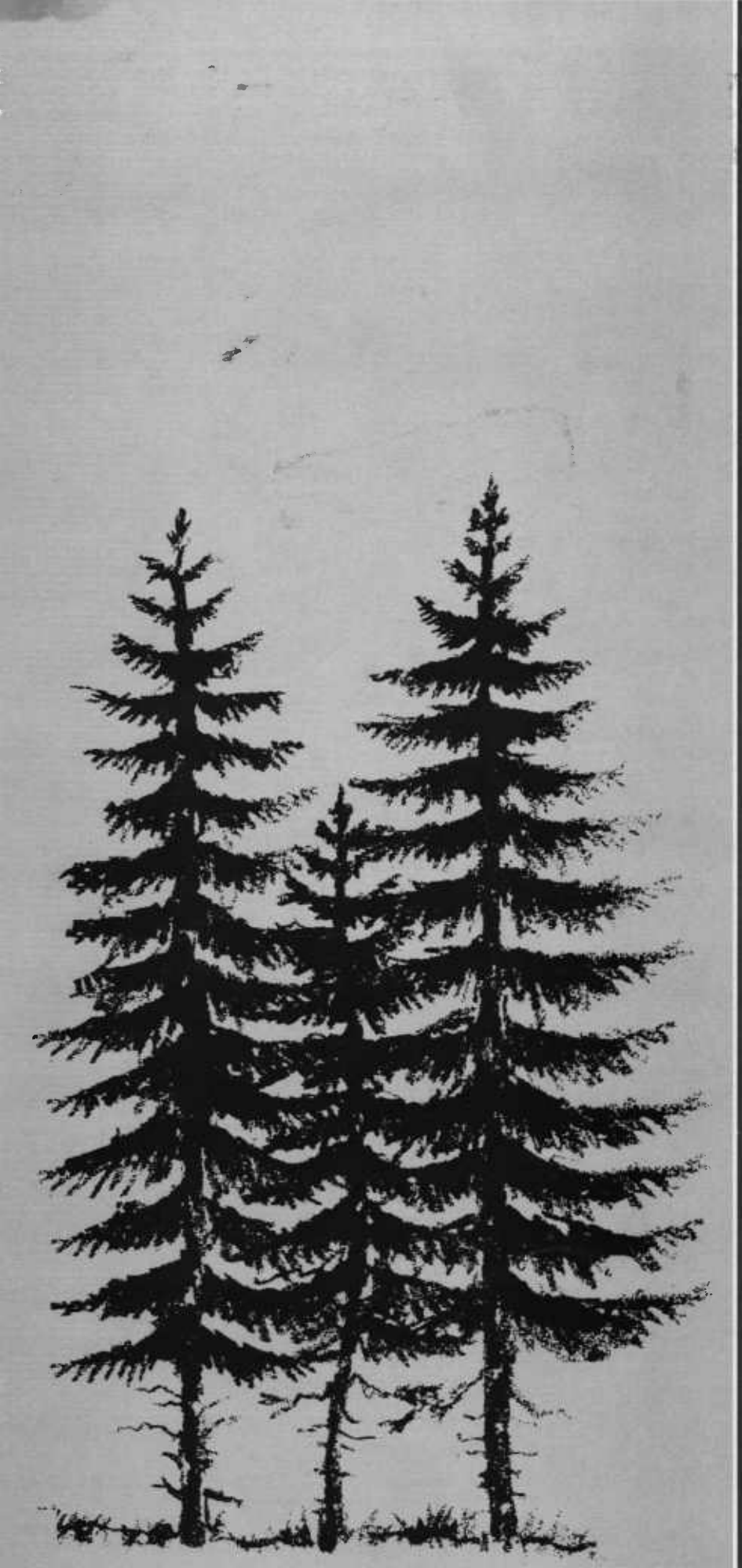
Manual 9 • October 1995
$14.50



Forest Disease Ecology and Management in Oregon

OREGON STATE UNIVERSITY EXTENSION SERVICE

Forest Disease Ecology and Management

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Acknowledgments

Technical Review

The authors wish to thank Everett Hansen, professor of forest pathology, Botany and Plant Pathology Department, Oregon State University; and Jerry Beatty, forest pathologist, USDA Forest Service, Troutdale, Oregon.

Contents

Introduction

Good forest health is a condition that implies a resiliency to natural disturbances. Disease is one of many natural disturbances that can affect forest health. Whereas forest health involves the condition of populations of trees and other associated organisms, tree health is a status of individual trees. A tree with poor health does not mean that the forest is unhealthy, but poor forest health implies that a majority of trees are unhealthy.

