause of first-year transplant mortality, it will not persist in the forest environment and cause continuing problems.

Control

Cultural methods on the whole have not been effective in controlling this disease. Soil amendments, which showed promise in controlling certain *Fusarium* diseases of agricultural crops, have not yet proved to be adequately effective in the forest nursery.

Fumigating the soil with methyl bromide, chloropicrin, or other soil fumigants before planting currently is the best method of control. Fumigation has been used effectively in many parts of North America. Soil fungicides have in most cases been less effective than soil fumigants.

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3. CHARCOAL ROOT DISEASE

Macrophomina phaseoli (Maubl.) Ashby

RICHARD S. SMITH, JR.

The charcoal root disease caused by the fungus *Macrophomina phaseoli* (*Sclerotium bataticola*) is one of the most important diseases in forest nurseries of western North America. It was originally described as a disease of agricultural crops, but became important in the 1950's and 1960's, when the forest nursery program began to expand. The general shift to large, more efficient forest nurseries in the warmer agricultural areas resulted in an increase in the charcoal root disease.

Distribution

This disease has worldwide distribution in the temperate and tropical areas of both hemispheres. It is found in most of the warmer agricultural areas of the United States. In the South and Southeast, this fungus, together with other fungi, causes the black root rot of pine. In the western United States it is a problem in the warmer lowland agricultural soils, but not in the cooler, higher elevation forested areas. It is also absent from the cool coastal areas of the Pacific Coast States.

Hosts and Damage

M. phaseoli attacks more than 300 species of plants including many agricultural plants, forest tree seedlings, and native weeds. All conifers are probably susceptible to some degree. Field observations suggest that ponderosa and Jeffrey pines are the least susceptible while sugar and Monterey pines, Douglas-fir, red and white fir, and giant sequoia are the most susceptible.

Life History

The fungus invades the root tips, lateral roots, and root crown, and destroys the cortex, phloem, and cambium. The gradual destruction of the root system causes the seedlings to become stunned and chlorotic, and finally to die.

Nursery managers usually feel the impacts of losses in three ways: (a) seedling mortality in the nursery; (b) increased number of cull (stunted) seedlings; and (c) increased losses of outplanted seedlings due to damaged root systems.

The fungus overwinters or lies dormant in the soil in the form of sclerotia—small, black, spherical resting structures. It does not grow or develop in the soil in the absence of a host plant. When the growing root of a susceptible plant comes in contact with a sclerotium, it is stimulated by the root to germinate. The fungus grows over the root surface and penetrates between the epidermal cells into the root cortex. In the cortex the fungus grows through the root toward the root crown, destroying the cortex, phloem, and cambium as it proceeds. The fungus continues to develop within these dead tissues, forming masses of new dormant sclerotia both in the cortex and between the xylem elements.

These sclerotia can be seen easily in the roots through a hand lens, and hence are helpful in diagnosis. As other saprophytic microbes decay the remains of the infected roots, and as soil movements break up rotten roots, the new sclerotia are liberated into and distributed within the soil to await the next crop of seedlings. These sclerotia may remain dormant for many years awaiting a suitable host.

Temperature plays a critical part in this disease. The fungus requires rather warm soils (15°–18° C) before it can become damaging. In California nurseries, this disease does not occur until midsummer.

Depending on environmental conditions of the outplanting site, the fungus may or may not continue to threaten the transplanted seedlings. In an infested nursery, many seedlings become mildly infected and never show any symptoms. When outplanted, these seedlings carry the disease with them. The fungus ceases to develop and gradually dies in seedlings planted in cool forested sites. In warm soils, the fungus continues to develop and may kill the transplant. Infected transplants which are placed under stress, such as a lack of moisture, are most susceptible.

Control

Most cultural control measures are not effective against this pathogen. The ability of the sclerotia to survive many years in the soil and their wide host range limit effectiveness of fallow periods and cover crops as control measures. In some areas where soil temperatures are marginal for disease development, seedling mortality may be reduced by shading, but the general level of seedling infection is not usually reduced markedly.

The most successful control measures to date are preplant soil fumigations with broad-spectrum biocides such as methyl bromide or mixtures of methyl bromide and chloropicrin. Soil fumigation either in spring or early fall before planting is effective in controlling this disease. Both pre- and post-planting applications of several fungicides have been tested, but none has done an adequate job.

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4. BLACK ROOT ROT OF PINE

Sclerotium bataticola Taub.

CHARLES S. HODGES*

Black root rot is the most serious root disease of pine seedlings in southern forest tree nurseries. Mortality of seedlings is not too great in the nursery as long as adequate water is available, but survival of outplanted diseased seedlings is low.

Distribution and Hosts

Black root rot has been found in nurseries in most southern States as far west as Texas, but it occurs most commonly in southern Georgia and northern Florida. A somewhat similar disease has been reported from California.

All species of southern pines tested, including slash, loblolly, longleaf, pond, shortleaf, Virginia, and sand, are highly susceptible to black root rot. Spruce pine appears to be highly resistant.

Symptoms

The earliest symptoms of black root rot are detectable when the seedlings are about 2 months old. These consist of a slight enlargement and blackening of the distal portion of the taproot and larger laterals (fig. 4–1). By midsummer, the lower part of the taproot and most of the lateral roots may be dead. Numerous new lateral roots are usually formed above the dead portion of the taproot, and these may also be killed.

The above symptoms are characteristic of many root diseases. The distinctive symptom of black root rot is rough, dark enlarged areas on the taproot and larger laterals. Enlargements are usually observed about 1 month after the onset of symptoms, and first appear as small, reddish-brown areas which may remain localized or eventually may cover the entire taproot. They may reach two or more times the diameter of the normal taproot, are corky in texture, very rough, and eventually turn black. Few lateral roots remain alive on such affected portions of the taproot.

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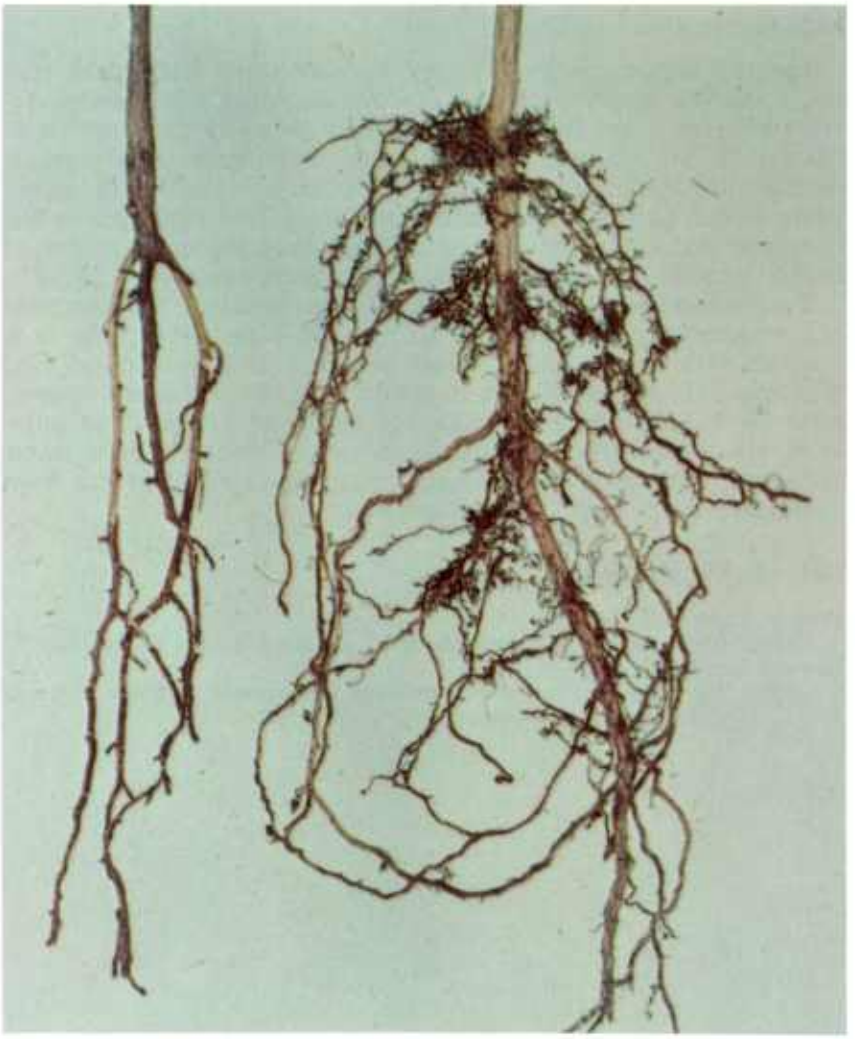

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Figure 4–1.—Roots of a healthy pine seedling (*right*) and roots damaged by black root rot.

The enlargements on the roots are due to the proliferation and enlargement of the cells of the phelloderm. The phellum of diseased roots is also many times thicker than on healthy roots, and this thickening accounts for the roughened appearance of the root surface.

Aboveground symptoms are not always correlated with root symptoms. Because the seedlings in the nursery grow under optimum conditions of moisture and nutrition, it is not unusual to find seedlings with advanced root symptoms that show no top symptoms. Chlorosis and stunting may occur in some instances, however.

Life Cycle and Control

The two organisms most commonly associated with black root rot, *Fusarium oxysporum* and *Sclerotium bataticola (Macrophomina phaseoli)*, are facultative saprophytes which can survive in the soil in the absence of their hosts. Both organisms produce thick-walled resting spores which can persist in the soil for many years under adverse conditions. Soil fumigation is therefore the recommended means of control. Methyl bromide or mixtures of methyl bromide and chloropicrin are the most effective fumigants.

Two nurseries where black root rot became severe the first year of operation were established on cleared pine sites. This fact, coupled with the finding of black root rot in a number of pine plantations in north Florida, indicates that the organisms responsible for the disease are indigenous to forest areas. Thus nurseries should not be established on newly cleared forest land. With one exception, all nurseries where black root rot has been found are located on light, sandy soil.

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5. PHYTOPHTHORA ROOT ROT

Phytophthora cinnamomi Rands

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In the past, Phytophthora root rot has caused losses in many species of coniferous and hardwood seedlings throughout the United States. In recent years, however, soil fumigation prior to planting has reduced the loss to this disease in most areas, except where seedlings are held in nursery beds for several years. The concern over the possible spread of this disease to new areas is apparent from some States' quarantine regulations forbidding intercounty shipment of infected stock.

Distribution and Hosts

Phytophthora cinnamomi has been isolated from soil and from forest and ornamental plants throughout the United States. It is most common in forest and agricultural lands in the southeastern United States and in the warmer agricultural lands and forest nurseries in western North America. Recent surveys indicate, however, that the fungus is present in only a few forest nurseries. It probably is absent from our western forests.

Over 100 species of plants have been reported as hosts for *P. cinnamomi.* In forest nurseries, major losses have been experienced in red pine, Fraser fir, black walnut, and yellow-poplar. The wide distribution and large host range make this fungus a potential threat in many nurseries.

Symptoms

Symptoms in nurseries often are not evident until seedlings start dying, since infection of the host plant commonly occurs through the feeder roots. Infected plants may remain symptomless until the tops wilt. They then rapidly turn a grey-green-brown and die. In some nursery beds this disease appears as enlarging circles of chlorotic and stunted seedlings, with the most severely affected seedlings at the center. In most hosts the disease is characterized by a red-brown discoloration of the cambial region, particularly at the root collar and sometimes extending several inches up the stem. In black walnut and chestnut, this discoloration is a blue-black "inky" color. The characteristic red-brown color is also present in the cambium and wood or roots, so that diseased seedlings can be identified prior to the appearance of top symptoms. In some hosts only root tips are killed, which results in chlorosis and stunting.

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Life Cycle

Chlamydospores or oospores formed in host tissue enable the fungus to survive for years in the soil. When fresh host material becomes available, these spores germinate and produce motile zoospores or infective mycelium. Most commonly, zoospores swim a few inches or are carried in ground water to the root tip. Infection occurs in the region of elongation or through wounds. Chlamydospores, oospores, and more zoospores are formed soon after infection. The former provides means of long-term survival whereas zoospores provide rapid spread along the root or to adjacent roots. Depending on the host species, the fungus may spread up through the roots to the root crown, killing the cambium and inner bark as it grows, or it may be limited to the root tips.

High soil moisture levels are necessary for zoospore formation and release. Soil temperatures above 15° C are necessary for infection and colonization of the host. These conditions are common in most nurseries.

Control

Fumigation of the soil prior to planting is the most certain control for Phytophthora root rot. If stock is only in the nursery for 1 year, soil fumigation with combinations of methyl bromide and chloropicrin will prevent this disease. When stock is held in a nursery for several years, extreme care should be taken to prevent introduction of this pathogen. Any stock or ornamentals brought into the nursery should be thoroughly checked and certified disease free. If the pathogen is present in a nursery, immediate fumigation of the infested area and a 2- to 3-foot surrounding buffer zone is necessary. Any effort to save apparently healthy stock in the infested area will result in the spread of the fungus to other areas of the nursery, as well as to outplanting areas. Particular care should be taken to treat border areas that may have been contaminated from soil carried on equipment. All equipment should be thoroughly cleaned before proceeding from the infested area. Soil drenches have not been effective in control of the disease, probably because chlamydospores and oospores are not affected by the drenches.

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6. PHYTOPHTHORA ROOT ROT OF BLACK WALNUT SEEDLINGS

Phytophthora sp.

RALPH J. GREEN, JR.*

Black walnut seedlings are highly susceptible to a root rot disease in the seedbed caused by fungi of the genus *Phytophthora*. Infection may occur at any time after seeds begin to germinate. Early infection reduces emergence and usually kills young seedlings. Older seedlings may survive, but the disease continues to develop in overwinter storage and may cause serious losses in the spring during handling and shipping.

Distribution

Phytophthora root rot occurs wherever black walnut seedlings are grown under nursery conditions. It is apparently limited to nursery seedbeds, however, and is not a recognized problem in natural regeneration.

Hosts and Damage

Although black walnut is highly susceptible to Phytophthora root rot, the disease is not confined to this host species. It also occurs on seedlings of the oaks, American and oriental chestnuts, and numerous other hardwood and coniferous species.

Infection may occur at any time after the seed splits prior to germination. If the germinating seed is invaded, the seedling is killed before it emerges above ground. Young seedlings that are infected usually collapse suddenly, blacken, and die in a few days. Older seedlings usually wilt and yellow before the plants are killed. These plants also turn black and the leaves hang on.

The root system of diseased plants shows extensive brown, water-soaked lesions (fig. 6–1) that begin at the root collar or at the point of attachment of a lateral root. The root rot progresses rapidly and may involve the lower stem as well as the root system.

Infected seedlings usually occur in spots or patches in the seedbed, which suggests that the fungus spreads from plant to plant after infection begins. Root rot is always more severe in low areas of the plant beds where drainage is poor.

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F–522497

Figure 6–1.—Symptoms of root rot of black walnut seedlings caused by *Phytophthora citricola.*

When walnut seedlings are lifted in the fall, many plants have dead or discolored lateral or fibrous roots and often show restricted discolored areas in the taproot cortex. After winter storage, these infected seedlings may develop root rot symptoms very rapidly during handling and shipping.

Symptoms on the oaks and chestnuts are similar to those on black walnut, except that infected seedlings turn brown or tan rather than black. Also, these species are apparently much less susceptible to this root rot than are walnut seedlings.

There is little evidence that this disease is a continuing problem after disease-free walnut seedlings are outplanted. However, seedling stock that is not carefully inspected and graded or that is improperly handled may be seriously reduced in vigor, and mortality may be high after transplanting.

Life History

Phytophthora root rot of black walnut seedlings and other tree species may be caused by any one of at least three different species of this fungus. These include *Phytophthora cinnamomi, P. cactorum* and, more recently, *P. citricola.* The symptoms produced and the general disease cycle for all three fungi are similar.

The causal fungi are soilborne, and persist indefinitely in soil on plant debris and other organic matter. They are considered weak parasites and are usually limited to very young seedlings or plants that have been damaged or weakened. These fungi are also favored by high soil moisture for active migration of their motile spores.

Experimental studies have shown that one or more of these fungi is present in virtually all nursery soils. Conditions favorable for infection include heavy soils, high water tables, or heavy rains which saturate the soil for periods of several days. The fibrous roots of seedlings are damaged or killed by lack of oxygen in saturated soils, and the root rot fungi rapidly colonize the injured root tissues. These fungi then grow into the sound roots and lower stems, where they kill young seedlings rapidly, or produce root infections in older seedlings. The disease is most common under cool temperatures, but soil temperature and other environmental factors are not so important in the infection process as high soil moisture.

Control

Attempts have been made to control this disease by soil fumigation and by various chemical treatments of soil and seed. Although root rot incidence may be somewhat reduced, there is no completely effective chemical control.

Walnut root rot can be controlled to some degree by avoiding areas of poor drainage. The nursery area should be tiled and well drained for effective water management.

Seedlings should be carefully inspected and graded when lifted in the fall for winter storage. Bulk storage in cold rooms should be avoided because these fungi grow rapidly at rather low temperatures. After overwinter storage, walnut seedlings should not be held at temperatures above 4° C for periods longer than necessary to process and ship the seedlings.

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7. CYLINDROCLADIUM ROOT ROT

Cylindrocladium scoparium Morgan, *C. floridanum* Sobers & Seymour

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Cylindrocladium root rot causes severe losses to both hardwood and conifer seedlings in forest tree nurseries. The problem has increased in the past few years due to internursery movement of infected stock. The damage was formerly attributed only to the fungus *Cylindrocladium scoparium*. However, a closely related species, *C. floridanum*, is also a part of this disease complex.

F-522499

Figure 7-1.—Cylindrocladium (*C. floridanum*) root rot damage (approximately 70 percent) to 1-0 yellow-poplar seedlings in a North Carolina forest tree nursery.

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Distribution

The disease is well distributed in nurseries throughout the eastern, southeastern, and Lake States regions. Although the disease is not reported in western nurseries, many western tree species are known to be susceptible to the fungus. The disease has also been reported from one forest nursery in Quebec.

Hosts and Damage

C. scoparium and *C. floridanum* have a wide host range. The disease caused by these fungi has been reported on plants in 66 genera of 31 families in many areas of the world. Susceptible conifer tree species include Austrian, jack, Mugo, red, Scotch, and white pines; black, blue, Norway, and white spruces; balsam and Fraser firs; and Douglas-fir. The most serious losses have occurred on red and white pines and black and white spruces. Yellow-poplar and black walnut are the most commonly infected and the most severely damaged hardwood species (fig. 7–1).

Cylindrocladium spp. cause several kinds of damage, including root rot, damping-off, needle and leaf blight, and stem cankers.

Root Rot

This is the most common symptom observed. Infected conifer roots become necrotic and discolored, and the cortex is easily removed in advanced stages of rot. The roots die from the tips back toward the root crown. New lateral roots may develop prolifically above the infected area, usually just below the root crown. Aboveground symptoms of conifer root rot are yellowing of needles as the seedlings enter periods of moisture stress. The foliage gradually becomes reddish brown as moisture loss progresses (fig. 7–2).

Roots of infected hardwood seedlings, such as yellow-poplar, develop a pronounced blackened condition and longitudinal cracking of the root cortex in contrast to the smooth white roots of healthy yellow-poplar seedlings (fig. 7–3).

Damping-Off

This disease occurs on newly emerging seedlings. Infected conifer stems above and below the ground become shrunken, dry, and turn brick red. In advanced stages, the seedling top can be lifted from the soil surface because the root system has been destroyed. Because other damping-off fungi cause similar symptoms, a laboratory diagnosis is necessary for positive identification of the pathogen.

Needle and Leaf Blight

On red pine, infection occurs at the base of needle fascicles during moist weather; new needles are the most susceptible. The

F-521562

Figure 7-2.—Cylindrocladium root rot on 2-1 red pine in a Wisconsin nursery.

F-522501

Figure 7-3.—*C. floridanum* root rot symptoms on a 1-0 yellow-poplar seedling. Note the blackened and longitudinally cracked cortex of the infected root.

infected needles turn yellow and develop brown lesions that enlarge, become sunken, and darken. The needles finally turn brick red and fall off. On white pine, infected needles yellow from their tips toward the base, and the brick red color is not as conspicuous. On black, Norway, and white spruces, the foliage turns yellow and the new growth may wilt.

Infected hardwood foliage shows necrotic leaf spots. Many hardwood seedlings that have severe leaf blight symptoms die.

Stem Cankers

Stem cankers have been most commonly observed on white pine seedlings, generally following the needle blight stage. They may occur randomly along the seedling stem, but usually originate at the base of needle fascicles. Brownish, sunken areas gradually enlarge, may girdle the stem, and kill the seedling.

Life History

The causal fungus overwinters in the infected roots or needles as thick-walled resting bodies called microsclerotia. Under favorable environmental conditions the microsclerotia germinate, and germ tubes invade the hypocotyls of germinating seeds or the roots of seedlings or transplants. Airborne conidia are produced

during the growing season on infected foliage. During hot, humid weather the conidia germinate and their germ tubes enter the needle stomates. This results in the needle blight stage. Resting bodies produced within and/or on infected needles, roots, and soil organic matter contribute to soil infestation when dead needles fall to the soil surface and when invaded roots die.

Control

The intensity of Cylindrocladium root rot can be significantly reduced by soil fumigation to a depth of at least 12 inches before seeding or transplanting. Use a soil fumigant containing 67 percent methyl bromide and 33 percent chloropicrin at a rate of at least 300 pounds of the product per acre. Cover the fumigated beds immediately with plastic tarping. DMTT (Mylone) at 200 to 250 pounds per acre has been used in several nurseries; however, control has not been quite as good as with methyl bromide. Because no tarping is used, a fumigant such as DMTT is effective only to the depth that it is mixed with the soil.

Bordeaux mixture has been used as a foliage spray to prevent needle blight of seedlings.

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8. CROWN GALL

Agrobacterium tumefaciens (E. F. Smith & Towns.) Conn.

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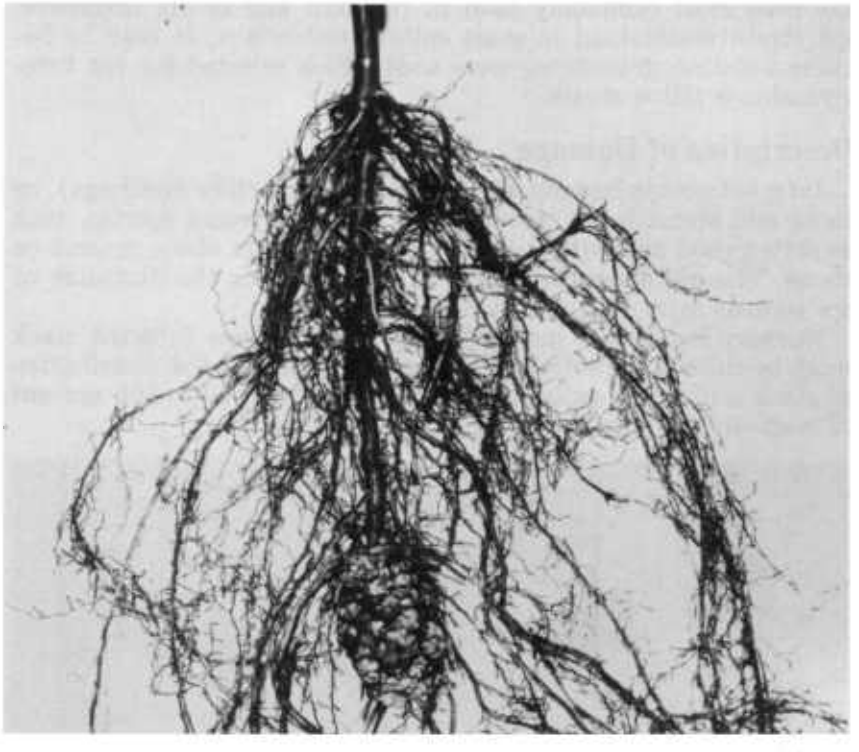
Crown gall, caused by the bacterium *Agrobacterium tumefaciens*, is an economically important disease in commercial nurseries, especially those growing numerous hardwood species. Although this disease has so far been of minor importance in forest tree nurseries, it warrants attention because of its potential for economic loss and the difficulty in controlling the disease.

Distribution and Hosts

Crown gall disease is widespread, occurring on this and other continents. A wide range of trees are susceptible, including both conifer and broadleaf species. Natural infection of species of



F-522503

Figure 8-1.—Crown gall that developed on eastern redcedar root following root pruning.

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Juniperus, *Cupressus*, and *Libocedrus* has been reported. There seems to be no report of infection of pines in nurseries. Several *Abies* species have been infected following artificial inoculation.

Two strains of *A. tumefaciens* have been isolated from naturally galled plants. The strains are most conveniently classified by whether they produce a ketoglycoside from lactose, but Keane et al. (1970) have listed a number of other biochemical properties which the strains do not have in common.

Recent work in Australia and Oregon has shown that the auxotrophic ketoglycoside-negative strain of the bacterium has been responsible for most infections occurring in those two geographic areas. The ketoglycoside-negative strain has been isolated from galled *Prunus* and *Rubus* species, willow, roses, apples, Norway maple, and soil from nurseries. Both ketoglycoside-negative and ketoglycoside-positive strains have been simultaneously isolated from naturally occurring galls on birch and mountain ash. This repeated isolation of the ketoglycoside-negative strain is curious inasmuch as the prototrophic ketoglycoside-positive strain has been most commonly used in research and is the predominant strain maintained in most culture collections. It may be because isolation procedures were used which selected for the ketoglycoside-positive strain.

Description of Damage

Infected plants usually develop galls (tumorlike swellings) on roots and stems below ground (fig. 8–1). On some species, such as cottonwood and willow, galls are also common above ground on stems. The galls may be a few to several times the diameter of the root or stem tissue on which they develop.

Nursery losses are incurred primarily because infected stock must be culled. Inspection agencies do not permit the distribution of stock containing galls. Such losses vary from 10 to 100 percent of a given nursery crop.

F–522504

Figure 8–2.—Natural infection of crown gall on nursery-grown mazzard cherry seedlings. Note common point of formation of galls.

The adverse influence of infection on some species grown for fruit or nuts has been established; however, little or no such information is available for susceptible forest trees. Early infection of young ornamental flowering cherry seedlings at critical points such as the crown can greatly reduce root numbers and stunt the tree (fig. 8–2).

Life History

Crown gall bacteria are apparently soilborne. They have been reported to persist in the soil from 1 to 2 years in the absence of a host. Because these bacteria enter plants through manmade or naturally occurring wounds, the common nursery practice of rootpruning encourages infection. Eastern redcedar in a central Nebraska nursery had galls develop at the point where roots had been pruned. Galls are commonly observed on the pruned end of taproots in hardwood nursery stock.

Natural wounds caused by chewing insects (such as grubs and wire worms), nematodes, and lateral root initiation have also been implicated as aiding infection by crown gall bacteria.

The crown gall bacteria are easily spread and dispersed by contaminated irrigation or runoff water that has passed through orchards containing galled trees. This source should be considered when watering stratified seed that is being planted in the spring.

Shipment of diseased planting stock obviously contributes to dispersal of the crown gall bacteria to other geographic areas. Although galled trees are usually detected easily, latent infections have been shown to occur in several *Prunus* species and in apples. Latent infections are difficult, if not impossible, to detect by visual inspection. The problem of recognizing latent infections in interstate shipment of nursery stock is compounded because of differing environmental conditions (especially cool vs. warm soil and air temperatures) from one geographic area to another which affect symptom expression.

Control

Fumigants have reportedly killed crown gall bacteria in host tissue in the soil when used at rates about twice that needed for nematode control. Control by fumigants has not been consistent, however. The disease was not controlled in beds in a Plains nursery which were fumigated with methyl bromide to control nematodes. In some cases, soil fumigation has resulted in increased infection.

Because the bacterium can reportedly overwinter in the soil in the absence of a susceptible host, beds containing stock with crown gall should be replanted with an immune or highly resistant species. Populations of crown gall bacteria probably are reduced by using nonsusceptible cover crops (grain crops) in rotations. However, the possibility exists that the bacteria could survive as saprophytes in the rhizosphere and rhizoplane of

nonsusceptible hosts. Several growers of ornamental *Prunus* species observed a relatively high incidence of crown gall when they replanted as long as 4 years later to fields in which a severe crown gall problem had previously occurred.

The effectiveness of preplanting treatments in preventing crown gall infection has been sporadic, perhaps due to the possibility of latent infections and subsequent inoculation and infection as a result of growth cracks and wounds sustained at a later period.

Because crown gall disease is highly contagious, good sanitary practices and pathogen-free planting stock are a must to reduce the incidence of disease. Tools and equipment, grading tables, and seed beds (if possible) should be surface sterilized. Nursery stock should be carefully handled, planted, cultivated, harvested, and stored to minimize wounding of susceptible tissue.

Irrigation water from deep wells should be free of crown gall bacteria, but water from irrigation lines or ditches that have passed through fields or plots with galled trees should be avoided. Similarly, any cultural practice that places healthy trees with galled trees in standing water is very risky.

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9. NEMATODES

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Nursery seedlings are particularly vulnerable to soilborne nematode diseases. Continuous cultivation of the same or similar plant species within restricted areas is conducive to the rapid buildup of parasitic nematode populations, and if not controlled, nematode diseases can result in significant losses. Such losses reduce the economic value of nursery crops of both conifers and hardwoods. Additional sales may also be lost because regulations restrict shipments of nematode-infected plants.

Distribution and Hosts

Plant-parasitic nematodes are found in forest nurseries throughout North America. Root-knot nematodes are not usually important parasites in areas with a cool climate. Conversely, certain species of lesion nematodes, serious in cooler regions, are rarely found in the southern United States.

Some parasitic nematodes have a wide host range and feed on many forest tree species; others are quite restrictive in their feeding habits. The pine cystoid nematode feeds only on certain conifers, and several species of root-knot nematodes infect certain hardwoods but not conifers. The nematodes most damaging to forest nursery crops are root-knot nematodes, *Meloidogyne* spp.; pine cystoid nematodes, *Meloidodera* spp.; lance nematodes *Hoplolaimus* spp.; lesion nematodes, *Pratylenchus* spp.; stunt nematodes, *Tylenchorhynchus* spp.; stubby-root nematodes, *Trichodorus* spp.; and dagger nematodes, *Xiphinema* spp.

Description of Damage

The reaction of plants to attack by plant-parasitic nematodes varies considerably with the host parasite combination. Nematode diseases ordinarily cannot be diagnosed by symptoms alone because other parasitic organisms or certain environmental factors can cause similar damage to roots. The aboveground symptoms of nematode diseases of forest nursery seedlings are generally similar and are usually the same as symptoms on any plant which has been deprived of a properly functioning root system. Diseased seedlings are stunted, lack vigor, and their

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leaves are reduced in size and number and become chlorotic; they often show symptoms of mineral deficiency, even when adequate levels of soil fertility are maintained. Affected plants usually cannot withstand long periods of low soil moisture.

Nurseries with nematode disease usually have patches of stunted seedlings, characterized by low vigor, having small, distorted root systems (fig. 9–1). These patches enlarge and coalesce as nematode populations increase and spread through the nursery beds.

The belowground symptoms appear as root impairment or distortion. Root-knot nematodes may cause galling of roots and in some cases a proliferation of roots near the galls. Dagger nematodes on certain hosts cause apical root-galling and distortion of lateral roots. Pine cystoid nematodes cause little, if any, galling and are apparent only when the swollen females erupt through the root epidermis. In contrast, lesion nematodes cause a necrosis of the cortex resulting in a general decay of the feeder roots. The lance nematode, an endoparasite particularly damaging to pine seedlings, also migrates through the cortical tissue and causes extensive internal cell destruction. This type of damage by internal parasites often allows fungus pathogens to enter and accentuate the general decay of the root system.

Nematodes that do not enter the roots generally have less obvious effects on forest tree seedlings. Stunt, stubby-root and dagger nematodes cause discoloration and surface lesions, but more often the effect of their feeding is a general stunting of lateral roots without noticeable decay (fig. 9–2).

F–522505

Figure 9–1.—Root lesion nematode damage to ponderosa pine seedlings, and control by methyl bromide fumigation.

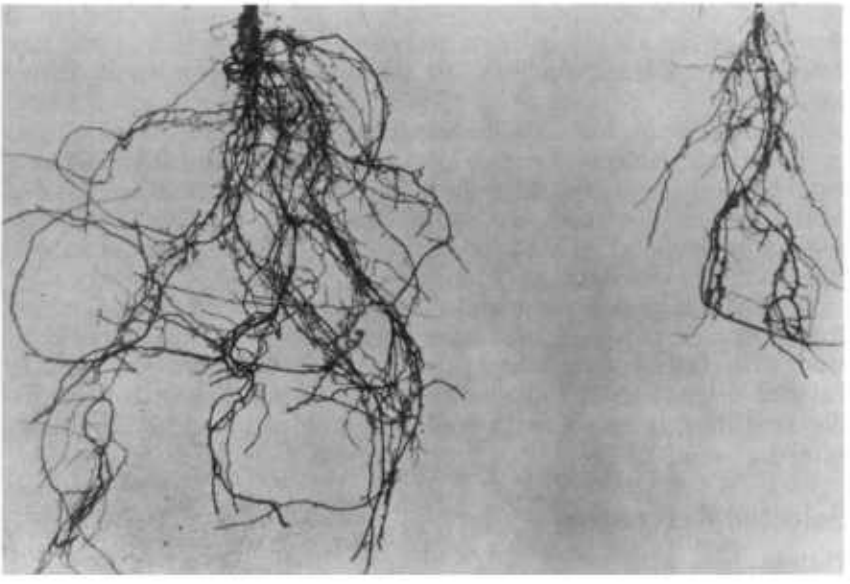

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Figure 9–2.—Comparison of healthy root system of sand pine seedling (left), and poorly developed root system caused by parasitic nematodes (right).

Nematodes often interact with fungi to produce disease complexes of tree seedlings. Certain rhizosphere fungi are destructive only when they occur in combination with nematodes parasitizing feeder roots. The damage caused by certain fungus pathogens capable of infecting feeder roots is often increased by joint action with plant-parasitic nematodes. Certain nematodes also prevent normal synthesis of mycorrhizal development on feeder roots by either directly attacking the fungus symbiont or damaging root tissue before mycorrhizal synthesis.

Many soilborne viruses are transmitted by nematodes. *Xiphinema* spp. and *Longidorus* spp. are known vectors of certain viruses that infect tree seedlings.

Control

Since nematodes cannot be eradicated in nursery soils even with broadcast fumigation, nematode control in nurseries is based on the concept of lowering nematode populations to levels below which yield is not affected.

Soil fumigation with methyl bromide at rates of 300–500 pounds per acre gives excellent control of nematodes in forest nurseries. Methyl bromide produces additional benefits by controlling insects, bacteria, fungi, and weeds and by increasing the availability of certain soil nutrients. Dowfume MC–33 and Brozone, general purpose fumigants containing methyl bromide,

also give excellent nematode control. When nematodes are a problem in small areas in a nursery, spot injection with less volatile nematicides, such as DD mixtures, will give satisfactory control.

Recently, bare roots of infected hardwoods have been dipped in chemicals to control root-knot nematodes. Organophosphate nematicides, such as Zenophos, have satisfactorily controlled nematodes on dogwood. Hot water treatment (52° C for 2 minutes to bare roots) gives good control of lesion nematodes infecting eastern redcedar.

Crop rotation is recommended for the control of certain nematodes. For example, if root-knot nematode is the problem, rotation with fescue (a nonhost) is recommended over soybean (a favorable host). Nonhost seedlings may be rotated with susceptible seedlings if space is limited and production must be maintained.

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10. MYCORRHIZAE OF FOREST NURSERY SEEDLINGS

DONALD H. MARX*

All species of trees growing under normal soil conditions in the forest are dual organisms—part plant and part root-inhabiting fungi. These highly specialized, root-inhabiting fungi are not pathogenic and, thus, do not cause root disease. They are beneficial to their tree hosts. They infect, symbiotically, the cortical tissues of young feeder roots of trees and form mycorrhizae. The word mycorrhizae means "fungus-roots." Mycorrhizae are the normal feeder roots of all forest trees, as they are for the vast majority of other plant species, including agronomic, horticultural, ornamental, and turf crops.

Classes of Mycorrhizae

The three classes of mycorrhizae are identified by the arrangement of the hyphae of the fungi within root cortical tissues.

Ectomycorrhizae.—This class occurs normally on roots of pine, spruce, fir, beech, birch, eucalyptus, alder, oaks, hickories, and certain other trees. Ectomycorrhizae usually can be distinguished from nonmycorrhizal roots because of their swollen appearance and in the case of *Pinus,* a forking habit (fig. 10–1).

F–522507

Figure 10–1.—Three different ectomycorrhizae of *Pinus taeda* seedlings. Note branching habits and color differences. Each type was formed by a different species of ectomycorrhizal fungus. Unbranched ectomycorrhizal roots on right segment are very similar to nonmycorrhizal roots.

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Not all forked feeder roots of pine are ectomycorrhizal, however, since feeder roots of many species can be stimulated to fork by many factors other than ectomycorrhizal fungus infection. The fungi which form ectomycorrhizae are among those that mainly produce mushrooms and puffballs periodically throughout the year. Over 2,400 species of ectomycorrhizal fungi are found in North America alone.

Under microscopic examination, hyphae of ectomycorrhizal fungi can be observed growing internally only around the cortical cells of the root forming the Hartig net, thus the prefix ecto. The Hartig net hyphal arrangement appears to replace the middle lamella, which is normally composed of pectins, and cements the cortical cells. Hyphae of the fungal symbionts that are present around the outside of the feeder root usually are tightly interwoven, and are designated the fungus mantle. The mantle of ectomycorrhizae may be only one or two hyphal diameters in thickness, or several dozen hyphal diameters. Ectomycorrhizae may be white, brown, yellow, black, blue, or blends of these colors. The color of ectomycorrhizae is apparently determined by the hyphal color of the specific mycorrhizal fungus encompassing the root.