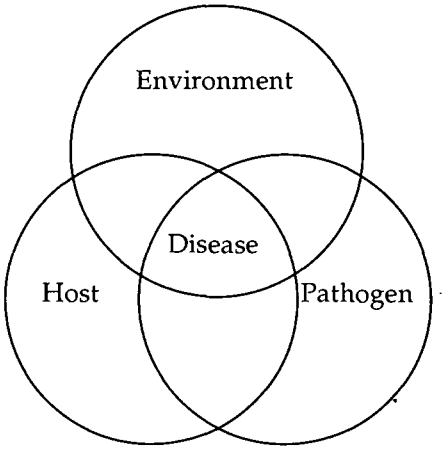
While forest health is a concept relating to forested landscapes, tree health is more appropriate for intensively cultured populations of trees such as in nurseries, Christmas tree plantations, or urban settings. The term tree health also applies to valuable groups of native trees found in developed recreation areas and to nonnative ornamental trees.

Definition and causes of disease

Disease can be defined as "a sustained disturbance to the normal function or structure of a tree as provoked by biological (biotic), chemical, or physical (abiotic) factors of the environment." A pathogen is an entity, usually biological (such as a fungus), that can cause disease. But disease does not simply result from the meeting of a pathogen and a tree. Disease is the product of three interacting factors: the host (tree), the pathogen, and the environment. The relationship among these factors has been depicted as the disease triangle (Figure 1).

If any one of the three factors is missing or unfavorable, disease will not occur. For example: A susceptible tree species is present, and spores from a canker fungus have been released, but there is no wound on the tree for the spores to infect. In this case, the environment for infection is not proper—there is no wound—so disease does not occur even though the host and fungus are present.

Figure 1.—The disease triangle.



Agents that cause disease usually are divided into two groups: nonliving (abiotic) or living (biotic). Abiotic diseases are caused by nonliving factors such as water stress, temperature extremes, chemicals, or soil compaction. Biotic diseases are caused by living organisms such as fungi or parasitic seed plants (mistletoes). Although nematodes, bacteria, and viruses can cause serious plant diseases, they rarely are involved in tree diseases in Oregon.

Important disease groups

There are several economically and ecologically important tree diseases that usually are grouped by the part of the host they affect. These include root diseases, stem decays and diseases, and foliage diseases. Other diseases include rusts, mistletoes, and abiotic diseases.

Diseases of hardwood or deciduous trees have not received much attention in Oregon. This situation is changing as species previously considered "noncommercial" or "weeds" gain commercial or ecological value.

Effects of biotic and abiotic factors on disease

Once a disease is present in a forest or a tree, the course of the disease can be altered by other biotic or abiotic factors. For instance, a pathogen such as dwarf mistletoe successfully attacks and becomes established in the branch of a Douglas-fir. A second biotic agent, a butterfly larva, feeds on the mistletoe plants, causing them to die. This is what is termed "biological control." In another case, a root pathogen infects the roots of a ponderosa pine tree and weakens the tree. Disease occurs but does not kill the tree. Then a serious drought occurs, and the weakened pine tree dies.

Several other biotic or abiotic factors can affect disease. Wind by itself or in combination with snow and ice can result in changes to already diseased trees. Wind can topple trees with root disease more easily, because decayed major roots are insufficient for support. Wind can snap trees off at places where stem decay is present in tree trunks. This may kill the tree, depending on where the trunk snaps and how much live crown is left. Wind can break branches with dwarf mistletoe brooms, especially if the abnormally large branches are loaded with snow and ice.

Insects often are associated with tree diseases. Probably the best example is the relationship between root disease and bark beetles (Figure 2). Root disease weakens trees, predisposing them to bark beetle attack. Some root pathogens and stem-decay fungi actually are spread by bark beetles. Many bark beetles carry spores of fungi that cause black stain root disease. These fungi infect trees, make the trees more suitable for beetle colonization, and provide better habitat for beetle reproduction.

Figure 2.—Galleries of the bark beetle *Scolytus ventralis*, the fir engraver, under the bark of a killed grand fir.

Biology of fungi that cause disease

The most common living agents that cause tree diseases are fungi. Fungi can infect needles, leaves, cones, stems, and roots—essentially all parts of a tree. Fungi do not have chlorophyll and therefore cannot make their own food. They have a mycelial (threadlike) growth form and can reproduce by microscopic spores. The individual microscopic threadlike filaments of all fungi are called hyphae (Figure 3). Hyphae are the basic units of fungi. A mass or collection of hyphae is called mycelium. When hyphae aggregate into a specialized structure for producing spores, the structure is called a fruiting body or sporophore. Mushrooms and conks are fruiting bodies. The fruiting

bodies produce microscopic spores that are the principal reproductive units of fungi. They are produced on hyphae and are dispersed via wind, water, or insects. Under the proper conditions, spores germinate and produce new hyphae.