Endomycorrhizae.—This class occurs normally on roots of maple, sycamore, ash, sweetgum, walnut, cypress, cedar, some poplar, and certain other hardwoods. Endomycorrhizae cannot be readily distinguished from nonmycorrhizal roots without the aid of a microscope. Endomycorrhizal infection does not normally cause color or physical changes in the root. The fungi which form endomycorrhizae are different from those of ectomycorrhizae and lack conspicuous aboveground fruiting bodies.

Most endomycorrhizal fungi of forest trees apparently belong to the genus *Endogone.* Dozens of species of endomycorrhizal fungi have been identified; undoubtedly many more exist. Under microscopic examination, hyphae of the endomycorrhizal fungi are observed growing into (thus, prefix endo) the cortical cells of the roots forming arbuscules. Arbuscules are specialized absorbing hyphae or haustoria of these fungal symbionts. Large vesicles, which are swollen hyphae, may also be seen in endomycorrhizal roots. If both vesicles and arbuscules are present, the endomycorrhizae are called "vesicular-arbuscular" (VA) mycorrhizae. The Hartig net and fungus mantle which characterize ectomycorrhizae are not present in endomycorrhizae. Endomycorrhizal fungi will frequently produce large, conspicuous, pearl-colored spores (either zygospores or chlamydospores) on hyphae attached to endomycorrhizal roots (fig. 10–2). The presence of these spores on root surfaces, which are detectable under low magnification, is a reasonably reliable indication of endomycorrhizal fungus infection of the root.

Ectendomycorrhizae.—This class is apparently an intermediate between the other two classes. These fungi grow into the

F-522508

Figure 10-2.—Spores of an endomycorrhizal fungus formed on endomycorrhizae of sweetgum. The diameter of these spores is approximately 0.004 inch. Note hyphae of the fungus radiating from the root surface.

cortical cells of the root with an appearance quite different from the arbuscular formation of endomycorrhizae. These fungi grow around the cortical cells in a Hartig net arrangement. They may or may not develop a fungus mantle over the feeder root surfaces. Ectendomycorrhizae have been observed on roots of certain species of tree seedlings in nurseries, especially in the Pacific Northwest and Northeast, but their significance is unknown. The species of fungi which form this class of mycorrhizae are also unknown at present.

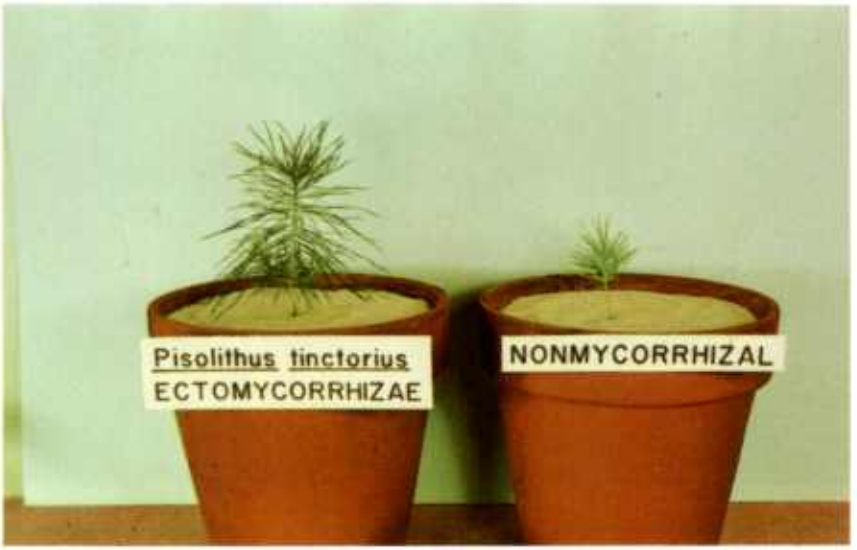
Benefits of Mycorrhizae to Trees

A considerable amount of research has been done on the benefits of ectomycorrhizae to trees (fig. 10-3). Only limited research has been done on the importance of endomycorrhizae to plant growth, and research on benefits of ectendomycorrhizae to trees is nearly nil. The following list of benefits of mycorrhizae to tree growth is based almost exclusively on work with ectomycorrhizae; however certain of these benefits are thought to be derived from endomycorrhizae as well.

1. Tremendous physical increase in absorbing surface of root system; includes both mycorrhizae and hyphae growing from mycorrhizae into soil.

2. More selective ion absorption and accumulation, especially phosphorus.

3. Solubilization of normally nonsoluble minerals and their constituents.



F–522509

Figure 10–3.—Growth differences between ectomycorrhizal and nonmycorrhizal Virginia pine seedlings. The 4-month-old seedlings were well fertilized and watered.

4. Increased longevity of feeder root function; mycorrhizal roots persist longer on root systems than do nonmycorrhizal roots.

5. Resistant to feeder root infections caused by pathogens, such as *Phytophthora* and *Pythium* spp., present in many forest and nursery soils. This benefit apparently applies only to ectomycorrhizae.

6. Increased tree tolerance to soil toxins (inorganic and organic), extremes of soil acidity, and high soil temperatures.

General Considerations

1. A given tree species may enter into mycorrhizal association with one or many different species of mycorrhizal fungi at a given time.

2. Some species of mycorrhizal fungi have very broad tree-host ranges, whereas, others have very narrow host ranges.

3. Some species of mycorrhizal fungi are more beneficial to tree survival and growth than others.

4. Some trees, in particular *Pinus*, have an obligate need for mycorrhizae in order to survive. This may not be true for all tree species, even though mycorrhizae occur normally on their roots.

5. Certain mycorrhizal fungi are more ecologically adapted to certain forest sites than are other fungi; trees with adapted fungal symbionts on roots grow better than trees with non-adapted fungal symbionts growing on the same site.

6. Spores of ectomycorrhizal fungi are produced above ground and are readily wind disseminated. Spores of endomycorrhizal fungi are produced underground and are not wind disseminated.

7. Many species of ectomycorrhizal fungi can be grown in pure culture or artificial media. Endomycorrhizal fungi cannot be grown in pure culture in the absence of their plant hosts.

8. Mycorrhizal fungi rarely exist in an active physiological state in soil in the absence of their hosts. However, they may remain in a dormant condition, as spores or resistant hyphae, in soil for many years without a tree host.

9. Not all species of mushrooms or puffballs are ectomycorrhizal. Many are saprophytic in nature and decompose organic matter.

Practical Considerations

Soil fumigation is becoming more and more a routine practice in tree nurseries to control weeds, nematodes, and fungal root pathogens. Most soil fumigants also eradicate the beneficial mycorrhizal fungi. The eradication of ectomycorrhizal fungi from nursery soil is usually not a problem since these fungi produce wind-disseminated spores periodically throughout the year which recolonize the soil. One of the major ectomycorrhizal fungi to colonize fumigated nursery soils, especially in the south, is *Thelephora terrestris* (fig. 10–4).

F–522510

Figure 10–4.—Basidiocarp of *Thelephora terrestris* formed around stem of a pine seedling. This fungus is ectomycorrhizal with and beneficial to numerous pine and fir species. Its epiphytic habit on stems does not damage seedlings.

Deficiencies of ectomycorrhizal fungi in previously fumigated nursery soils have been reported. Colonization of soil from airborne spores did not occur in these instances. Deficiencies could be due to unfavorable weather conditions for mushroom production in forests adjacent to the nursery. Spores from these mushrooms serve as inoculum for soil colonization. Also, the nursery may be too far from forests harboring specific mushrooms. Research is currently underway to determine the feasibility and practical value of artificially infesting nursery soils with specific ectomycorrhizal fungi known to be more beneficial to tree survival and growth than those ectomycorrhizal fungi which naturally occur on roots from airborne inoculum.

The consequence of soil fumigation to endomycorrhizal fungi is another problem. Once these symbiotic fungi have been eradicated from soil, reinfestation is very slow because their spores are not normally wind disseminated. Therefore, deficiencies of these fungi in nursery soil following fumigation are not unusual. Endomycorrhizal development following successful soil fumigation is from inoculum of the symbionts (1) still viable in soil depths beyond effective fumigant penetration, (2) washed in from water runoff or heavy rain splash from nonfumigated areas of soil, (3) brought in by nonfumigated soil on cultivation equipment, or (4) possibly from windblown nonfumigated soil. Each of these possibilities would result in a slow development of endomycorrhizae.

Considerable research has demonstrated that ectomycorrhizae are not only beneficial to growth of trees, but are actually indispensable for survival and growth of transplant stock, in particular *Pinus* spp. Thus, to insure survival and good growth of normally ectomycorrhizal trees, seedlings used in reforestation should have abundant ectomycorrhizae. It is not known if this statement can be applied to endomycorrhizae. The significance of endomycorrhizae to survival and growth of several species of hardwood seedlings is currently being investigated.

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II. STEM AND BRANCH DISEASES (CANKERS)

The diseases of stems and branches are known as cankers, diebacks, or galls, depending upon the symptoms they cause on the host. Cankers are sunken areas of dead tissue in the bark, while diebacks are the dying and/or curling of the shoot tip. Galls are local enlarged or swollen portions of the branch or stem. The causal fungi which may often fruit on the canker or gall are helpful in diagnosing these diseases. Stem and branch diseases are easily noticed in the beds when associated, as they often are, with dead foliage. Without the dead foliage they may go unnoticed until the grading and sorting operation. The following eight diseases are the most important and better-known stem and branch diseases of forest tree seedlings.

11. PHOMOPSIS CANKER OF DOUGLAS-FIR

Diaporthe lokoyae Funk, *Phomopsis lokoyae* Hahn

RICHARD S. SMITH, JR.

Phomopsis canker caused by *Diaporthe lokoyae* (imperfect stage = *Phomopsis lokoyae*) is one of the most frequently encountered cankers of Douglas-fir in western North America. It is a native disease which is most severe in the seedling and sapling stages in natural stands, plantations, and forest nurseries.

Until recently only the imperfect stage, *Phomopsis lokoyae*, of this fungus was known, and the disease was named after this stage. In 1968, Funk described the perfect stage *Diaporthe lokoyae* of this pathogen. Both stages of the fungus may occur on the same specimen, but the fruiting bodies do not intermingle. The perfect stage is usually on the distal portion of the killed twig.

Distribution

Phomopsis canker was first described in 1933 from northern California. It has since been found in the forests of Oregon, Washington, and British Columbia, and has been reported as a problem in some nurseries in these areas.

Hosts and Damage

Both the Pacific Coast and Rocky Mountain forms of Douglas-fir are susceptible to the disease. Western hemlock and western redcedar have been reported as rare hosts.

The disease causes cankers on the stems and branches. The foliage and branch distal to the infection then yellows and dies quickly (fig. 11–1). The disease kills some trees and causes others to be forked and misshaped. The major loss results from the culling of misshapen stock.

Life History

The sunken appearance of the canker that develops on young stems and branches is due to continued diameter growth of neighboring uninfected tissue while infected tissue has died. Small, black, spherical fruiting bodies (pycnidia) develop in the

F-522511

Figure 11-1.—Douglas-fir seedling infected by *Diaporthe lokoyae*.

cankered areas. These pycnidia produce spores which are water-borne, and spread to new hosts by the splashing drops of rain or sprinkler irrigation. Under favorable conditions the spores germinate and infect the new hosts. The perfect stage may develop later on the infected branch. This stage releases a wind-borne spore which is responsible for the long-distance spread of the fungus.

In natural stands the intensity of infection of Phomopsis canker varies greatly from year to year. Infection appears to be more frequent when protracted rains are followed by periods of drought. The intensity of this disease also fluctuates in nurseries from year to year, for reasons not yet explained.

Control

Periodic spraying with protective fungicides throughout the growing season has effectively controlled this disease.

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12. SIROCOCCUS TIP BLIGHT

Sirococcus strobilinus Preuss.

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Sirococcus tip blight is a disease affecting chiefly the new shoots of conifers. It is caused by a fungus producing only water-borne (spread) spores, and is also referred to by the name *Ascochyta piniperda.*

Distribution

This disease occurs in both forests and forest nurseries in Europe, and in eastern North America. Recently it has been found in natural reproduction in the Lakes States, Alaska, and British Columbia. In California, it is restricted to the north coastal fog belt.

Hosts and Damage

S. strobilinus has been reported in the United States and Canada on seedlings of Norway and red spruces, Coulter, Jeffrey, sugar, red, lodgepole, and ponderosa pines, and western hemlock.

The disease causes a tip dieback and stem and branch cankers in the current year's growth. The foliage distal to the infection becomes yellow and dies. This disease kills trees with multiple infections, and causes others to become forked and misshapen. Nursery losses are due both to tree death and to culling of misshapen stock.

Life History

Initial infection occurs in the juvenile needles. The fungus spreads down into the succulent stem tissues, where it causes a small purplish canker (fig. 12–1). The infection enlarges longitudinally more rapidly than around the stem, producing an elongate, sunken canker. A small drop of resin is often exuded

F–522512

Figure 12–1.—A Coulter pine seedling infected with *Sirococcus strobilinus*. Curling of the top is typical of this disease.

in the center of the canker at the needle base from which stem infection originated. When infection occurs in the region of elongation, the restricted growth in the cankered area causes the shoot tip to curl over and form a crook (fig. 12–2). As the canker matures, small, black fruiting bodies begin to appear on the infected needles and the older portions of the canker (fig. 12–3). These fruiting bodies produce large masses of spores which are spread to new hosts by the splashing of water drops from rain or sprinkler irrigation. Under favorable conditions of high humidity and mild temperatures (above 10° C), the spores germinate and infect the new host.

F–522513

Figure 12–2.—A young canker caused by *Sirococcus strobilinus* developing at the base of an infected juvenile needle of a 2-year-old Jeffrey pine.

F–522514

Figure 12–3.—The small, black fruiting bodies of *Sirococcus strobilinus* developing in an older cankered area.

Control

Periodic spraying with protective fungicides throughout the growing season will effectively control this disease. Chlorothalonil (Bravo W–75 and 6F) has successfully controlled this disease in West coast conifers.

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13. GREY MOLD OF GIANT SEQUOIA

Botrytis cinerea (Fr.) Pers.

RICHARD S. SMITH, JR.

Grey mold—a disease of many plants, including forest tree seedlings—is caused by the fungus *Botrytis cinerea;* its perfect stage is *Sclerotinia fuckeliana* (deBy) Fckl. This fungus, also a saprophyte, is almost universally present on dead and dying vegetable matter. As a plant pathogen, the fungus usually must first become established on dead or moribund parts of a host

F-522515

Figure 13-1.—A 1-year-old giant sequoia with a stem canker caused by *Botrytis cinerea.*

plant. It then can spread into adjacent healthy tissues. A few species of conifers, particularly giant sequoia, are so susceptible to *B. cinerea* that the fungus can successfully infect healthy green tissues.

Distribution

Since the fungus is found in all temperate regions, the presence of the disease is dependent only on having a susceptible host under suitable conditions. It has been reported on various forest species from Asia, Europe, and North America.

Hosts and Damage

This pathogen has an extremely large host range. Nearly all forest trees are susceptible to some degree, but it is a serious problem only on a few species such as redwood, giant sequoia, and Monterey and Italian cypress.

The disease causes a branch dieback and canker of green succulent tissues. In 1-year-old seedlings, stem cankers originating from infected lower branches frequently girdle the stem (fig. 13–1) and kill the entire seedling. In California, when conditions are suitable, whole sections of nursery beds of giant sequoia have been destroyed.

Life History

Under prolonged periods of high humidity and cool temperatures, this fungus infects the juvenile bracts and young branches, particularly the lower shaded branches. Once established, it proceeds downward into the stem, killing the tissues it infects. In the succulent 1-year-old stem tissues, it forms a black sunken canker which eventually girdles the stem. The portion of the seedling distal to the girdle dies. Since the lower shaded branches are infected more often than the upper branches, the girdles are usually low on the stem in a position where most, or all, of the crown is killed. This disease is favored by cultural practices such as high planting densities or lath shading, which limit air movement and raise the humidity around the seedling.

Under moist conditions, *B. cinerea* can be seen as a thin grey web of mycelium on infected plant parts. From this mycelium and from infected tissue, tufts of black conidiophores arise bearing clusters of white to grey spores. When the conidiophores are brushed, clouds of spores are released.

Control

Proper cultural practices which increase aeration and decrease humidity will help reduce or prevent losses in most species. In highly susceptible species, fungicidal sprays may be required.

Most fungicides act as protectants; they do not cure the disease once it is in the plant. Therefore, fungicides must be applied at the first sign of the disease, and their use continued as long as conditions favorable to the disease persist.

Grey mold is also a serious problem during the cold storage and transportation of forest seedlings. Whole shipments of seedlings have been lost to this disease. To reduce chances of losses, all seedlings infected with grey mold must be culled out at the time of sorting and packaging. The seedlings should be protected further by a fungicide spray or dip just before packaging.

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14. SCLERODERRIS CANKER

Scleroderris lagerbergii Gremmen

DARROLL D. SKILLING

Scleroderris canker kills both seedlings and plantation trees. Mortality is most rapid among nursery seedlings; stems are killed by girdling. Planting infected seedlings is especially serious, as the infected trees provide inoculum for future disease buildup.

It is not known if Scleroderris canker is native to the United States, but it has been present for many years. The fungus responsible for this disease was not identified until 1964 in the United States. It is currently causing heavy losses in red and jack pine plantations in northern Wisconsin and Michigan.

Distribution

The disease is known to be present in Michigan, Wisconsin, Minnesota, Vermont, and New York. It is also widely distributed throughout Ontario and Quebec, and occurs in New Brunswick and Nova Scotia. Short growing seasons, with attendant prolonged damp springs and autumns, and heavy snowfall seem to favor disease development.

Hosts and Damage

Scleroderris canker has been found on Scotch, red, and jack pines in the United States. In addition, white and lodgepole pines and white and black spruces have been infected in Quebec. Austrian, Corsican, Swiss Mountain, Swiss stone, and Maritime pines are known hosts in Europe, as well as Norway spruce and Douglas-fir, and Todo fir in Japan.

Damage to pines includes death of lateral and terminal branches. Infection is usually limited to the lower 6 feet of the tree. The fungus commonly grows into the main stem from infected branches, resulting in a stem canker. A characteristic yellow-green discoloration is frequently observed in the cambial zone of recently killed tissue. Infected needles on red pine nursery stock are orange at their base during early May. These needles are loose and can easily be removed from the seedling (fig. 14–1).

In Quebec the symptoms of Scleroderris infection on black spruce include dead or dying stems, leaders, and branches.

F–522516

Figure 14–1.—Red pine seedlings (3–0) infected with *Scleroderris lagerbergii*. The orange discoloration at the base of the needles is obvious during early spring in North America.

Life History

Primary infection is by windblown ascospores disseminated during moist weather from May to October, although most are released during June and July. Infection is through buds or needles. Infected branch tips are usually dead the following summer. The fungus grows down the branch until it reaches the main stem of the tree. On trees less than 4 years old the stem is quickly girdled, and the tree is dead within a few months.

A few months after a branch dies, pycnidia appear near the base of dead needle fascicles. Asexual spores ooze out of these pycnidia during wet weather. These spores are transported by rain splash to nearby branches, which may become infected. Apothecial fruiting bodies appear in early summer on branches that have been dead for 1 or 2 years. The apothecia are also usually found at the base of dead needle fascicles, and are often closely associated with the pycnidial stage. Ascospores become mature in the apothecia by late May, and are discharged following rainfall from then until October.

Control

Infection by *S. lagerbergii* can be prevented in nursery beds by applying approximately seven sprays of chlorothalonil (Bravo W–75 and 6F) at 3 pounds of 75 percent wettable powder or 1½ quarts of 6-pound a.i. liquid formulation per 100

gallons of water. Applications should begin as soon as new shoot growth starts. They should be repeated at 2-week intervals until the first of July, then at 4-week intervals until the first of September. One or two extra sprays may be needed if rainfall is unusually heavy during June.

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15. SOUTHERN FUSIFORM RUST

Cronartium fusiforme Hedgc. & Hunt Ex Cumm.

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Fusiform rust is the most serious nursery disease of slash and loblolly pines in the southern United States. Without fungicidal control, this disease may affect up to 90 percent of all slash and loblolly pine seedlings in a given nursery. Although fungicidal sprays are routinely used in southern nurseries, in most years up to 20 percent of the slash and loblolly pine nursery stock is infected in high-hazard sites by this pathogen. Because infected nursery seedlings rarely survive the first year of outplanting, nursery infection is essentially equivalent to seeding mortality.

F-522517

Figure 15-1.—Typical galls on loblolly pine seedlings caused by *Cronartium fusiforme.*

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Distribution

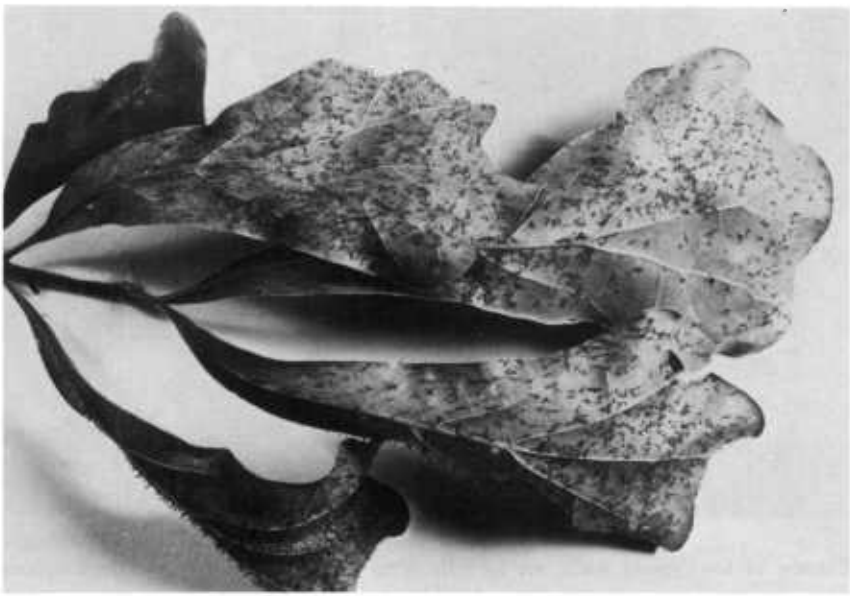
Cronartium fusiforme is indigenous to the southern United States from Maryland to Florida and westward to Arkansas and Texas. The incidence of the disease is highest in a zone approximately 150 miles wide extending from coastal South Carolina to Texas.

Hosts and Damage

Natural and artificial inoculations have proved 32 species of pines to be susceptible to the disease. Slash, loblolly, and South Florida slash pines are highly susceptible, longleaf and pond pines are intermediate, and pitch and shortleaf pines are resistant.

Natural and artificial inoculations have also proved 33 species of oak to be susceptible to the disease. Four species of chestnut, two species of chinkapin, and tanoak have also been shown to be susceptible, but members of the black oak group are the most common alternate hosts of the fungus.

Typically spindle-shaped stem and branch galls result from infections of pine (fig. 15–1). Seedlings infected in the early stages of growth often die when outplanted whereas others develop bushy crowns because of the loss of apical dominance. The reduced mechanical strength of infected stem tissue of older



F–522518

Figure 15–2.—Telia on the underside of water oak leaves caused by *Cronartium fusiforme*.

pines results in increased wind breakage. Infections of the alternate (oak) hosts generally cause minor leafspot injury; in unusually heavy infections, there may be some defoliation.

Life History

Aeciospores, produced on pine galls in early spring, infect expanding oak leaves. Uredial pustules develop rapidly on infected oak leaves and produce uredospores which may infect other young leaves. As these infected leaves mature, telia appear and produce basidiospores which infect new pine needles and shoots (fig. 15–2). The basidiospores are produced from spring to early summer during periods of high humidity and moderate temperatures. On nonhost tissue, the basidiospores may form secondary spores which eventually can germinate directly on pine tissue and cause infection.

Control

Fusiform rust infections can be prevented by spray applications of ferbam or ziram. Ferbam is the most commonly used fungicide. A spreader-sticker is usually added to improve coverage and reduce weathering. The frequency and coverage of fungicidal sprays is important for effective control. During the infection season, sprays are usually applied twice each week and after rains. Sprays applied before hazardous rust weather is forecast are more effective than those applied after such periods. All infected seedlings should be culled and only healthy seedlings outplanted.

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16. WESTERN GALL RUST

Peridermium harknessii J. P. Moore

RICHARD S. SMITH, JR.

Western gall rust, although often found in the forests of western North America, has been only an occasional problem in forest nurseries. Although not generally a problem to nursery production of pines, western gall rust can have serious consequences when outplanted. Not only are infected trees doomed, but the rust could easily spread from the seedlings to the surrounding forest.

Distribution

This disease is found generally throughout the pine forests of western North America, and in scattered locations in eastern United States and Canada. In western forest nurseries the infection has been limited to a few nurseries with a natural source of inoculum nearby.

Hosts and Damage

The western gall rusts caused by the fungus *Peridermium harknessii* (sometimes called *Endocronartium harknessii*), attacks a large number of native hard pines, including knobcone, jack, lodgepole, Coulter, Jeffrey, bishop, ponderosa, Monterey, and Digger pines as well as the exotic Canary Island, Aleppo, Swiss Mountain and Scotch pines.

This disease causes globose galls or swellings at the site of infection (fig. 16–1). Gall rust may kill the entire seedling, or just that portion distal to the infection. The seedling may live and grow, but as the gall enlarges, the stem becomes misshapened and weakened (fig. 16–2). Excess branching and loss of apical dominance may result in "brooming" of the tree at the site of infection. Such damage is not usually seen in the nursery, but develops after the seedling is outplanted. Nevertheless, damage is all directly attributable to the disease contracted in the nursery; hence, all gall-bearing seedlings should be culled.

F-522519

Figure 16-1.—A lodgepole pine seedling infected with western gall rust shows a globose gall and the orange spore masses in the bark cracks.

F-522520

Figure 16-2.—Lodgepole pine seedlings infected with western gall rust.

Life History

Because it takes 2 to 4 years before an infection starts producing spores, the source of infecting spores is usually outside the nursery beds. The source may be infected pines in ornamental plantings, in windbreaks, or in surrounding pine forests.

In spring and early summer, orange-colored aeciospores are produced on infected trees and dispersed by wind. Under the right temperature and moisture conditions, the spores deposited on young shoots of a susceptible pine germinate and infect the seedling. Either late the same year or during the next growing season, a globose gall or swelling begins to develop at the site of infection. In spring or early summer of second or third year, orange-colored masses of aeciospores are produced on these galls coloring them a distinctive bright orange. The aeciospores are formed within thin white sacs underneath and between the bark scales. When the sacs mature, they rupture and release aeciospores into the wind.

Control

The most effective and economical method of control is to remove the source of infecting spores. All gall-bearing pines within a half mile of the nursery should either be pruned free of all galls or felled. These sanitation measures have reduced the amount of western gall rust in the nurseries in which the disease was a problem.

All gall-bearing seedlings should be destroyed. Because there are many latent infections with this disease, it is also desirable to check all plantations in which stock had been exposed to infection. Also, care should be exercised in the movement of hard pine nursery stock from presently infested areas into those areas which are presently free of this disease.

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17. JACK PINE RUSTS
Cronartium sp., *Peridermium* sp.

DARROLL D. SKILLING

Jack pine seedlings are attacked by a number of stem rust fungi that frequently cause much mortality. These fungi include *Cronartium quercuum,* eastern gall rust; *C. comptoniae,* sweetfern rust, *C. comandrae,* comandra rust; *C. coleosporioides,* stalactiform rust; and *Peridermium harknessii,* western gall rust. With the exception of *P. harknessii,* which is discussed in article 16 of this Handbook, all of these rust fungi require an alternate host to complete their life cycle. Seedling losses often occur where an alternate host of these rusts is present near jack pine nurseries.

Distribution

Eastern gall rust is found east of the Great Plains from Canada to southern United States. Sweetfern rust is transcontinental in distribution from Nova Scotia west to British Columbia and Alaska, in the eastern United States south to North Carolina, and in the western United States in Washington and Oregon. Comandra rust occurs from New Brunswick to the

F-522521

Figure 17-1.—Jack pine stem rusts on seedlings: Left to right, eastern gall, comandra, sweetfern.

Yukon and British Columbia in Canada and southward to California, New Mexico, and Alabama in the United States. It is present in all States west of the Rocky Mountains. Stalactiform rust is present from New Brunswick to British Columbia and south to California, central Colorado, Minnesota, and Michigan.

Hosts and Damage

Disease	*Aecial Host*	*Uredial and Telial Host*
Eastern gall rust	Jack, Scotch, shortleaf, Virginia, and other hard pines	Various species of oaks
Sweetfern rust	Jack, Scotch, lodgepole, ponderosa, pitch, and other hard pines	Sweetfern and sweetgale
Comandra rust	Jack, Scotch, lodgepole, ponderosa, loblolly, shortleaf, and other pines	Bastard toad flax and northern comandra
Stalactiform rust	Jack, lodgepole, Jeffrey and ponderosa pines	Cow-wheat, Indian paintbrush, lousewort, and yellow-rattle

Eastern gall rust produces globose-shaped galls on the stems of young pine seedlings. In problem areas, nursery losses frequently exceed 25 percent. Infected seedlings that are outplanted are subject to wind breakage in later years, because the galls greatly weaken the stem, or result in considerable cull.

Sweetfern rust usually is evident as an elongate swelling on the stems of infected seedlings (fig. 17–1). This swelling appears 1 to 2 years following infection. The disease is most conspicuous in early spring when orange-yellow aeciospores are produced on the gall. Nursery seedlings grown near alternate host plants are often heavily damaged by the girdling action of this rust. In one Michigan nursery, sweetfern rust destroyed nearly all the lodgepole and ponderosa pine seedlings.

The first indication of comandra rust on seedlings is a spindle-shaped swelling of the bark near the base of the stem. Cankers originate on needle-bearing twigs and stems. Cankered bark, on which aecia are produced, is cracked and pitted. In time branches and stems are girdled. Mortality, however, is usually not excessive.

The spindle-shaped cankers on seedlings infected with stalactiform rust are not easily distinguishable from those produced by sweetfern and comandra rusts. Elongate diamond-shaped cankers are produced on mature trees. Ponderosa pine nursery stock has been severely damaged by stalactiform rust in Montana.

Life History

The biological development of the four rusts reported here is similar. All require an alternate host to complete their life cycle. In each case this is a broadleaf plant entirely unrelated

to the pine host. A typical life cycle starts with a basidiospore from the alternate host germinating on a pine needle. The rust fungus penetrates the needle and grows down into the stem of the seedling. During the summer, 1 to 3 years after infection, drops of a thick, somewhat sticky and reddish-orange liquid begin to exude from the diseased bark. These drops contain pycniospores that probably function in the sexual phase of reproduction of the fungus. Approximately 1 year later, orange-colored aeciospores are produced in the same area as the pycniospores.

The wind-disseminated aeciospores can infect the alternate host under proper temperature and moisture conditions. About 2 weeks after the alternate host is infected, orange-colored urediospores are produced on the undersurface of the leaves. These urediospores can infect additional alternate host plants, increasing the disease inoculum in the area. In late summer or early fall, brownish, hairlike structures called telia are produced on the infected leaves. In the case of eastern gall rust this may occur in early spring. Teliospores germinate under cool, moist conditions and produce windblown basidiospores which again infect the pine host.

The two critical periods in the development of rust epidemics are the period of aeciospore dissemination to the alternate host, and the period when basidiospores are formed and infect the pine seedlings. Long periods of moist weather during these spore discharge periods are conducive to severe infection.

Control

Control measures may be used to reduce jack pine stem rust losses. The weak link in the life cycle of these rusts is their need to live for a time on a broadleaf host. Nurserymen can take advantage of this weakness by removing the broadleaf hosts from the nursery area whenever practical. The exact distance necessary to eradicate alternate hosts is not known, but is probably from ¼ to 1 mile.

Infected seedlings should be eliminated at the nursery. Planting infected seedlings may allow the fungus to spread to the alternate host and back to other pines in the plantation on nearby forests, creating an epidemic.

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18. WHITE PINE BLISTER RUST

Cronartium ribicola J. C. Fisher

DARROLL D. SKILLING

White pine blister rust, caused by the fungus *Cronartium ribicola,* was introduced into North America from Europe about 1900. Economically, it is one of the most important forest diseases in the United States and Canada; it causes an annual growth loss and mortality in excess of 200 million cubic feet. In many areas of North America where conditions for blister rust development are favorable, it is responsible for the virtual abandonment of susceptible pine species from forest planting programs.

Distribution

White pine blister rust is distributed throughout most of the range of white pines in North America. In Canada the disease is present in British Columbia, Ontario, Quebec, and portions of Newfoundland. In the western United States it is present throughout Washington, Oregon, northern California, northern Idaho, Montana, and Wyoming. In the eastern United States it is present in the New England States west to Minnesota and south into the highlands of North Carolina.

Hosts and Damage

The fungus responsible for the blister rust disease spends part of its life cycle on the five-needled pines and the remainder on plants in the genus *Ribes* (currants and gooseberries).

The most susceptible pines are western white, sugar, limber, and white-barked pines. Eastern white pine and foxtail pine are moderately susceptible. Swiss stone pine and Balkan pine, both native to Europe and Asia, are quite resistant. Pinyon pine and Oriental white pine are not susceptible to the blister rust fungus.

All of the native North American *Ribes* are susceptible to blister rust. The European black currant, *Ribes nigrum,* is highly susceptible. In the white pine regions of Idaho, *R. petiolare* and *R. inerme* are most susceptible; *R. viscosissium* is moderately susceptible and *R. lacustre* is the least susceptible.

White pine blister rust is evident as an elongate swelling on the stems of infected nursery seedlings. This swelling frequently becomes yellow orange to copper 1 year after infection. The disease is most conspicuous in early spring when orange-yellow aeciospores are produced on the swollen area. White pine seedlings grown near large concentrations of *Ribes* plants are often heavily infected by *C. ribicola*. Infected stock is frequently shipped to the field because new infections are difficult to recognize.

Blister rust kills white pines of all ages, with the smaller trees dying more rapidly. The fungus grows directly from infected needles into the main stem of nursery seedlings. Stem girdling kills the seedlings. Branches girdled by the fungus on older trees develop brown needles called "flags" which can be easily recognized. The fungus frequently grows into the main stem from these infected branches, resulting in stem cankers. These cankers may persist for years on larger trees, and eventually kill the tree by girdling the stem.

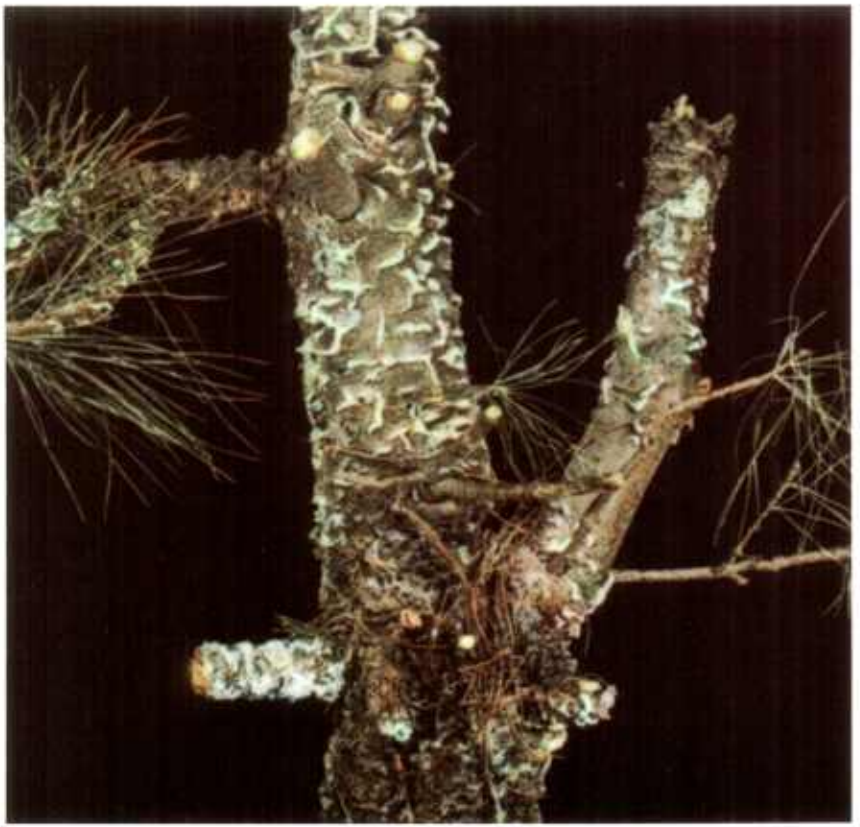

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Figure 18-1.—White pine blister rust canker on young pine. The cream-colored blisters contain the aeciospores that infect *Ribes*.

Life History

The life cycle starts with a basidiospore from an infected *Ribes* plant germinating on a pine needle during late summer or early fall. The rust fungus penetrates into the needle and grows down into the stem of the seedling. This mycelium continues to advance as long as the host remains alive. During the early summer, 2 to 4 years after infection, yellow-brown blisters appear on the bark and drops of a sticky reddish-orange liquid ooze out. These drops contain pycniospores that probably function in the sexual phase of reproduction of the fungus. Approximately 1 year later, aeciospores are produced in the same areas as the pycniospores. Cream-colored blisters appear which burst and release the orange-colored aeciospores (fig. 18–1). The aeciospores are carried by the wind, sometimes for several hundred miles, to infect *Ribes* leaves.

About 2 weeks after infection, orange-colored urediospores are produced on the undersurface of the *Ribes* leaves. These urediospores can infect additional *Ribes* leaves, which increases the disease inoculum in the area. Up to seven generations of urediospores may be formed in a season. In late summer or early fall, brownish, hairlike structures called telia are produced on the infected leaves. Teliospores are produced on the telial columns. These germinate and produce basidiospores, which are blown to the needles of pine to complete the life cycle.