Fungi obtain nourishment through hyphae that penetrate within and among cells of the host plant. The hyphae secrete enzymes that dissolve all or part of the tissues they have penetrated. The dissolved material then diffuses into the hyphae where it is converted to usable energy for normal cell processes and growth of the fungus. Many fungi in the forest are beneficial and play a major role in nutrient cycling by decomposing wood and organic matter. However, some fungi obtain food from living trees and in the process injure, deform, weaken, or kill the tree. The result is disease. How we evaluate the disease—as being beneficial or detrimental—may depend on the management objectives for the tree (such as fiber production, nutrient recycling, or wildlife habitat) and for the forest. For example, wood decay aids nutrient recycling but is detrimental to fiber production.

Figure 3.—Hyphae of a wood-invading fungus in a wood cell (magnified 600 times).

Root Diseases

Root diseases are the most difficult group of diseases to identify, quantify, and manage in Oregon's trees and forests. Root diseases can be caused by abiotic factors such as flooding or soil compaction, but the most important root diseases are caused by fungi. These fungi attack and destroy the tree's root system, resulting in growth retardation, decay, death, or windthrow of infected trees. Trees affected by root disease also have increased susceptibility to insect pests, especially bark beetles.

Identification

Root diseases usually are inferred by groups of dead, dying, and windthrown trees called disease patches or centers or canopy gaps. These groups of trees become progressively larger over time as the disease-causing fungi spread from tree root to tree root. The result is altered areas in the forest that can range in size from a few trees to hundreds of acres.

Trees in a disease patch show a progression of symptoms; they do not die at the same time, nor will they be in the same stage of decline. Some near the middle of the disease patch are dead, while those at the edge may show only a slight reduction in height or slight yellowing of the foliage (chlorosis). In contrast, groups of trees killed by insects or fire usually are in the same stage of decline, indicating that all trees died at about the same time.

Trees affected by root diseases can be recognized by several symptoms and signs (Table 1, page 6). Symptoms are the reaction of the host tree to the disease. Signs are the actual parts of the pathogen present or near the diseased tree.

Most root diseases cause foliar yellowing and thinning of the crown. These symptoms result from destruction of the root system and the subsequent reduced supply of water and nutrients to the foliage. Abundant undersized cones, called a distress cone crop, often are produced by trees in advanced stages of decline. Trees with these symptoms usually die within 1 or 2 years.

Crown symptoms can reliably indicate root diseases, but they are not sufficient to distinguish among specific root diseases. This can be done only by examining roots. Accurate identification is extremely important because management prescriptions may vary depending on which root disease is present.

Host susceptibility

Susceptibility to infection and to associated damage by root pathogens varies with tree species (Table 2, pages 8 and 9). Susceptibility is defined as the likelihood that a tree species will become damaged if it contacts inoculum of the root disease fungus. Damage susceptibility is rated on a scale of 1 to 4. These ratings are based on field observations in Oregon. The susceptibility of some tree species (especially hardwoods) to some fungi are unknown. Hardwoods are not affected by laminated root rot, black stain root disease, or Port-Orford-cedar root disease, three of the five most important root diseases in Oregon. This is why hardwoods are recommended to plant or, if already present, to favor in many root-diseased areas.

Table 1.—Symptoms and signs of five important root diseases in Oregon.[1]

Symptoms	Laminated root rot	Armillaria root disease	Annosus root disease	Black stain root disease	Port-Orford-cedar root disease
Reduced height	✓	✓	✓	✓	
Crown thinning	✓	✓	✓	✓	✓
Yellow foliage	✓	✓	✓	✓	✓
Distress cones	✓	✓	✓	✓	
Basal resin (sap)		✓		✓	
Brown stain in inner bark					✓
Black stain in sapwood				✓	
Laminated decay	✓		✓		
Yellow, stringy decay		✓			
Signs					
Mycelial fans		✓			
Leathery conks			✓		
Mushrooms at base		✓			
Setal hyphae	✓				
Ectrotrophic mycelium	✓		✓		

[1]From Hadfield, et al.,1986.

Laminated root rot

From the standpoint of wood fiber production, laminated root rot is the most damaging root disease in Oregon. It is found in both western and eastern Oregon. The disease is caused by the fungus *Phellinus* (=*Poria*) *weirii*.[2] It affects all conifer species to some degree but is most damaging to Douglas-fir, grand and white fir, and mountain hemlock. Hardwoods are immune to this disease.

The disease affects trees by decaying their root systems, causing death, growth loss, and windthrow. One of the best indicators of the disease is the presence of root balls, which form when roots decayed by *Phellinus* break near the root collar leaving an abnormally small "ball" of roots on fallen trees. In contrast, healthy trees blown over by wind have a large mat of mostly undecayed roots.