Moisture and cool temperatures (15° C is optimum for spore germination) favor infection of both hosts. Basidiospores are produced only when the relative humidity is high, especially after rains and during fogs. The basidiospores are quite delicate, and if exposed to dry air or sunlight they die quickly. The effective range over which pines can be infected by basidiospores is usually a few hundred feet. However, under ideal conditions of cool, moist weather, it is possible for basidiospores to infect pines over a mile away.

Control

White pine blister rust can be controlled by removing the *Ribes* bushes within ¼ to 1 mile of the nursery. This method of control is possible because the fungus cannot spread from pine to pine, but must live for a time on the *Ribes* plant to complete its life cycle. When possible, white pine seedlings should be grown only in nurseries where the environment is unfavorable for blister rust infection because of high temperatures and low humidity.

Infected seedlings should be culled at the nursery whenever disease symptoms appear. The planting of infected seedlings may allow the fungus to spread to *Ribes* plants and back to other pines in the plantation, creating an epidemic.

The most promising control for blister rust is the selection and breeding of resistant trees. Substantial progress has been

made in this area in the last 10 years. Seed orchards are now being established with trees that have shown resistance to *C. ribicola.*

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III. FOLIAGE DISEASES

Foliage diseases attack leaves, needles, and young shoots. They are called leaf spots when only a portion of the leaf is affected, and leaf and needle blights when the whole leaf or needle is killed. Anthracnoses are foliage diseases caused by a particular group of fungi in which both the leaves and young developing shoots are killed. In general, foliage diseases are more easily recognized in early stages and easier to control than are the root and canker diseases. The following diseases are the most important and common foliage diseases of forest tree seedlings in North America.

19. LOPHODERMIUM NEEDLE CAST OF PINES

Lophodermium pinastri (Schrad.) Chev.

DARROLL D. SKILLING

Although long considered a serious disease in European nurseries and plantations, *Lophodermium pinastri* has only recently received attention as a major disease in the United States. It is currently causing serious losses in red and Scotch pine nurseries in the eastern United States.

Distribution

The disease occurs throughout the United States. Severe damage to nursery stock has been reported in Michigan, Wisconsin, Minnesota, West Virginia, Washington, Massachusetts, Pennsylvania, North Carolina, South Carolina, and Nova Scotia.

Hosts and Damage

L. pinastri has a wide host range. It is known to attack at least 26 species and varieties of pines. In North America the

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Figure 19–1.—Red pine seedlings (2–0) infected by *Lophodermium pinastri*. Green trees in foreground were protected with the fungicide maneb.

most serious damage has been to red and Scotch pines. Other pine hosts include eastern white, jack, ponderosa, Mugo, Austrian, Jeffrey, western white, Monterey, lodgepole, shortleaf, pitch, loblolly, longleaf, and slash.

The first symptoms of the disease are brown-black spots on infected needles. Greenish-brown bands appear on infected needles in late summer and fall. These infected needles turn brown the following spring. On most pine hosts the damage from *L. pinastri* has been limited to premature dropping of only part of the needles. In many forest nurseries, however, entire beds of red and Scotch pine seedlings have been killed by extensive needle defoliation or terminal bud dieback (fig. 19–1). Since 1966, several million trees 1 to 4 years old have been killed by this disease. Mortality has been especially severe in 1- and 2-year-old trees. In addition, the quality and vigor of millions of surviving trees have been reduced by severe needle loss. Heavy losses in field plantations have resulted when seedlings were outplanted before it was apparent that they were infected. In many cases, symptoms developed after field planting.

Life History

Infection appears to be entirely by airborne ascospores, which germinate on the pine needles. The spores are produced in tiny, black, elongate fruiting bodies called hysterothecia. These develop on either attached or fallen needles during the late summer or fall following infection. During wet weather, the ascospores are forcibly discharged into the air. The major infection period is from August to October.

Nursery infection may develop from either infected needles in the seedbeds or from nearby infected windbreaks.

Control

Infection by *L. pinastri* can be prevented in nursery beds by repeated applications of maneb or chlorothalonil (Bravo W–75 or 6F). Normally, four to six applications from mid-July to September will give adequate protection.

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20. BROWN SPOT NEEDLE BLIGHT
Scirrhia acicola (Dearn.) Siggers, *Systremma acicola* (Dearn.) Wolf and Barbour

ALBERT G. KAIS*

Brown spot needle blight, the most serious disease of longleaf pine, both in the forest and in the nursery (fig. 20–1) has been a primary reason for the widespread planting of other pine species on former longleaf sites. Its effect on growth and vigor in the nursery is severe, and a control program is essential.

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Figure 20–1.—Longleaf pine seedlings infected with brown spot.

Distribution and Hosts

Brown spot needle blight is common in nurseries in the natural range of longleaf pine (U.S. Coastal Plain from North Carolina to Texas). It may infect loblolly and slash pines within

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or beyond the range of longleaf. It is occasionally found on other southern pines, and on some exotic nursery stock in the south. Recently, it was reported on white pine nursery stock in Tennessee.

The range of *Scirrhia acicola* is by no means restricted to the south. The fungus has been reported in Missouri and Ohio, and has recently appeared in jack pine plantations in Canada. It has also caused severe damage in Scotch pine Christmas tree plantations in six North and Central States. Only the conidial (imperfect) state of the fungus, *Lecanosticta acicola* (Thum.) Syd., has been found in northern areas.

Symptoms

Brown spot lesions may develop on secondary needles at any season of the year, but appear most commonly from May through October. The first evidence of infection is the presence of small, circular, grayish-green spots. These become straw yellow and later change to a light brown. A darker chestnut-brown discoloration often borders the spot when fruiting bodies form in the lesion. The individual spots, which are about one-eighth inch in diameter, frequently coalesce. Eventually the needle tissue beyond and between groups of spots dies. Needles with multiple infections take on a mottled appearance. Another type of lesion, known as bar spot, consists of an amber-yellow band with a small brown center encircling the needle. Both types of spots have well-defined margins.

The infected needle usually has three definite zones: (1) the basal portion, which is green; (2) the middle portion, which is spotted with lesions that alternate with green needle tissue; and (3) the apical portion, which consists of dead needle tissue. The loss of needle tissue stunts the growth of nursery seedlings and may kill them. If badly infected seedlings are outplanted they may be too weak to survive competition and dry weather, or may remain in the grass stage for many years.

Life Cycle

The macroscopic fruiting bodies (acervuli), which are found on lesions, produce conidia which rupture the leaf surface. The conidia are cylindrical, curved, 1–4 septate, and olive green to brown in color; they are exuded in a sticky mass which may form a ribbon up to 1 mm in length. Conidia are disseminated for short distances by rain splash and cause local intensification of the disease.

In 2 to 3 months after initial infection, ascospores are produced in perithecia that are embedded within dead leaf tissue. They are hyaline, oblong-cuneate, unequally bicellular, and have two prominent brown oil drops in each cell. Mature ascospores are discharged during periods of rain, heavy dew, and fog. They are disseminated by wind. It is thought that these spores

initiate light, scattered infections, sometimes at great distances from their source, and are the principal means by which the fungus invades nursery beds.

Control

Control in nurseries can be achieved with chemical sprays. A freshly made 4–4–50 bordeaux mixture (4 pounds copper sulfate, 4 pounds hydrated lime, and 50 gallons of water) is economical and effective. No spreader-sticker is required. This mixture is applied at the rate of 60 gallons per acre at 300 pounds pressure. Seedlings should be sprayed at frequent enough intervals to protect new foliage as it develops. Four to seven applications of bordeaux mixture from the end of May to October are usually sufficient. Nursery managers in high-hazard areas probably should spray every 2 weeks throughout the infection period, and should schedule a final spraying just prior to lifting.

Maneb and chlorothalonil (Bravo) have been registered for use on brown spot needle blight. Applications of maneb or chlorothalonil should begin in spring or early summer, before infection occurs, and should be repeated at 2-week intervals through the end of summer. Extra applications should be made after heavy rains. Sporulation and dissemination of the fungus occurs in the deep South during unseasonably warm periods in the winter months. Therefore, seedlings that are held beyond the usual lifting date may benefit from a precautionary spray.

Recent research has revealed the existence of heritable resistance to brown spot needle blight, and resistant longleaf nursery stock may thus become available.

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21. DOTHISTROMA NEEDLE BLIGHT OF PINES

Scirrhia pini Funk & Parker, *Dothistroma pini* Hulbary

GLENN W. PETERSON

Dothistroma needle blight is the most damaging disease of pines in the southern hemisphere (East Africa, New Zealand, Chile). Severe damage has also been caused by *Dothistroma pini* in North America, especially in plantings. Although infection of pine seedlings in North American nurseries has seldom been observed, it has become evident that infection in new plantings has frequently resulted from use of infected nursery stock.

Distribution and Hosts

Dothistroma pini has been found in 23 of 50 States in the United States and in three Provinces in Canada; it has not been reported in Mexico. Twenty pine species and hybrids have been reported as hosts in North America. Dothistroma needle blight has caused considerable consternation in the past decade because of the devastation caused in pine plantations (chiefly *Pinus radiata*) in the Southern Hemisphere.

The fungus has commonly been found on 4- to 5-year-old pines in nurseries in the central United States which produce pines for landscape plantings. Seedlings infected with *D. pini* have seldom been detected. Yet, experience with epidemics in new plantings in the Great Plains indicates that trees infected in the nursery have been responsible for outbreaks in the plantings.

Symptoms and Life Cycle

Symptoms develop in the fall of the year of infection in the central United States and in British Columbia. Early symptoms consist of yellow and tan spots, and bands that appear water soaked, on the needles (fig. 21–1). The bands and spots may turn brown to reddish brown. Commonly, the distal ends of infected needles become chlorotic, then necrotic, with the base of the needles remaining green. Extensive necrosis of needles may occur within 2 to 3 weeks of the first appearance of symptoms.

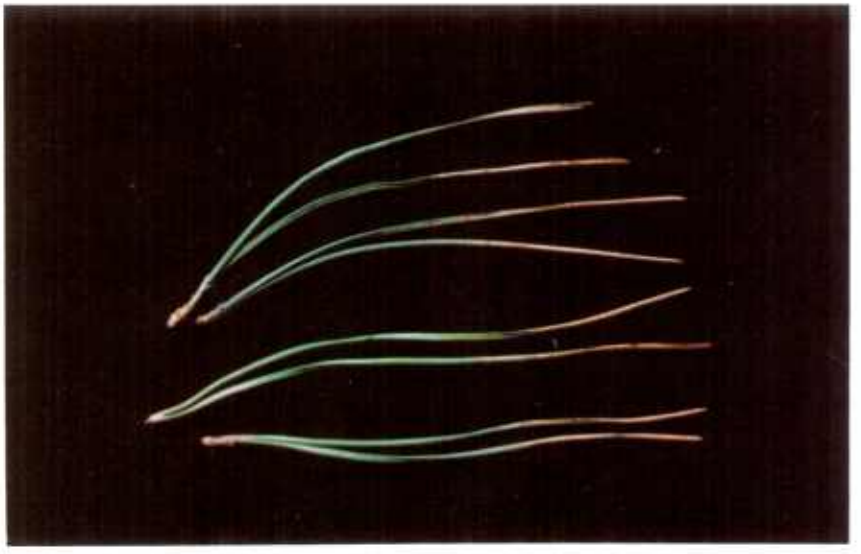

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Figure 21-1.—Symptoms of Dothistroma needle blight on Austrian pine needles.

Infected needles are cast prematurely. Infected second-year needles are cast before infected first-year needles. In some seasons, second-year needles are cast extensively in late fall of the year they became infected. In other seasons, casting is not extensive until late spring or early summer. Infected first-year needles often are not shed until late summer following the year they were infected.

The fungus has both a sexual stage (*Scirrhia pini*) and a nonsexual stage (*Dothistroma pini*). The sexual stage has been found in British Columbia, Alaska, and California, but not yet elsewhere in North America. Ascospores are produced by the sexual stage; their role in development of epidemics is not known. However, much is known about the role of conidia (produced by the asexual stage) in disease development. Conidia are cylindrical, curved, 1- to 5- but usually 3-septate, and hyaline (fig. 21–2). The conidia are about 3.5 μm wide and vary in length from 16 to 64 μm. Conidia from western North America are considerably longer on the average than are conidia found elsewhere on the continent.

Conidia (spores) of *D. pini* are borne in stromata on needles. Stromata may begin to develop in the fall in the central United States and British Columbia, but most stromata mature in the spring following the year of infection. Stroma development in the fall may be sufficient to cause splits in the needle epidermis, but spores have not been observed in these stromata in the fall (fig. 21–3). The conidia are disseminated by rainsplash throughout the growing season (May–October). Infection occurs throughout the growing season; symptoms do not develop, however, until late summer or early fall in the central United States.

Two growing seasons are required for completion of the life cycle in most areas of North America, although in California the cycle may be completed in 1 year.

Control

Copper fungicides effectively control *D. pini*. Bordeaux mixture (8–8–100) applied twice in the growing season (mid-May, mid-June) has provided essentially complete control in shelterbelt, Christmas tree, and other plantings in the central United States. The first application protects previous seasons' needles;

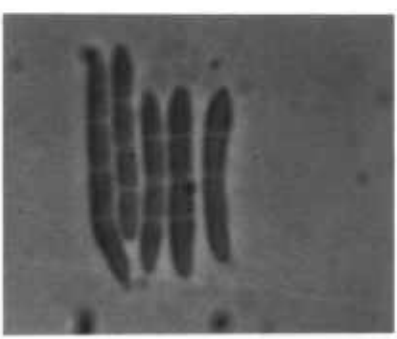

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Figure 21–2.—Conidia (spores) of *Dothistroma pini* obtained from needles of Austrian pine.

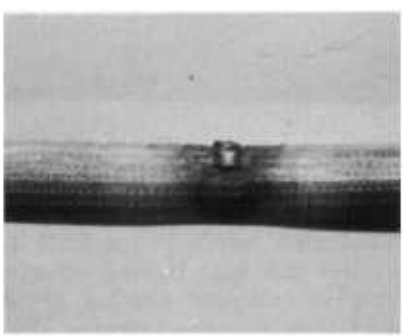

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Figure 21–3.—Fruiting body of *Dothistroma pini* raising the epidermis of an Austrian pine needle.

the second application protects current-year needles. The second application may be delayed until mid-June when controlling this disease in plantings of Austrian or ponderosa pines, since new needles of these species are resistant to infection until midsummer (July).

Effective control has also been obtained in plantings in the Midwest with a single application made after considerable growth has occurred (early June). There is some risk in this procedure, since infection could occur prior to the early June application. A single application will control this disease on trees which do not have susceptible first-year needles. The above control measures can be used by nurserymen to control Dothistroma blight in nursery windbreaks.

Detection of infected seedlings in the nursery may be difficult for two reasons: the level of infection is likely to be low, and symptoms do not develop until late summer or early fall. Although bordeaux mixture will effectively control this disease in seedling beds, no tests have been made in nurseries to determine the number and timing of applications that would be required. The two-spray schedule so effective on older trees might not be effective on seedlings. In New Zealand, monthly applications of bordeaux mixture effectively controlled the disease in seedling beds of *P. radiata.*

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22. PHOMOPSIS BLIGHT OF JUNIPERS

Phomopsis juniperovora Hahn

GLENN W. PETERSON

Blight caused by the fungus *Phomopsis juniperovora* is a serious nursery disease of junipers in the United States. Epidemics in nurseries in the Great Plains have resulted in total losses in seedling and transplant beds.

Distribution

Phomopsis juniperovora is common in the Great Plains from North Dakota to Texas, and eastward to the Atlantic coast. It is also known to occur in Ontario.

Hosts and Damage

Members of the Cupressaceae belonging to the genera *Chamaecyparis, Cupressus, Juniperus,* and *Thuja* are infected by the fungus. Losses have been most severe in seedling and transplant beds of eastern redcedar and Rocky Mountain juniper (fig. 22–1). High mortality can be expected in outplantings if blighted stock is used.

Small yellow spots appear on needles of eastern redcedar and Rocky Mountain juniper 3 to 5 days after infection. The fungus permeates needles and rapidly invades young stem tissue of seedlings and transplants. As a result, terminals and branches become light in color, then red brown, and finally ashen gray. Lesions on stem tissue frequently develop into cankers, which may girdle small stems and branches ⅓ inch or less in diameter. The main stem is frequently girdled at the base of infected branches. One-year-old infected seedlings are usually killed, but older stock is less likely to be killed because the causal fungus does not spread far below girdling cankers.

Damage due to drought may be confused with Phomopsis blight, since in both cases tips of branches may be killed. It is easy to distinguish the damage, however, as the line of demarcation between green and dead tissues will be sharp in Phomopsis-blighted seedlings and gradual in seedlings affected by drought.

F-522529

Figure 22-1.—Phomopsis blight infection of second-year seedlings of eastern redcedar.

Damage from the lesser cornstalk borer can be distinguished from Phomopsis blight by the straw color of the dead tops and by the feeding wounds on the lower stem and taproot. Another fungus, *Cercospora sequoiae,* causes a blight of junipers and other species in the Cupressaceae. This disease can be easily distinguished from Phomopsis blight, since Cercospora affects only the needles and infection starts on the oldest needles of lower branches and spreads upward and outward, whereas Phomopsis infection starts in newly developed needles.

Life History

The most important sources of inoculum early in the growing season are spores produced in fruiting bodies (pycnidia) which are formed on leaves and stems of seedlings infected the previous year. Spores are dispersed primarily by rain splash. Young nonwounded needles are susceptible. If spores land on these young needles, infection can occur if there is a short period (7 hours) of 100 percent relative humidity. Spore germination, germ tube development, and infection are optimum near 24° C; however, disease development is enhanced by higher temperatures (32° C). Symptoms may develop within 3 to 5 days. Pycnidia with viable spores may develop within 3 to 4 weeks. Pycnidia develop not only on the infected needles, but also on stems to which the fungus has spread from the young needles. Pycnidia usually are not well developed until infected

tissues have dried considerably. They are most commonly found on tissues which have turned ashen gray. The pycnidia are at first embedded in needles and stems, but later partially erupt through the epidermis. Spores are extruded in whitish tendrils. Two types of spores (A- and B-) develop in the same or different pycnidia. A-spores are colorless, one-celled, ellipsoid, contain two oil globules, and commonly are 7.5 to 10 by 2.2 to 2.8 μm; B-spores are colorless, one celled, filamentous, slightly curved, and commonly 20.2 to 26.9 by 1 μm. Intermediate type spores occur infrequently. Infection is caused by the A-spores; the B-spores do not germinate. The fungus can persist as long as 2 years as mycelium in dead parts of infected plants and produce spores. The perfect (sexual) stage of the fungus is unknown.

Control

Seedlings and transplants need protection during the entire growing season, since spores are disseminated throughout the period and young foliage of junipers is susceptible. Phomopsis blight has been effectively controlled in nurseries by frequent (usually weekly) applications of phenyl mercury fungicides. Now that mercury fungicides cannot be used, other effective fungicides need to be found. No effective nonmercury fungicides were found in earlier tests of over 40 fungicides. In recent tests, benomyl have been effective under low infection conditions; however, there is evidence that it may not be effective on seedlings under high infection conditions. Benomyl is now registered for use in controlling Phomopsis blight.