Wood decayed by *Phellinus* characteristically separates into sheets along annual growth rings, hence the name "laminated" root rot (Figure 4). The decayed wood is pale yellow-brown with numerous small oval holes or pits on both sides of the wood sheet. The pits appear similar to shot holes and are about 1 millimeter in diameter. A very close examination of the decayed wood with a hand lens reveals

[2] As scientific research advances, organisms' names and classifications are continually changing. In this publication, the current scientific name for an organism is the first one given; sometimes, former names also are given, as "(=*Poria weirii*)," for example.

Figure 4.—The laminated root rot fungus causes wood to separate at the annual rings.

the key diagnostic indicator of laminated root rot: setal hyphae. Setal hyphae are small, wiry, reddish-brown hairs found between sheets of decayed wood. The presence of setal hyphae is positive proof of laminated root rot.

Another good indicator of laminated root rot, especially on young living trees, can be found on the surface of roots after the soil has been scraped away. The surface of healthy conifer roots is reddish-brown. In contrast, roots infected with *Phellinus* are covered with a white to grayish crust of fungal growth called ectotrophic mycelium. The growth occurs only on the outside of the root, not in the wood or between the bark and wood. Setal hyphae also may be seen mixed with ectotrophic mycelium on the root surface.

Armillaria root disease

Armillaria root disease is the most widespread root disease in Oregon and the most damaging to hardwoods, especially Oregon white oak. This disease also affects hardwood and fruit trees throughout the world. The species of *Armillaria* affecting most hardwoods has not been determined, although *Armillaria bulbosa* has been reported on Oregon white oak.

Armillaria root disease is caused by the fungus *Armillaria ostoyae* (=*Armillaria mellea* or *Armillaria obscura*). At least two other species of *Armillaria* have been identified in Oregon, but these species are mostly saprophytes that decay dead wood. In western Oregon, the disease commonly affects small groups of 10- to 30-year-old trees, and all tree species are affected to some degree. East of the Cascades and in southern Oregon, Armillaria root disease is more widespread and economically damaging.

Trees infected by *Armillaria* usually produce a flow of pitch or sap just above ground level. This flow is the tree's response to the fungus growing beneath the bark. Extensive growth of the fungus beneath the bark eventually girdles and kills the tree. Chopping into the bark reveals white to cream-colored sheets of fungus called mycelial fans. Fans occur inside the bark—never outside—in the lower main stem of the tree, close to the ground, or in roots. Fans have a rubbery texture and sometimes can be peeled from the wood like latex paint. They are a positive indicator of Armillaria root disease. *Armillaria* mushrooms occasionally appear in autumn and can be a useful indicator of the disease. Mushrooms also serve as food for humans and wildlife.

Table 2.—Relative susceptibility of Oregon trees to damage by root diseases.[3]

Tree species	Laminated root rot	Armillaria root disease	Annosus root disease	Black stain root disease	Other root diseases
Conifers					
Cedar					
Alaska-	3	2	3	4	Port-Orford-cedar root disease
Incense-	4	3	3	4	
Port-Orford-	4	3	3	4	Port-Orford-cedar root disease
Western red	3	2	3	4	Yellow root rot
Fir					
Coastal Douglas-	1	2	3	1	Schweinitzii root/butt rot
Inland Douglas-	1	1	3	2	Schweinitzii root/butt rot
Grand or White	1	1	1	4	Schweinitzii root/butt rot
Noble	2	2	2	4	Schweinitzii root/butt rot
Pacific silver	2	2	1	4	Schweinitzii root/butt rot
Shasta red	2	2	2	4	Schweinitzii root/butt rot
Subalpine	2	2	2	4	Schweinitzii root/butt rot
Hemlock					
Mountain	1	2	1	3	Yellow root rot
Western	2	2	2	3	Schweinitzii root/butt rot
Juniper	4	2	3	4	
Larch	2	3	3	4	Schweinitzii root/butt rot
Pine					
Jeffrey	3	2	2	2	Schweinitzii root/butt rot
Knobcone	3	2	2	3	Schweinitzii root/butt rot
Limber	3	2	3	4	Schweinitzii root/butt rot
Lodgepole	3	2	2	3	Schweinitzii root/butt rot
Ponderosa	3	2	2	2	Schweinitzii root/butt rot
Sugar	3	1	3	4	Schweinitzii root/butt rot
Western white	3	2	3	4	Schweinitzii root/butt rot
Whitebark	3	2	3	4	Schweinitzii root/butt rot
Redwood	4	3	3	4	
Spruce					
Brewer	4	?	3	4	
Engelmann	2	2	3	4	Tomentosus root rot
Sitka	3	2	3	4	Tomentosus root rot
Yew	4	3	?	4	Port-Orford-cedar root disease

Table 2.—continued.

Tree species	Laminated root rot	Armillaria root disease	Annosus root disease	Black stain root disease	Other root diseases
Hardwoods					
Alder	4	3	3	4	
Ash	4	?	?	4	
Aspen	4	2	?	4	Ganoderma rot
Birch	4	3	?	4	Ganoderma rot
Buckthorn	4	?	?	4	
Cherry	4	3	?	4	
Chinkapin	4	3	?	4	Ganoderma rot
Cottonwood	4	2	3	4	Ganoderma rot
Dogwood	4	3	?	4	
Madrone	4	3	2	4	
Maple	4	3	3	4	Ganoderma rot
Myrtlewood	4	?	?	4	Ganoderma rot
Oak					
California black	4	3	3	4	
Oregon white	4	2	?		
Tanoak	4	2	?	4	
Willow	4	3	?	4	

[3] From Hadfield, et al., 1986.
1 = severely damaged 2 = moderately damaged 3 = seldom damaged 4 = not damaged

Black stain root disease

Black stain root disease is unique among the root diseases affecting Oregon's forests because it is spread by root-feeding insects. There are two forms of the disease in Oregon: one that attacks fir and one that attacks pine. This disease is caused by the fungus *Leptographium wageneri* (=*Verticicladiella wageneri* or *Ophiostoma wageneri*). The disease affects mainly young (10- to 30-year-old) Douglas-fir. In eastern Oregon, ponderosa pine is affected at any age. Other conifers seldom are affected.

Black stain is a wilt disease, similar to Dutch elm disease, that kills trees rapidly by plugging the water-conducting tubes of the root wood. Black stain is most severe, to the point of being epidemic, in parts of southwestern Oregon; 25 to 50 percent of 10- to 30-year-old Douglas-fir stands in this region have the disease to some extent. In western Oregon, black stain occurs most commonly along roadsides, in stands that have been pre-commercially thinned, and in stands with a history of soil disturbance from tractor logging. It frequently occurs in association with other root diseases.

Like Armillaria root disease, black-stain-affected trees often produce resin at the base of the main stem. Affected trees, however, do not have white mycelium on the root surface or inside the bark. Black stain is diagnosed by chopping into the wood of roots or of the root collar of dying or recently killed trees. Black to brown streaks in the most recent growth rings should be apparent (Figure 5). This stain is the result of the darker colored hyphae growing in the water-conducting tubes of the sapwood. Hyphae plug the tubes and disrupt the water supply to other parts of the tree.

Long-distance spread of the black stain fungus is by insects, mostly root-feeding bark beetles and weevils. The spores of the fungus form in the insect tunnels, stick to the insects, and are spread when the insects fly or walk. The insects feed and breed in low-vigor trees and are attracted to freshly cut stumps. Once the insects introduce the fungus into a tree, the mycelium can spread to other trees across root grafts or by growing short distances through the soil. The fungus does not live long after the tree dies.

Figure 5.—Black stain in an infected ponderosa pine.

Annosus root disease

Annosus root disease is one of the more difficult root diseases to diagnose because it often causes only a butt rot and has signs that are difficult to detect. The disease is caused by the fungus *Heterobasidion annosum* (=*Fomes annosus*). There are two forms in Oregon: an "S-group" that primarily affects fir, hemlock, and spruce, and a "P-group" that chiefly affects pine.

Heterobasidion also causes a stem decay (see page 18). In western Oregon, the fungus causes serious degrade in western hemlock due to decay and stain in the valuable butt log. The disease usually is identified by the appearance of decayed wood that contains silvery white mycelium and small black flecks about the size of rice grains.

Brown leathery conks with white undersides occasionally are found in rotten stumps and among roots of windthrown trees. A conk is a specialized structure produced by wood-decay fungi to disperse spores, which are the chief means of spreading the disease. Spores need freshly cut stump surfaces or fresh tree wounds to infect and colonize the wood. After stumps are infected, the fungus grows through the root system and can infect living trees through root contacts.

In eastern Oregon, annosus root disease is most damaging to true fir and ponderosa pine. Stands partially cut over many years show the most disease. In many areas, groups of pine are killed without substantial decay, especially on very dry sites. A

progression of symptoms usually is present: long-dead trees near the center of the opening and recent kills near the perimeter.

Annosus root disease has been reported on bigleaf maple, red alder, and Pacific madrone. Annosus root disease has not been reported on other hardwoods, but that may be because no surveys have been conducted to determine host ranges.

Port-Orford-cedar root disease

In southwestern Oregon throughout the range of Port-Orford-cedar, the principal host, this disease is very damaging. It is believed that the disease may have been introduced into Oregon, possibly on ornamental cedars. The disease also affects Pacific yew and Alaska-cedar when they grow with Port-Orford-cedar. In some areas, the disease threatens the commercial status of Port-Orford-cedar.

Port-Orford-cedar root disease is caused by the fungus *Phytophthora lateralis*. This fungus causes a discoloration of the foliage from light green to yellow to red and finally to brown. The disease is best identified by the brown-colored inner bark that abruptly joins white, healthy inner bark at the base of infected trees. This symptom is most apparent on trees with yellow foliage.