Protective fungicides need to be applied frequently to protect susceptible new foliage. To obtain control by limited applications, systemic fungicides would likely have to be used. Fungicide applications could possibly be limited to periods in which flushes of new growth occur. Commonly, new growth flushes on eastern redcedar occur early in the growing season and again late in the summer in nurseries in the central and northern Great Plains, but during most of the growing season in the South. Research is needed to determine if limiting applications of protective fungicides to such periods would be effective.

Juniper seed should not be sown adjacent to beds containing juniper stock. Poorly drained areas should also be avoided. If overhead sprinklers are used, seedlings should be irrigated so that water on seedlings dries before nightfall. Because shading frames increase the length of time moisture remains on foliage, they should not be used unless absolutely necessary. Junipers or other hosts of this fungus should not be used in nursery wind breaks or in landscape plantings on nursery grounds, since they may be a source of inoculum (spores) for nursery stock. Such trees are likely to be extensively infected if pruning results in development of juvenile foliage.

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23. CERCOSPORA BLIGHT OF JUNIPER

Cercospora sequoiae Ell. & Ev.

CHARLES S. HODGES

Cercospora sequoiae commonly causes a needle blight in Arizona cypress in Christmas tree and ornamental plantings throughout the southern United States, but is seldom noted on eastern redcedar in such plantings. Occasionally, however, *C. sequoiae* will cause severe damage to redcedar seedlings in the nursery.

Distribution and Hosts

Cercospora sequoiae has been reported from most of the southern United States and as far north as Pennsylvania. Eastern redcedar and giant sequoia are the only known seedling hosts of the fungus. Several species of cypress, especially Arizona cypress, Italian cypress, and Monterey cypress, and Oriental arborvitae are susceptible in the field.

F-522530

Figure 23-1.—Eastern redcedar seedlings infected with *Cercospora sequoiae*.

Symptoms

The disease first affects needles adjacent to the stem on the lowest branches. Infection then progresses upward and outward until only the needles at the tips of the branches remain green on severely affected plants (fig. 23–1). The disease is thus easily distinguished from the more common Phomopsis blight, *Phomopsis juniperovora* Hahn, which kills the tips of the branches.

Life Cycle

Stromata of the fungus form profusely on the needles shortly after they turn brown, appearing as tiny dark pustules about 100 μm in diameter. Several brown, geniculate, 50–125 μm high conidiophores arise from each stroma (fig. 23–2). The conidia are yellow brown, cylindrical, 5 to 6 septate, echinulate, and average about 40 x 5 μm. They are produced dry and are spread by wind.

In all cases where redcedar seedlings are infected in the nursery, heavily infected Arizona cypress trees were present in the vicinity of the beds. These infected trees probably served as sources of infection for the seedlings. The fungus overwinters on infected needles on living trees.

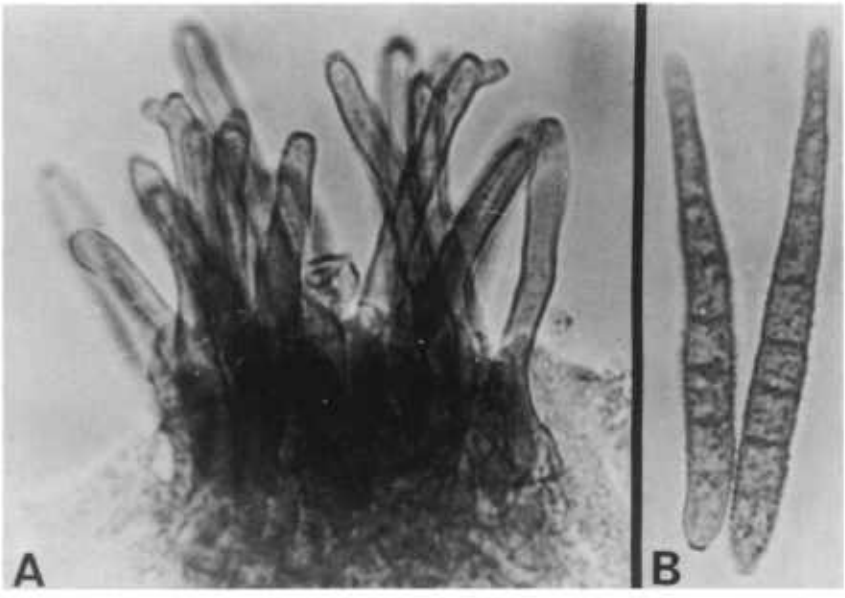

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Figure 23–2.—Conidiophores (*A*) and conidia (*B*) of *Cercospora sequoiae*.

Control

Seedlings should be sprayed with 8–8–100 bordeaux mixture at 7- to 10-day intervals, once the disease is determined to be present. Where it occurs frequently, this spray schedule can be made standard practice, starting June 1. Sprays should be continued throughout the summer.

All Arizona cypress trees around the nursery should be examined for the disease, and infected trees removed or sprayed.

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24. ROSELLINIA NEEDLE BLIGHT

Rosellinia herpotrichoides Hept. & Davis

RICHARD S. SMITH, JR.

Rosellinia needle blight was originally described as a needle blight of mature eastern hemlock in the forests of the southeastern United States. Since the early 1950's, it has appeared occasionally on seedlings in the forest nurseries of western North America.

Distribution

This disease appears to be limited to California, Oregon, Washington, and British Columbia.

F-522532

Figure 24-1.—A 2-year-old Douglas-fir seedling attacked by *Rosellinia herpotrichoides* shows defoliation, and matting of the remaining foliage.

F-522533

Figure 24-2.—Perithecia of *Rosellinia herpotrichoides* grouped on stem and lower needles of an infected Douglas-fir seedling.

Hosts and Damage

In the nurseries of western North America, the disease has been reported on Douglas-fir and Sitka spruce.

This disease appears to kill few seedlings. The pathogen grows over the outside of the seedling, infecting and killing needles and small branches. The infected needles fall, and in many cases the seedlings may be completely defoliated (fig. 24–1). This defoliation causes many seedlings to be culled, which results in the greatest loss.

Life History

Rosellinia needle blight usually starts in the lower portions of the seedling crown in the center of crowded beds. The fungus grows over the outside of the seedling, infecting individual needles as it comes to them. The fungus can be seen on the seedling surfaces. At the advancing front of the infection, the mycelium forms a fine white mycelial web which encompasses whole tufts of needles and small branches. The needles then turn yellow and die. As the mycelium ages, it and the dead needles darken, become dense, and flatten, forming dark brown wet mats of mycelium and needles (fig. 24–1). Groups of perithecia may be found on these old mats at the base of the seedling crown (fig. 24–2). The perithecia produce ascospores which may be released to start new centers of infection.

This disease requires mild temperatures and long, continuous periods of high humidity to become damaging. It usually appears after long periods of rain or fog, and becomes most severe in the center rows of dense, overstocked nursery beds.

Control

Reduced stocking to improve aeration and to lower humidity within the seedling beds is one of the most effective means of controlling this disease. Some standard fungicides have been used successfully.

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25. SNOW BLIGHT OF CONIFERS

Phacidium sp., *Lophophacidium* sp., *Sarcotrichila* sp., *Hemiphacidium* sp.

DARROLL D. SKILLING

Tree seedlings are frequently damaged severely by snow blight in the nursery. Damage is primarily limited to pines, spruces, and firs. Snow blight in North America was formerly attributed to the fungus *Phacidium infestans,* the cause of snow blight on Scotch pine in Europe. Recent taxonomic work has shown that snow blight in the United States and Canada is due to a number of different fungi.

F-522534

Figure 25-1.—White spruce seedlings that show symptoms of snow blight in early spring.

Distribution

Snow blight occurs in the northern United States and Canada wherever deep snow covers the nursery beds for long periods. Snow blight fungi are apparently native to North America, and are widely distributed in natural stands of spruce and fir.

Hosts and Damage

The principal hosts of snow blight in North America are Norway, white, black, blue, red and Engelmann spruces; Douglas-fir; ponderosa, white, and limber pines; and balsam, white, grand, and subalpine firs.

The fungi that collectively are called snow molds can grow at the low temperatures found under deep snow. These include *Phacidium abietis, Lophophacidium hyperboreum, Sarcotrichila piniperda, S. balsameae,* and *Hemiphacidium planum.*

Foliage infected with snow blight fungi turns a glaucous-brown color in the spring after the snow melts (fig. 25–1). Infection frequently occurs in sharply defined patches, and is usually most severe in nursery areas where snowmelt is delayed. After the snow melts, a covering of white mycelium can be seen on the browning foliage. Mild attacks kill only the foliage below the snowline, but heavy infections kill large numbers of seedlings. Frequently, however, the roots live for some time after the shoots are dead.

Life History

In late summer, small, brownish-black, disk-shaped apothecia appear on the undersurface of infected, browned needles. Ascospores are discharged from these apothecia during moist weather until winter. Tiny, black microsclerotia, resistant to drought and other unfavorable climatic factors, are also produced on the surface of infected needles.

Primary infection of needles occurs in the fall from the wind-disseminated ascospores. The ascospores resting on the needle surfaces germinate as soon as the needles become embedded in snow, from which the spores receive the required moisture. Needles of all ages are susceptible. The microsclerotia probably also function like spores. Because the fungus is able to grow under a deep snow cover, seedlings that appear healthy in the fall may show severe foliage browning or mortality the following spring. Secondary infections also occur, as the fungal mycelium from infected needles grows out under the snow onto adjacent foliage and enters healthy needles.

Control

Snow blights may occur inconspicuously on low branches of trees up to the pole size; in spruces, even later. Therefore, care

should be taken to minimize such sources of infection near or within a nursery.

As far as possible, localities where snowmelt is delayed should not be used as nursery sites. Moreover, susceptible tree species should not be grown in nursery beds where snow tends to accumulate in drifts. If only scattered patches of this disease are found in nurseries, its spread may be eliminated by careful removal of visibly infected seedlings and those adjacent to them.

In Sweden, seedlings from a northern provenance are more resistant to snow blight than those from a southern provenance. Finnish workers found that snow blight is severe in nurseries where the soil is low in potassium.

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26. SYCAMORE ANTHRACNOSE
Gnomonia platani Kleb., *Gloeosporium platani* Oud.

WINAND K. HOCK AND FREDERICK H. BERRY*

Sycamore anthracnose, caused by the fungus *Gnomonia platani* Kleb. (asexual stage, *Gloeosporium platani* Oud.), is often responsible for the shabby and ragged appearance of sycamores during May and June. Although usually more unsightly than lethal, repeated defoliation by this disease for several successive years can so weaken trees that they may succumb to other pests or to environmental pollutants and climatic stress.

Distribution

Anthracnose occurs throughout the entire range of sycamores.

Hosts and Damage

The disease is extremely severe in some years on American and California sycamores, although all species of sycamore—including the less susceptible London plane tree—can be attacked when conditions are suitable for the fungus.

Four distinct stages of sycamore anthracnose are generally recognized: twig, bud, shoot, and leaf blight. Any one or more of these stages can be seen during the growing season, although most damage to nursery seedlings results from the leaf blight stage unless plants are grown for more than one season in the nursery. The entire developing shoot of nursery seedlings may die. This shoot blight is probably the result of direct infection and is comparable to the leaf blight stage of older trees.

The twig and bud blight stages develop early in the spring while the trees are still dormant. Cankers develop on infected twigs and ultimately girdle and kill the buds and tips of the twigs. Often the buds are killed even before they begin to swell. Repeated killing of twigs throughout the crown results in a gnarled and abnormally branched tree. Small, black fruiting bodies (acervuli) of the fungus appear in the bark of infected twigs and branch cankers.

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Shoot blight appears as the sudden dying of developing shoots and immature leaves. The fungus girdles and kills tissues surrounding the shoots, which kills the young leaves on the emerging shoots. This damage is often confused with frost injury.

The leaf blight stage is produced by direct infection of the developing or mature leaves by fungus spores produced on twigs and branch cankers. Irregular, necrotic areas form adjacent to and around diseased spots on the midrib and principal veins (fig. 26–1). Creamy brown pustules of the fungus are evident on the diseased tissues, particularly on the midrib and veins.

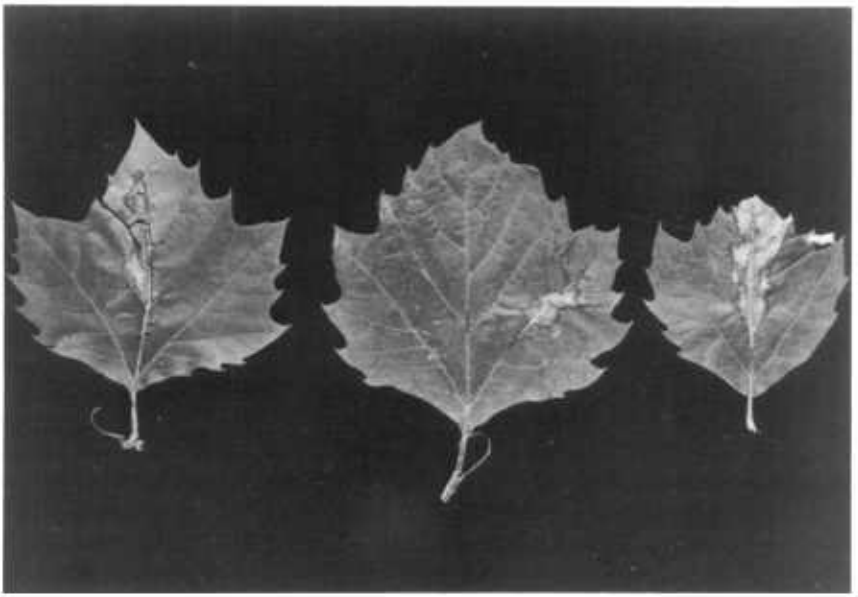

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Figure 26–1.—Anthracnose of sycamore leaves causes irregular, necrotic areas adjacent to and surrounding diseased spots on the midrib and principal veins. (Photo courtesy of Illinois Natural History Survey, Urbana, Illinois).

The disease is severe in some years and virtually nonexistent in others. Temperature is critical during the first 2 weeks after leaf emergence. Shoot blight is usually severe if mean daily temperatures average between 10° and 13° C during this period. Shoot blight decreases as the average temperature increases above 13° C.

Life History

The principal sources of inoculum for early spring infections are conidia produced on infected twigs and branches and on fallen leaves. Ascospores borne in perithecia are also produced on fallen leaves under certain conditions. Conidia and ascospores are carried from infected twigs and leaves by wind and splashing rain to new leaves, where they germinate and cause new infections. Conidia, borne in structures called acervuli, are hyaline, one celled,

ovoid to oblong, sometimes slightly curved, and average 9.7 x 5.0 μm in size. Ascospores, on the other hand, are asymmetric in shape, have rounded ends and two cells, the smaller cell being only about

F-522537; F-522538

Figure 26-2.—*Above,* Control of sycamore anthracnose on 1-year-old American sycamore seedlings with two applications of triarimol fungicide; *Below,* unsprayed control plot.

one-fifth to one-fourth the length of the larger. Ascospores average 13.4 x 3.7 μm in size.

During wet weather, small creamy brown pustules containing conidia develop in the underside of the leaf on dead tissue along the veins. These conidia may be washed to other leaves by rain to cause new infections. The fungus then spreads through the veins, midribs, and petioles into the twigs, where it overwinters and becomes active the following spring. The cycle is repeated when acervuli appear in the bark of infected twigs, and conidia are released to initiate new infections.

Control

There are two control procedures: sanitation and chemical sprays.

Fallen leaves and twigs should be gathered and burned before the fungus becomes active in the spring. Infected twigs and branches should be pruned and burned whenever possible. Application of a complete fertilizer will improve the vigor of trees weakened by repeated attacks.

Control of sycamore anthracnose is possible with properly timed applications of a suitable fungicide. Current recommendations call for three or more applications of zineb, dodine, or copper fungicide. The first spray should coincide with a swelling of the buds, and the second and third should be made at approximately 10- to 14-day intervals after the initial spray.

Newer experimental fungicides such as triarimol, benomyl, and thiophanate-methyl have shown considerable promise for combating sycamore anthracnose in an Ohio nursery (fig. 26–2). They may eventually replace the current generation of materials, which are less effective than the environmentally unacceptable organic mercury compounds.

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27. WALNUT ANTHRACNOSE

Gnomonia leptostyla (Fr.) Ces. & de N.

FREDERICK H. BERRY AND WINAND K. HOCK

Walnut anthracnose, sometimes called leaf blotch, is a destructive disease of certain walnut species, particularly the eastern black walnut. It is caused by the fungus *Gnomonia leptostyla* (Fr.) Ces. & de N., the imperfect stage of which is *Marssonina juglandis* (Lib.) Magn. The disease may quickly reach epidemic proportions during wet weather in the growing season and cause many walnut trees—from seedlings to mature trees—to lose almost all their leaves by late July or early August. This premature defoliation slows growth, weakens trees, and sometimes kills them. Nuts from infected trees are often poorly filled and of low quality.

Distribution

Walnut anthracnose is distributed worldwide. Besides infecting *Juglans* species in the United States, the disease has been reported from most of the countries of Europe and from Argentina, Canada, and South Africa. It is likely to be present in all nurseries where black walnut is grown.

Hosts and Damage

Eastern black walnut trees vary in their susceptibility to walnut anthracnose. But when weather favors development of the fungus, even the less susceptible trees become severely infected and defoliated. Butternut, Persian (English) walnut, and first-generation hybrids of Persian and black walnuts are also attacked. Two species of walnut native to California, the Hinds walnut and the California walnut, are both susceptible.

Leaves, nuts, and occasionally shoots of the current season's growth are attacked by the fungus. Tiny dark brown or black spots, circular to irregularly circular, appear on infected leaves (fig. 27–1). These spots gradually become more numerous, enlarge, and often merge to form still larger dead areas. The leaf area bordering these spots is usually chlorotic. Most infected leaves and leaflets fall prematurely, but a few sometimes remain attached to the tree for most of the season.

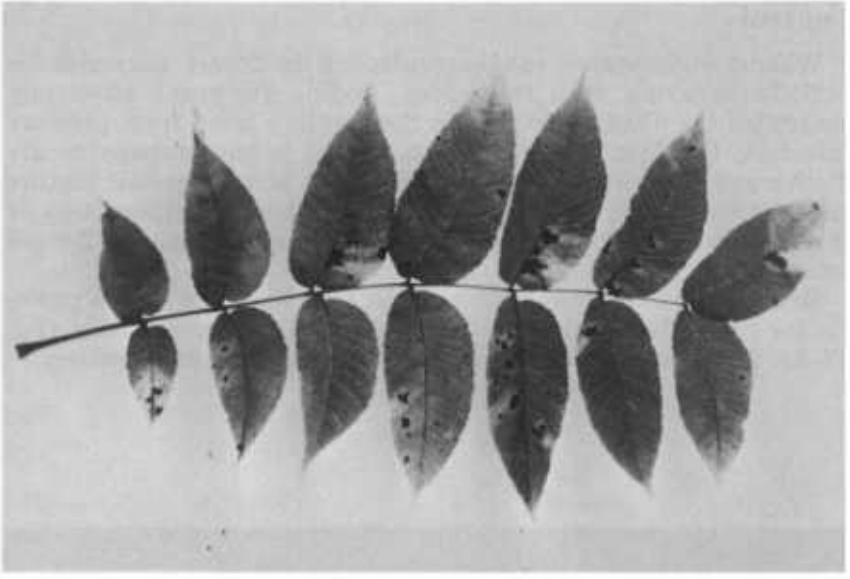

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Figure 27-1.—Anthracnose lesions on a black walnut leaf.