Roots are infected by special swimming spores called zoospores that are produced by the fungus. Zoospores spread downslope or downstream in water. Tree mortality often is seen within riparian zones or along roadsides, especially the downhill side. The disease spreads mainly by human activity; infested soil on machinery, tires, or on transplanted seedling roots can spread the fungus from one site to another. The disease can spread upslope through root contacts.

Other root diseases

Schweinitzii root and butt rot is caused by *Phaeolus schweinitzii*, often called the velvet-top fungus. It is one of the most important causes of butt decay in old-growth trees, especially Douglas-fir (Table 3, page 16). Sometimes it will kill young trees. Infected trees usually show no above-ground symptoms; the only reliable way to diagnose the disease is by the red-brown conks that are produced on or near the tree base. When these conks are older, they resemble "cow-pies." This disease often is associated with trees that have old fire scars, and the fungus has been associated with nesting cavities of woodpeckers in larch.

Tomentosus root rot, caused by the fungus *Inonotus tomentosus*, is the most important root disease of Engelmann spruce in Oregon. It also causes substantial stem decay. The best way to identify the disease is by the mushrooms. The fungus forms small (1 to 3 inches in diameter) yellow to rusty brown tough and leathery mushrooms. They are produced during wet periods in the autumn. Spores can infect tree roots, but most infection occurs as a result of root-to-root spread from one tree to another. Infected trees will not die until they have been infected for several years. Infected trees often are windthrown.

Other root diseases of conifers include **yellow root rot** caused by *Perreniporia subacida*. Other root and butt rots reported on Oregon hardwoods include **Ganoderma rot** caused by *Ganoderma applanatum* on bigleaf maple, black cottonwood, quaking aspen, birch, and myrtlewood, *Ganoderma oregonense* on chinkapin, and *Inonotus dryadeus* on bigleaf maple and tanoak.

Ecologic roles

The ecologic roles of root diseases in undisturbed forests in Oregon have not been well studied. Much of what we know is mainly from observations. Because root diseases can cause widespread mortality in many forest ecosystems, they are important in creating gaps in the forest canopy. These gaps or openings change the light, moisture, and temperature in the forest and thus change the habitat for plants and animals. Some plants and animals require more light than others and thus are favored by these openings. Other plants and animals favor the edges of the openings. Still other plants and animals prefer closed canopies. Root diseases provide a variety of openings within the forest and thus create habitat for a wide variety of organisms.

Many animals and birds require dead standing trees (snags) and down trees for their habitat. Because root diseases kill trees of all sizes, snags are continually formed as the disease progressively kills trees over many decades. Root-disease-killed trees often windthrow because of decayed root systems, and these fallen trees provide habitat for a different set of animals and plants than did the snag.

Many of the root pathogens continue to live in roots and stumps for 50 or more years after trees have died or been harvested. These root pathogens continue to decay the stump and major roots, thus returning nutrients to the soil and providing habitat for a wide variety of animals.

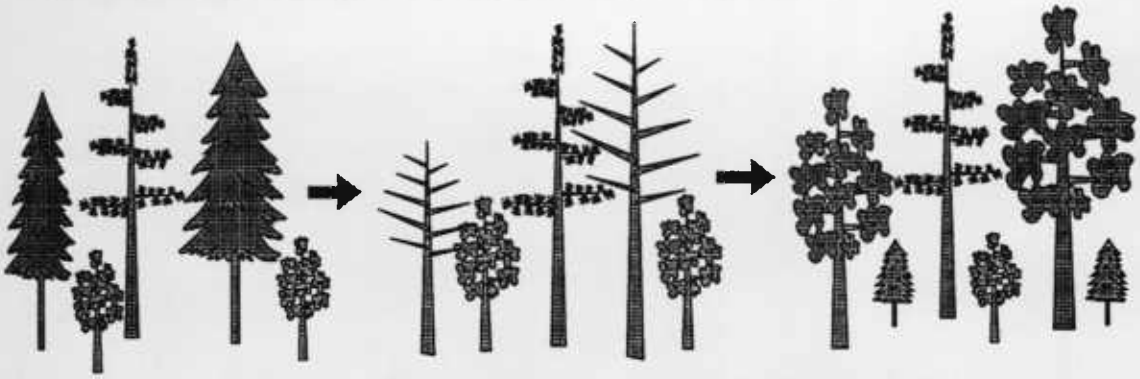
Root diseases also affect the successional pattern of forest development. After a disturbance—either natural, such as a fire, or artificial, such as a clearcut—forest vegetation goes through a predictable series of species changes until the next disturbance. This is called forest succession. Root diseases can affect forest succession by selectively killing certain tree species (Figure 6). For example, in coastal forests, Douglas-fir commonly regenerates sites after disturbance and can live for 500 or more years. Western hemlock will grow in the understory of these Douglas-fir forests but will not attain full height until the overstory of Douglas-fir is killed. Laminated root rot is one of the few biological agents that kills Douglas-fir. Because western hemlock is more resistant to laminated root rot than Douglas-fir, hemlock survives in openings created by root disease and eventually becomes the dominant tree species, or climax species. The rate at which the laminated root rot spreads and kills the Douglas-fir determines the rate at which the hemlock becomes dominant.

Management

Management of root diseases is based on two important factors. The first is the means of spread. All root diseases spread by root contacts when the causal fungus grows from an infected root of a tree or stump to a healthy tree. In general, disease patches expand radially about 1 to 2 feet a year this way. One strategy to stop spread is to break the chain of root contacts between healthy and infected trees by cutting the "bridge tree." However, it is difficult to determine whether a tree is healthy or infected if there are no aboveground symptoms; in other words, it's hard to tell where the "bridge" is if you can't see the water.

Fungal survival is the second factor. Many root-disease fungi can survive in roots for decades after infected trees have died. If a diseased stand is harvested and replanted without considering the disease, seedlings eventually will become infected. Damage in the new stand may be worse than in the preceding stand. One exception to long-term survival in roots is black stain root disease; the causal fungus

Figure 6.—Disease pathogens can either advance or reverse forest succession by selectively killing certain tree species. This illustration shows succession reversal.



Mixed conifer type of forest, with grand fir, western larch, and ponderosa pine.

Grand fir killed by interactions of root disease *(Armillaria* spp.), western spruce budworm, and fir engraver beetle.

Mixed conifer type of forest, dominated by seral western larch and ponderosa pine.

dies within 1 to 2 years of tree death. Because this disease is spread by bark beetles and weevils, management aims at thinning stands soon after bark beetles have completed their seasonal flight, usually after July in most areas.

Based on our understanding of how root-disease fungi spread and survive in roots, the preferred management approach is to take advantage of the differences in tree species' susceptibility to root diseases (Table 2, pages 8 and 9). By planting resistant species or by favoring them during thinning or partial cutting, root disease losses can be greatly reduced. Some root diseases seriously affect only certain tree species; for example, tomentosus root rot on spruce, Port-Orford-cedar root disease on Port-Orford-cedar, and different forms of black stain root disease on Douglas-fir and on pine. Armillaria root disease, laminated root rot, and annosus root disease can cause severe mortality in several tree species, especially grand fir, white fir, and Douglas-fir.

Disease-tolerant and disease-resistant species (damage classes 3 and 4 in Table 2) can be favored during a variety of silvicultural operations including planting, precommercial thinning, commercial thinning, prescribed burning, and seed-tree, shelterwood, and group-selection harvest systems. If tolerant or resistant tree species are planted or regenerated for 50 or more years and ingrowth of more susceptible species (damage classes 1 and 2) can be periodically removed, root disease fungi should die out over most of the infected area. Subsequent rotations of susceptible species can be grown with little probability of reinfection. If tree species in damage class 2 are used, many trees will become infected but at levels less than if tree species in damage class 1 had been grown. Planting or favoring hardwood species, especially on sites affected by laminated root rot, can greatly reduce inoculum on the site.

Incidence of root disease on an infected site will decrease with time, even in highly susceptible tree species, if operators remove or chemically treat the principal inoculum sources in infected areas, primarily the infected stumps from harvested or dead trees. The effects of stump treatments on root disease are discussed on pages 47-48. Many stump treatments can be used during commercial thinning, mini-clearcuts, and group selections, and when favoring resistant species and cutting bridge trees.

Stem Decays

More than 25 percent of disease-caused losses in timber values in Oregon result from decay in living, often overmature, trees. Wood decay is caused by various species of fungi that enter trees through wounds or stubs of small branches. Decay fungi usually do not kill trees, and small amounts of decay will not significantly affect tree growth. However, decay greatly diminishes the value of forest products. In addition, decayed trees are structurally weakened and are likely to break during windstorms or harvesting operations. Decayed trees can become serious hazards when located near buildings or developed recreation areas. On the positive side, decay of living trees is a natural forest process that recycles nutrients and creates important wildlife habitat as standing trees and as down logs both on land and in streams.

Many decays can be recognized by the presence of conks on the stem of the tree. Conks usually indicate considerable wood decay behind the conk. In general, the more conks or the bigger the conk, the larger the amount of decay. The amount of decay associated with conks (and other external indicators) varies among the different species of wood-decay fungi and among different tree species.