Sunken, necrotic spots, usually smaller than those on the leaves, appear on the husks of infected nuts. When immature nuts become diseased, they do not develop normally, and may drop prematurely.

Lesions are occasionally found on current shoots. These lesions are dead, sunken areas, oval to irregularly circular.

Life History

The fungus usually overwinters in fallen walnut leaves infected during the preceding summer. The fungus develops in these diseased leaves, and produces ascospores in the spring. These ascospores, which at maturity are 2.5 to 4.0 by 18.0 to 25.0 μm in size, cause primary infection. They are discharged from the walnut leaves during rainy periods and carried upward by wind or a combination of wind and rain. If they lodge on a susceptible leaf under favorable conditions, the ascospores germinate; leaf spots appear in about 14 to 16 days.

Secondary spores, the conidia, are produced on diseased leaflets in black fruiting bodies called acervuli. Acervuli are more abundant on the under side of leaflets, but occasionally a few are found on the upper side. The conidia are colorless, usually crescent shaped, and divided by the crosswall into two approximately equal cells. They are 3 to 4 by 14 to 30 μm in size. Conidia are borne in large numbers, and are spread from leaf to leaf by wind and splashing rain. The rapid increase and spread of walnut anthracnose in the summer and fall is attributed to repeated generations of conidia. Leaves are most likely to be infected and fall off during wet weather.

Control

Walnut anthracnose can be controlled in forest nurseries by periodic spraying with fungicides. Dodine (Cyprex) effectively controlled the disease. To protect the seedling trees from primary infection, the first spray must be applied before ascospores are discharged. The foliage on black walnuts is about one-half mature size at this time. Then, depending on weather conditions, two or three additional sprays should be applied at approximately 2-week intervals.

Recently an experimental fungicide, triarimol, has shown promise for controlling walnut anthracnose in Ohio nursery beds (fig. 27–2). Further studies are in progress to test its effectiveness.

F–522540

Figure 27–2.—Control of anthracnose on black walnut seedlings with triarimol fungicide. Unsprayed plots in foreground.

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28. MARSSONINA BLIGHT OF BIGTOOTH ASPEN

Marssonina populi (Lib.) Magn.

LEON S. DOCHINGER*

A foliar blight caused by the fungus *Marssonina populi* (Lib.) Magn. is the most damaging disease of bigtooth aspen seedlings in Ohio forest nurseries. Occasionally plant mortality exceeds 90 percent in 1-year-old beds. Also, weakened trees do not survive when outplanted, and may serve as a source of inoculum for infecting mature poplars and aspens.

Distribution

Although Marssonina leaf blight of bigtooth aspen seedlings is reported only in Ohio forest nurseries, the organism is common on

F-522541
Figure 28-1.—Marssonina leaf blight symptoms on bigtooth aspen.

F-522542
Figure 28-2.—Marssonina blighted bigtooth aspen seedling.

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trees throughout Canada and the United States. The causal fungus attacks aspen and poplar species in California, the southern Rocky Mountain region, the Intermountain region (particularly Utah, western Wyoming, and southeastern Idaho), the North Central region (Iowa and Indiana), most of the Northeastern region, and into southern Canada.

Hosts and Damage

Criteria are insufficient for separating the various host-specific strains of *Marssonina* into more than a single species. Thus, all reported pathogenic species of *Marssonina* on poplar hosts will be considered here as *M. populi.*

In North America, *M. populi* is associated with leaf diseases on practically all native and exotic aspen and poplar species. Hosts of this organism include white poplar, bigtooth aspen, and quaking aspen of the genus *Populus* in section Leuce; eastern cottonwood and black poplar in section Aigieros; and Carolina poplar, balsam poplar, narrowleaf cottonwood, and black cottonwood in section Tacamahaca.

M. populi produces two types of leaf spots on bigtooth aspen seedlings. On more tolerant plants, superficial brownish spots about 1 mm in diameter develop over the entire surface of the leaf and at times on the petiole. On susceptible hosts, these minute spots occur along with irregular reddish-brown lesions (4 to 5 mm in diameter) which penetrate both sides of the leaf (fig. 28–1).

The infection process is accelerated by repeated rainstorms and increasing temperatures during the late spring and early summer months. With such favorable environmental conditions for disease development, lesions coalesce into rust-brown to blackened necrotic areas in which the fungus sporulates abundantly. Lower leaves absciss prematurely and defoliation progresses upwards toward the tips of the shoots. When infection becomes severe, shoots are blighted and seedling mortality results (fig. 28–2, 28–3).

Life History

The leaf blight organism overwinters on dead twigs and plant refuse. By mid-May, conidia from hibernating stroma provide primary sources of inoculum. Airborne conidia are probably responsible for most of the initial leaf infections of bigtooth aspen seedlings. In nursery beds disease development is conditioned by climatic factors. Maximum damage may occur within a temperature range of 15°–25° C and 30–44 mm rainfall.

Later in the season, acervuli are produced on the lesions of diseased leaves. From these fruiting pustules, both macroconidia and microconidia are exuded in whitish-yellow masses. Conidia are disseminated from the action of rain splash, and cause infections of bigtooth aspen plants throughout the nursery beds.

F–522543
Figure 28–3.—Marssonina blight can devastate entire beds of bigtooth aspen.

Control

Cultural practices are important in reducing losses attributable to Marssonina leaf blight. These control practices include (1) removal of infectious plant debris, (2) reducing the planting density of seedlings for better air movement, and (3) avoiding overhead irrigation systems.

F–522544
Figure 28–4.—Bigtooth aspen seedlings protected by fungicide (Benlate).

Recently, Benlate has shown promise of controlling infections on bigtooth aspen in Ohio nursery beds (fig. 28–4). Studies of its effectiveness are continuing for possible registration.

Since some seedlings tolerate *M. populi,* natural selection and breeding of trees should be examined. Resistant clonal material under varying environments should also be investigated.

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29. MELAMPSORA RUST ON COTTONWOOD
Melampsora medusae Thum.

T. H. FILER, JR.

Melampsora rust has been known to afflict native and introduced poplars in the northern United States since the turn of the century, but its presence in the lower Mississippi River valley was not recognized until 1967. Each year thousands of acres in this area are being planted to cottonwood, and leaf rust infections in these plantations cause defoliation prior to normal abscission in the fall. In some years defoliation occurs in summer, reducing growth and vigor of planting stock.

Distribution

Melampsora rust is widespread in Canada and the United States. Severe summer defoliation of nursery stock has been reported in Mississippi, Illinois, and Texas.

Hosts and Damage

Eastern cottonwood, quaking aspen, balsam poplar, plains cottonwood, Rio Grande poplar, and many hybrids are hosts of *Melampsora medusae*.

The first symptoms, which may appear as early as April in Texas, are minute chlorotic spots on upper or lower leaf surfaces.

F-522545

Figure 29-1.—*Melampsora medusae* uredia on cottonwood leaf.

The spots enlarge, and yellow, powdery pustules develop to produce urediospores (fig. 29–1), which start new infections. If humidity is high, infections may recur until the entire leaf is covered with yellow spores. Defoliation will result when rust covers 50 percent or more of the leaf tissue. In a cutting nursery, defoliation in midsummer reduces vigor and limits height growth, thereby reducing the number of merchantable cuttings.

Life History

Melampsora requires two hosts to complete its life cycle. Cottonwood leaves are infected by windborne urediospores in spring, summer, and fall. These spores form yellow, powdery pustules, which release spores to cause additional cottonwood leaf infection. In late summer, telia are formed in orange-yellow crusts in the same pustules which formerly produced urediospores. The telia, which overwinter on fallen leaves, change in color from yellow to brown, and finally to black. In the spring, the germinating teliospores produce basidiospores which infect larch, Douglas-fir, and lodgepole, ponderosa, Monterey, and Scotch pines. Teliospores cannot infect cottonwood. Spring infection of cottonwood is by windborne aeciospores produced on conifers, or by urediospores that overwinter on green poplar leaves in frost-free areas.

During the summer, temperatures above 35°C and dry weather usually limit the spread of rust. In the fall, new rust infections occur when the micro-climates favoring rust development reappear. It appears the rust spread in fall is from the northern range to the southern range by successive urediospore infections.

Control

Melampsora rust has been controlled in experimental nursery tests by one summer application of cupric oxide or benomyl. A number of rust-resistant clones developed by the Southern Hardwoods Laboratory are available through several State forest nurseries. Stoneville clones 75 and 92 are not defoliated by rust, and should be favored in areas where summer defoliation is known to occur.

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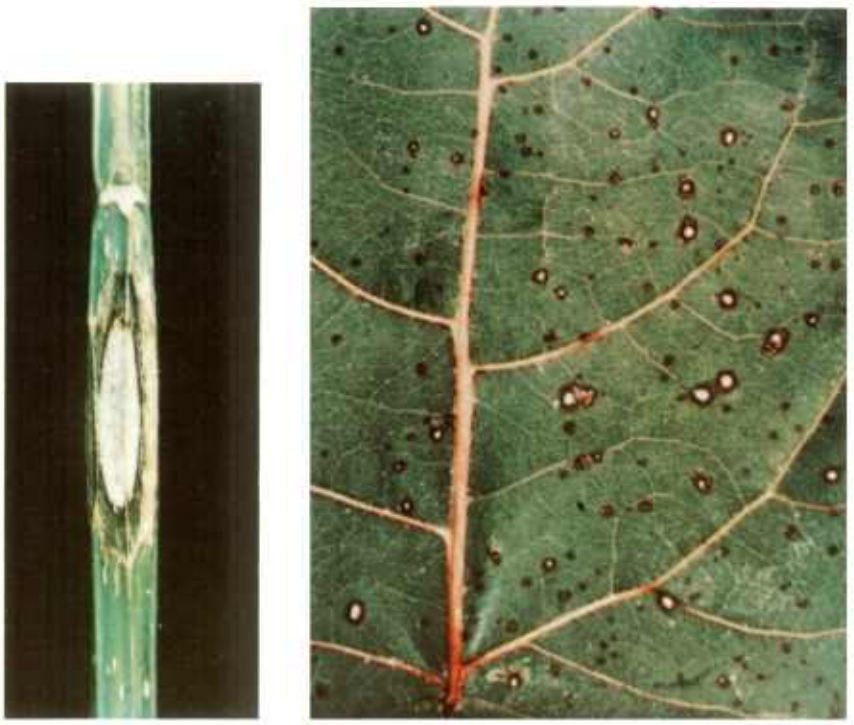
30. SEPTORIA LEAF SPOT AND CANKER ON COTTONWOOD

Septoria musiva Peck

T. H. FILER, JR.

Waterman reported in 1946 that *Septoria musiva* Peck caused cankers and leaf spots on hybrid poplars in the United States, but only recently was the fungus recognized as a cause of canker disease in cottonwood (*Populus deltoides* Bartr.) (fig. 30–1). *S. musiva* is currently causing high cull rates in nurseries producing cottonwood cuttings in the lower Mississippi River valley (Mississippi, Louisiana, and Arkansas). While the canker produced by the fungus does not girdle the stem, it provides entry courts for other fungi, such as species of *Cytospora, Phomopsis,* and *Fusarium,* which do girdle.



F–522547; F–522549

Figure 30–1.—*Left,* typical canker caused by *Septoria musiva* on 1-year-old cottonwood stem. *Right,* leaf spots on cottonwood caused by *Septoria musiva.*

Distribution

S. musiva occurs on poplars throughout the United States, Canada, and Argentina. It causes cankers on cottonwood in the lower Mississippi River valley.

Hosts and Damage

Septoria musiva causes leaf spot on all native species of aspen and poplars (fig. 30–1). It produces cankers on *P. deltoides* and *P. jackii* Sarg., a natural hybrid. It also causes cankers on many introduced species and hybrids. Since all native species of aspen and other poplars are susceptible to leaf infection, no poplar can be considered immune to *S. musiva* stem canker.

Leaf infections usually precede infection of stems. Tender leaves are infected at bud break, and leaf spots develop 1 to 2 weeks later. Spots first appear as depressed black flecks. Under favorable moisture conditions, flecks increase to a size of 2 to 5 mm. Spots coalesce on leaves with multiple infections, and as much as 50 percent of the leaf tissue can be affected. As the necrotic tissue dries, it fades to light tan or white in the center. Since the margin remains black, the leaf spot has a "bull's eye" appearance. Three to four weeks after infection, pycnidia appear as small, black, inconspicuous flecks in the centers of leaf spots. Spores, which exude during periods of high humidity, can infect leaves or stems.

In new cottonwood nurseries established from unrooted cuttings, leaf infections may occur during the first season from spores lodged on cuttings taken from infected stool beds, and from windborne spores from nearby plantations on native poplars.

Usually, less than 1 percent of 1-year-old cottonwood nursery stock has canker infections. Cankers are more frequent on 2- to 6-year-old stool beds. The increase in infection is due to the ascogenous stage, which overwinters on leaves and twigs on the ground. Cankers, which usually are within 3 feet of the ground, first appear as slightly sunken areas, smooth in the center, with raised margins. The infected cambium becomes black, the center bleaches to an ashy white, and tiny black pycnidia can be observed. The lesions average 1 cm wide by 4 cm long; they may persist to form perennial cankers. Stems infected with *S. musiva* are rapidly attacked by other fungi that cause additional damage. Planting stock containing cankers must be culled.

Life History

Most early spring infections on basal branches and leaves originate from ascospores. These spores are produced in tiny, black, flask-shaped fruiting bodies or perithecia which develop on fallen leaves or stems. When humidity is high, a pore is formed in the perithecia and ascospores are forcibly discharged into the

air. Throughout the growing season, further infection occurs during periods of high humidity, when pycnidiospores exude from leaves and stems. Spores are washed by rain or moved by air to new infection sites on stems and leaves.

Control

Loss of cuttings due to cankers can be greatly reduced through sanitation. After cuttings are harvested from stool beds in the winter, all leaves and stems should be plowed under or removed to prevent new shoots from being infected in spring by ascospores. Native poplars in or near the nursery should be removed to prevent early leaf infections by airborne pycnidiospores.

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31. YELLOWS, OR CHLOROSIS

S. J. ROWAN

"Yellows" and "chlorosis" are terms used to describe any physiological disturbance or disease of plants resulting in the yellowing or blanching of foliage (fig. 31–1). The destruction or reduced synthesis of chlorophyll may be caused by several biotic or abiotic factors, and is symptomatic of the decline in health of a plant. In general, chlorotic seedlings are unusually sensitive to injury by high or low temperatures, nematodes, fungi, bacteria, pesticides, and other factors. The interactions of chlorosis and these secondary agents cause the annual loss of up to 50 percent of seedling crops in forest tree nurseries. These losses result from both seedling mortality and the culling of seedlings which do not reach plantable size.

F–522550

Figure 31–1.—Chlorosis of loblolly *(left bed)* and slash *(right bed)* pine seedlings.

Description of Damage

All green plants are subject to chlorosis. The amount of growth reduction or mortality of seedlings depends upon the cause of the chlorosis, the length of time the plants are chlorotic, and the amount of chlorophyll that is deficient. Although chlorosis may be associated with rapid necrosis and death of the root system and plant, severe chlorosis may cause only slight growth reduction if it is of short duration, or it may cause high mortality if it persists for several months.

Causes

Although two or more agents often combine to cause chlorosis of forest tree seedlings, the more common agents are as follows:

Deficiencies or excesses of mineral elements essential to plants.—The elements most commonly associated with chlorosis in forest tree seedlings are iron, nitrogen, phosphorus, potassium, and calcium. Magnesium, manganese, zinc, boron, copper, molybdenum, and sulfur may also be associated with chlorosis.

Toxic levels of chemical compounds on foliage or in soils.—Excess herbicide, fungicide, nematicide, insecticide, or other compounds used in nurseries may induce chlorosis.

Insects and mites.—Feeding is more commonly associated with chlorosis than is oviposition, stinging, or other activities. Red spider mites, for example, commonly cause chlorosis of coniferous seedlings by feeding on foliage.

Parasitic fungi, bacteria, and nematodes.—Pathogens that cause root disease are most commonly associated with chlorotic seedlings, but foliar and stem pathogens may also cause chlorosis.

Viruses and mycoplasms.—"Yellows" is the descriptive term applied to those plant diseases caused by certain viruses and mycoplasms.

Genetic abnormalities.—The lack or loss of the ability to synthesize chlorophyll in plants may be caused by mutation or may be simply inherited. Albinism, a homozygous recessive condition, is the most common genetic abnormality associated with chlorotic forest tree seedlings.

Environmental stresses.—Excess soil water and high or low soil and air temperatures may cause chlorosis. Cold is often associated with the synthesis of pigments other than chlorophyll, leading to red, purple, yellow, or other pigmentation.

Mycorrhizae.—A deficiency of mycorrhizae will cause chlorosis. Excessive use of soil fumigants may reduce the number of mycorrhizal roots and cause chlorosis.

Miscellaneous agents.—Parasitic seed plants, such as dodder or seymeria, may cause chlorosis of tree seedlings.

Control

Because chlorosis of forest tree seedlings may be caused by any of several factors, cause(s) need to be accurately and rapidly diagnosed. The usual diagnostic procedure involves a process of elimination, with obvious and easily detected causes checked first. Excess soil water, insects, heat or cold injury, and disease symptoms are frequently easy to detect. Foliar and soil analysis will pinpoint any major nutritional inadequacies. These nutrient deficiencies can usually be corrected by applying the requisite element(s) to the foliage or soil.

The removal of excess fertilizer or toxic chemicals from soil is often difficult, and may require fallowing the contaminated area until the material has deteriorated or leached below the plant root zone by rain or irrigation. Contaminated soil may have to be removed. Insects and mites may be controlled with insecticides. Root diseases may be controlled by soil fumigation, before planting or after harvesting, with methyl bromide and other soil fumigants. Foliar and stem pathogens may be controlled by application of fungicides. Viral, mycoplasmic, and certain bacterial diseases may be controlled by excluding symptom-bearing plants from the nursery. Mulching, shading, and irrigating seedbeds to reduce air and soil temperatures provide some control for chlorosis caused by high temperatures. Proper soil drainage will eliminate excess soil water and the associated seedling chlorosis.

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32. AIR POLLUTION

LEON S. DOCHINGER AND KEITH F. JENSEN*

In the early 1900's, occasional damage to nursery crops in Canada and parts of the United States was attributed to fumes from local industrial operations. Today, besides such acute damage, the more insidious effects of long-term chronic pollution are being reported. Collectively, both the acute and chronic forms of air pollution are serious threats to the welfare of forest nurseries. In time, the primary offenders—ozone, fluorides, and sulfur dioxide—will probably become more widespread and may cause greater damage to forest tree nurseries. Also, new chemical forms of pollution arising from advancing technology may combine with current minor pollutants to cause more phytotoxicity to nursery plants.

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F-522552

Figure 32-1.—White pine: needle burn from oxidants under field conditions.

Ozone

Ozone (O_3) is the prime constituent of the photochemical smog that forms near urban areas. This oxidant damages more vegetation than any other pollutant in the temperate zone of North America.

Sources

A major portion of ozone is formed through light-energized reactions with the emissions from industries and automobile engines. Also, ozone is transferred to the earth's surface from the upper atmosphere by turbulence. Ozone also can be produced by electrical storms.

Symptoms

Conifers.—Rapid intake of toxic levels of ozone causes acute responses in forest tree species. Characteristic acute symptoms include needle banding and tip necrosis (fig. 32–1). Earliest manifestations from high concentrations of ozone are cellular collapse and the subsequent appearance of necrotic tissue on all ages of needles. Minute yellow, pink, or silver spots, along with mottled green and yellow patterns of chloronemia, are indications of chronic exposure to ozone. Intensification causes needle tipburn, defoliation, dwarfing of retained needles, and reduced seedling growth. Chronic needle symptoms result from low levels of oxidant concentrations for extended periods, and also may be observed on conifer needles displaying acute responses.

Hardwoods.—Both acute and chronic symptoms may appear on leaves of the same seedling. High concentrations of ozone cause necrosis on upper and lower leaf surfaces, and leaf drop of some broad-leaved species (fig. 32–2). Chronic exposure produces chlorotic and pigmented stipules and flecks on the upper surfaces of the foliage. When these metallic-like lesions coalesce, they impart an overall bronzed, silvery, or purple cast to damaged foliage. Little injury is expressed initially on some trees, but a few days after chronic exposure to ozone, premature loss of some mottled foliage results.

Fluorides

The fluorides are more harmful to vegetation than similar quantities of most other aerial phytotoxicants. Of the fluorides released into the atmosphere, the gaseous compounds of hydrogen fluorides and silicon tetrafluorides are more injurious than the particulate fluorides. Because of the expansion of industries that emit fluorides, there is potentially greater danger to seedlings from these pollutants if industrial control measures are ineffective.

F–522553; F–522554

Figure 32–2.—*Above,* Hybrid poplar: foliar injury from acute concentrations of O_3 fumigation; *Below,* bigtooth aspen: purple stippling and overall bronze cast from chronic levels of O_3 fumigation.

Sources

Fluorides are emitted into the atmosphere from electrolytic reduction of alumina; combustion of coal; manufacture of brick, phosphate fertilizer, and pottery; ferro-enamel processes; and steel and nonferrous ore smelting.

Symptoms

Conifers.—Symptoms vary widely in forest seedlings injured by gaseous fluorides. Usually, injury results from an accumulation of fluorides in plant tissues over an extended time. Chronic symptoms are chlorosis, banding, needle drop, and growth suppression. With continued absorption of low level (chronic) concentrations of fluoride, conifer needles, at first chlorotic, now become buff to reddish brown, and needle burn and tip necrosis occur over the entire conifer seedling. Acute damage is often characterized by tip and needle browning. Generally, older needles are more tolerant to fluoride pollutants, whereas new foliage is susceptible to both acute and chronic fluoride exposures.

Hardwoods.—In deciduous seedlings, fluorides are absorbed into the leaves and accumulate along the margins and apex. Usually, an initial chlorosis of the leaf tip develops and later extends downward along the margin and inward toward the midrib. With continued intake of fluorides, chronic areas become necrotic, and tissue degenerates. On some broad-leaved species, a given quantity of fluorides may cause necrosis of the more sensitive trees, growth reduction of less susceptible trees, and growth stimulation in more tolerant trees over similar exposure periods.

Sulfur Dioxide

For many years emissions of sulfur dioxide (SO_2) have damaged forest trees. Many of these harmful effects are produced not only by SO_2 but also from related oxides of sulfur, their acids, and acid salts. These sulfur-containing compounds may combine with other pollutants and create more plant injury than when they operate independently.

Sources

Primary sources of SO_2 are the combustion of fossil fuels, smelting of ores, production and manufacturing of natural gas, utilization of sulfur-containing compounds, and refining of petroleum products.

Symptoms

Conifers.—Sulfur dioxide may cause acute and chronic injury to conifer seedlings (fig. 32–3). The former results from a rapid absorption of high concentrations over a relatively short period. All ages of foliage are affected, but not all needles in a fascicle are harmed. Injured portions of the needle display a tannish-brown discoloration and tip necrosis. Chronic responses are usually found on the current year's needles. Chlorosis, necrosis, reduction in needle length, and needle loss often occur after exposure to sublethal concentrations of SO_2. Needles may also display both acute and chronic symptoms.

F–522555; F–522556

Figure 32–3.—*Above,* White pine: needle necrosis from acute levels of SO_2 fumigation; *Below,* Scotch pine: needle mottle from chronic levels of SO_2 in field.

Hardwoods.—After acute exposures to SO_2, tissues are dull and have a water-soaked appearance. In time, the tissues blanch and assume a gray-green color. These affected areas dry and bleach to a light-tan or ivory color in some species, but most often they become reddish brown. The final pattern consists of interveinal necrosis, but little effect is discernible across the veins of leaves unless injury is very severe. Chronic injury leads to a change in

leaf pigmentation, ranging from flecks to a diffuse mottling. Colors range from yellow, ivory, and bronze to black (figs. 32–4, 32–5).

F–522557; F–522558

Figure 32–4.—Yellow poplar, with chronic foliar symptoms of yellowing and necrotic spots: *Above,* new leaves blackened and deformed from SO_2 fumigation; *Below,* closeup.

Control

In Ohio forest nurseries, needle mottling proved to be an excellent characteristic for classifying eastern white pine seedlings as

F–522559; F–522839

Figure 32–5.—*Above,* White ash: chronic foliar symptoms of yellowing and flecking from SO_2 fumigation; *Below,* sugar maple: acute SO_2 injury from fumigation, with intercostal breakdown in tissues.

tolerant or sensitive to ambient pollution. Seedlings that show needle symptoms of air-pollution injury can then be eliminated during normal grading practices. Further selection and breeding of tolerant forest tree species should be encouraged.

IV. STORAGE MOLDS

DRAKE HOCKING*

Molds of many species may attack foliage or roots of stored seedlings, causing mortality, deformation, and stunting. Usually, losses from molding are a result of careless handling of stock or poor storage facilities.

Molding of coniferous foliage may cause severe losses by reducing survival and impairing first-year growth and form. It is sustained and encouraged by high temperature, high humidity, and free water. Water on foliage might originate by lifting stock during rain, wetting during packaging, use of excessively wet packaging materials, or condensation due to fluctuating storage temperatures.

Critical temperatures to prevent molding have not been established, but progressively lower temperatures at least down to 0° C result in progressively less molding. Seedlings stored at subfreezing temperatures generally do not mold, nor do seedlings heeled-in in snow. The hazard of molding may increase, however, during the thawing-out period at the end of storage. Danger and severity of molding tend to increase with duration of storage, if conditions conducive to molding persist.

Tree species differ in their susceptibility to molding, perhaps due to physiological differences or to morphological ones, such as better ventilation due to longer or stiffer needles.

Fungi that cause molding have seldom been identified, and only little studied. Genera mentioned as important are *Botrytis, Fusarium, Rhizoctonia, Cylindrocarpon, Phacidium, Pythium, Epicoccum, Phoma,* and *Penicillium.* Some authors report finding sterile mycelia.

Inoculum is commonly present on soil adhering to roots and foliage or in packing materials. Thirty-seven fungi isolated from moldy seedlings commonly occur in nursery soils; 10 isolated with significant frequency developed best under similar temperature and moisture conditions. Thus, storage conditions may be a more important factor in molding than the presence of any specific fungus or group of fungi.

Fungicides have been recommended to control molding during storage, but experimental testing generally provides negative or inconclusive results. Packing with peat has been found more

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effective than applying fungicides for mold control, and shingle-tow packing is more inhibitory than peat.

Hazard of root rotting has been reported serious among several deciduous species, the mortality reaching 100 percent among stock heeled-in in shingle tow. Some mortality may have resulted from toxins in the shingle tow, however.

An unknown agent, probably an infectious microorganism, caused a storage breakdown of hybrid poplar cuttings; pre-storage fungicidal dips gave satisfactory control.

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GLOSSARY

ABIOTIC—of or pertaining to the nonliving. Syn. inanimate.

ACERVULUS—a small subcuticular or subepidermal cushionlike asexual fruiting body, without a covering of fungus tissue, producing conidia in a moist mass which escapes through a break in the host tissue.

ACUTE—having a sudden onset and a sharp rise within a short period of time.

AECIOSPORE—one of several kinds of spores produced by a rust fungus. Formed in and released from a fruiting structure called an aecium.

ALTERNATE HOST—one or the other of the two unlike host plants parasitized by a heteroecious fungus such as a typical rust fungus, i.e., either the white pine or gooseberry host of the white pine blister rust fungus.

ANTHRACNOSE—a type of plant disease which typically is a leaf and twig blight. Common on many hardwoods.

APOTHECIUM(IA)—a cup or saucerlike sexual fruiting body which produces ascospores.

ASCOGENOUS STAGE—the ascospore producing stage of an Ascomycete.

ASCOMYCETE—a large group of fungi which are characterized by the free cell formation of spores, usually eight in number, in a saclike structure called an ascus.

ASCOSPORE—a spore produced in the sexual or perfect fruiting body of an Ascomycete.

ASEXUAL STAGE—either a vegetative stage or a reproductive stage in the life cycle of a fungus in which nuclear fusion is absent and in which reproductive spores are produced by mitosis or simple nuclear division. Syn. imperfect stage.

AUTOECIOUS—pertaining to a fungus which completes its life cycle on one host.

BASIDIOMYCETE—a large group of fungi which are characterized by the production of spores, usually four, on a basidium.

BASIDIOSPORE—the spore produced by the sexual stage of the Basidiomycetes.

BASIDIUM—a cell, usually terminal, in which nuclear fusion and meiosis occur and each of the four haploid nuclei pass into one of four forming spores.

BIOCIDE—a wide spectrum poison which kills a great number and variety of organisms.

BIOTIC—of or pertaining to living organisms. Syn. animate.

BLIGHT—a general term for a plant disease causing rapid death or dieback.

BROOM—an abnormally dense mass of host branches and foliage in which the typical host growth pattern is lost.

CAMBIUM—the layer of cells that lies between and gives rise by cell division to the secondary xylem (wood) and the secondary phloem (inner bark).

CANKER—a definite relatively localized necrotic lesion primarily of the bark and cambium.

CHLAMYDOSPORE—a thick-walled asexual resting spore typically formed by many soilborne fungi.

CHLOROSIS—an abnormal yellowing of the foilage.

CHLOROTIC—abnormally yellow.

CHRONIC—pertaining to a condition which is of long duration.

COLONIZE—to establish an infection within a host or part of a host.

CONIDIA—an asexual spore of a fungus, typically produced terminally on a specialized hyphae termed a conidiophore.

CONIDIOPHORE—a specialized hyphae which produces asexual spores called conidia.

CORTEX—the primary tissue of a first-year stem or root found between the epidermis and the primary vascular bundle.

COVER CROP—a crop, natural or introduced, that is grown alternately with the main crop. Used to prevent erosion and improve soil characteristics.

CULL—a seedling which is rejected because it does not meet certain specifications.

CULTURAL PRACTICES—a general term for those routine nursery operations required to help seedling growth, i.e., plowing, watering, weeding, etc.

CUNEATE—thinner at one end than the other; wedge shaped.

DAMPING-OFF—the killing of the seedling by microorganisms before emergence or the collapse of the seedling stem at ground level after emergence.

DECAY—the decomposition of plant tissue by fungi and other microorganisms.

DECLINE—the gradual reduction in health and vigor as a tree is in the process of slowly dying.

DIEBACK the progressive dying of stems and branches from the tip downward.

DISEASE—unfavorable change of the function or form of a plant from normal, caused by a pathogenic agent or unfavorable environment.

ECHINULATE—having many small spines or prickles.

ECTOPARASITE—a parasite (in particular, a nematode) which lives outside its host.

ENDEMIC—native to the country or region.

ENDOPARASITE—a parasite (in particular, a nematode) which lives inside its host.

EPIDEMIC—pertaining to a disease which has built up rapidly and reached injurious levels.

EPIDERMIS—the outermost layer of cells on the primary plant body.

EXOTIC—introduced from another country or area.
EXUDATE—matter which oozes out or is secreted out.
FACULTATIVE SAPROPHYTE—an organism which is normally parasitic but which is capable of living as a saprophyte.
FALLOW—cultivated land allowed to lie idle or unplanted during the growing season.
FLACCID—deficient in turgor, limp, or flabby.
FLAG—a conspicuous dead branch with the foliage remaining on.
FRUITING BODY—any of a number of kinds of reproductive structures which produce spores.
FUMIGATION—to apply vapor or gas to, especially for the purpose of disinfecting or destroying pests.
FUNGICIDE—chemical which is toxic to fungi.
FUNGI IMPERFECTI—a grouping of miscellaneous fungi which lack a known sexual stage and which are classified, therefore, according to the characteristics of their asexual stages.
GALL—a pronounced swelling on a woody plant caused by certain fungi, bacteria, insects, or nematodes.
GENICULATE—bent abruptly at an angle, like a knee.
GERMINATE—to begin growth from a seed or spore.
GIRDLE—to destroy or remove the tissue, particularly living tissue in a rough ring around a stem branch or root.
HETEROECIOUS—pertaining to a fungus which must pass a part of its life cycle on each of two different unrelated hosts, i.e., some rust fungi.
HOST—the plant on or in which a pathogen exists.
HOST RANGE—all hosts which a particular pathogen attacks.
HOST-SPECIFIC—a term used to describe those pathogens which attack only certain species of hosts.
HYALINE—transparent, having no color.
HYPHA—one of the filamentous threads which make up the fungus body.
HYPOCOTYL—that part of the axis of a developing embryo just below the cotyledons.
HYSTEROTHECIUM—a specialized fruiting body of needle cast fungi which produces ascospores, is usually elongate, covered, and opens at maturity by a long slit.
IMPERFECT STAGE—that part of the life cycle of a fungi in which only conidia and no sexual spores are produced. Syn. asexual.
INCITE—to cause a disease.
INDIGENOUS—native to a particular region or environment.
INFECT—to invade and cause a disease.
INFEST—to be present within an area in such numbers as to be a disease hazard.
INOCULATE—to place a pathogen on or in a host in a position in which it is capable of causing a disease.
INOCULUM—the spores, mycelium, sclerotia, or other propagules of a pathogen which initially infect a host or crop.

INTERCELLULAR—lying or growing between the cells.
INTRACELLULAR—lying or growing within the cells.
LATENT INFECTION—an established infection which does not show its presence.
LEAF SPOT—a leaf disease characterized by numerous distinct lesions.
LESION—a defined necrotic area.
MACROCONIDIA—the larger of two types of conidia produced by certain fungi, such as *Fusarium* spp.
MICROCONIDIA—the smaller of the two types of conidia produced by certain fungi.
MICROSCLEROTIUM—a very small (microscopic) sclerotium.
MORIBUND—being in a dying state.
MYCELIUM—a mass of hyphae which forms the vegetative filamentous body of a fungus.
NECROSIS—death of plant cells usually resulting in darkening of the tissue.
OOSPORE—the sexually produced resting spore of the water molds.
PATHOGEN—an organism which causes a disease.
PATHOGENIC—capable of causing a disease.
PARASITE—an organism living on and nourished by another living organism.
PERFECT STAGE—the stage in which the sexual spore stage is produced. Syn. sexual stage.
PERITHECIUM—a closed flasklike sexual fruiting body formed by certain Ascomycetes in which ascospores are produced.
PHELLUM—the suberized tissue produced by the cork cambium in the bark.
PHELLODERM—the parenchymatous type of tissue produced to the inside by the cork cambium.
PHLOEM—the tissues of the inner bark responsible for the transport of elaborate foodstuffs.
PHOTOLYTIC—pertaining to the chemical decomposition due to the action of radiant energy (sunlight).
PHYCOMYCETE—a group of lower fungi which includes the water molds.
PHYTOTOXIC—a chemical which is toxic to plants.
PYCNIDIUM—an asexual type of fruiting body, typically flask shaped, in which asexual spores or conidia are produced.
PYCNIDIOSPORE—an asexual spore or conidium produced within a pycnidium.
PYCNIOSPORE—a specialized spore produced in a pycnium by the rust fungi.
RESISTANT—able to withstand without serious injury, attack by an organism, or damage by a nonliving agency but not immune from such attacks.
ROOT CROWN—the uppermost portion of the root system where the major roots join together at the base of the stem.
ROT—see decay.

SAPROPHYTE—an organism using dead organic material as food.

SCLEROTIUM—a firm, frequently rounded multicellular resting structure produced by fungi.

SEPTATE—having cross walls which divide hyphae or spores into a number of separate cells.

SEPTUM—the cross wall which divides a hypha or spore into two or more distinct cells.

SEXUAL STAGE—the stage in the life cycle of a fungus in which spores are produced after sexual fusion. Syn. perfect stage.

SPORE—the reproductive structure of the fungi and other lower plants.

SPORULATE—to produce and release spores.

SPREADER—a chemical additive used in the preparation of fungicide sprays to improve the distribution of the spray on the plant foliage.

STICKER—a chemical additive which is added to fungicide sprays to improve the fungicide's retention on the plant surface.

STOMA, pl. STOMATA—a pore in the leaf epidermis, surrounded by two guard cells, leading into an intercellular space within the plant.

STROMA, pl. STROMATA—a cushionlike body on or in which fungus fruiting bodies are formed.

SUSCEPTIBLE—unable to withstand attack by an organism or damage by a nonliving agency without serious injury.

SYMPTOM—the evidence of disturbance in the normal development and function of a host plant, i.e., chlorosis, necrosis, galls, brooms, stunting, etc.

SYSTEMIC—affecting or distributed throughout the whole plant body.

TAPROOT—the primary descending root of a plant off of which the secondary or lateral roots branch.

TELIOSPORE—the spore of the rust fungi from which the perfect stage of the basidium and basidiospore arise.

TELIUM—an aggregation of teliospores of the rust fungi.

TRANSLOCATE—the transfer of elaborated food materials within the plant.

UREDIOSPORES—one of the many spore stages produced by the rust fungi in their complicated life cycle. These spores are produced in a fruiting body called a uredium.

UREDIUM—one of the many types of fruiting bodies formed by the rusts in their complicated life cycle. Urediospores are formed in this fruiting body.

XYLEM—the woody conducting tissues of the stem and root.

WILT—a type of plant disease characterized by the sudden loss in turgor and collapse of the succulent parts of the affected plants.

ZOOSPORE—a motile free swimming spore produced by the water molds.

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