General principles concerning stem decays

Each stem-decay disease has its own characteristics, but the diseases in general have the following characteristics in common:

- The amount of decay increases with frequency of tree wounding. Wounds both activate dormant infections and provide entry courts for spores.
- The amount of decay increases with wound size and age. In trees of the same size and age, basal wounds will have more decay than upper-stem wounds.
- The amount of decay increases with tree age and diameter, assuming diameter is directly proportional to age.
- Live trees "compartmentalize" decay; that is, decay columns will not exceed the diameter of the tree at the time it was wounded unless additional wounding takes place.
- The amount of decay is greater in nonresinous tree species such as true fir, hemlock, and hardwoods. Resinous species such as pine, Douglas-fir, and larch are more resistant to decay.
- The amount of decay is influenced by tree genetics: some trees within a species are more resistant than others to decay, all other factors being equal.
- Decay may be caused by a single species of decay fungus, but infections by two or more species are common.
- The percentage of tree volume that is decayed is less in trees that have been thinned and/or fertilized than in trees in unmanaged stands.

Indian paint fungus

The Indian paint fungus (*Echinodontium tinctorium*) is responsible for nearly 80 percent of the decay in old-growth grand fir and white fir in eastern and southern Oregon. Other species affected include mountain hemlock, western hemlock, noble fir, Shasta red fir, Pacific silver fir, subalpine fir, and rarely, Douglas-fir and Engelmann spruce. The incidence of Indian paint fungus appears to be decreasing as old-growth forests are replaced by younger forests.

The most conspicuous sign of the Indian paint fungus is the conk, which is large

Table 3.—Relative susceptibility of Oregon trees to damage by stem decays.

Host tree species	Indian paint fungus	Red ring rot	Schweinitzii butt rot	Brown crumbly rot	Gray-brown sap rot	Other decays
Conifers						
Cedar						
Alaska-	4	3	3	3	4	Brown-cubical butt rot
Incense-	4	3	3	3	3	Pecky rot
Port-Orford-	4	3	4	4	3	
Western red	4	3	3	3	4	Brown-cubical butt rot
Fir						
Douglas-	3	1	1	1	1	Yellow-brown top rot
Grand or White	1	1	1	1	1	Annosus decay
Noble	2	2	2	1	2	Annosus decay
Pacific silver	1	1	1	1	1	Annosus decay
Shasta red	2	2	2	1	2	Annosus decay
Subalpine	2	2	2	1	2	Annosus decay
Hemlock						
Mountain	1	1	2	1	2	Annosus decay
Western	3	2	2	1	2	Annosus decay
Juniper	4	4	4	4	4	Juniper pocket rot
Larch	4	2	1	1	2	Brown trunk rot
Pine	4	2	2	2	2	Brown trunk rot
Redwood	4	4	4	4	4	
Spruce	3	2	2	1	2	Annosus decay
Yew	4	3	3	4	4	
Hardwoods						
Alder	4	4	4	2	4	Hardwood trunk rot
Ash	4	4	4	4	4	
Aspen	4	4	4	2	4	
Birch	4	4	4	3	4	Hardwood trunk rot
Buckthorn	4	4	4	4	4	Hardwood trunk rot
Cherry	4	4	4	4	4	Hardwood trunk rot
Chinkapin	4	4	4	4	4	Hardwood trunk rot
Cottonwood	4	4	4	2	4	Hardwood trunk rot
Dogwood	4	4	4	4	4	Hardwood trunk rot
Madrone	4	4	4	4	4	Hardwood trunk rot
Maple	4	4	4	2	4	
Myrtlewood	4	4	4	4	4	
Oak	4	4	3	4	4	Inonotus trunk rot
Tanoak	4	4	4	4	4	
Willow	4	4	4	4	4	Hardwood trunk rot

1 = often infected 2 = occasionally infected 3 = seldom infected 4 = not infected

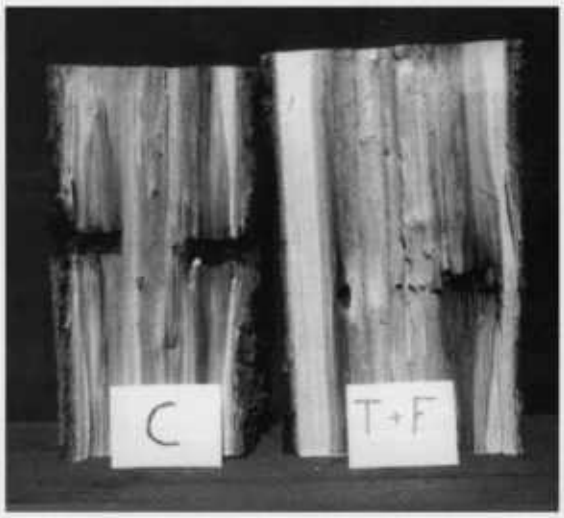
and woody with a black, cracked upper surface, a gray, toothed, lower surface, and a brick-red interior. Native Americans used the red interior for paint. Conks produce spores throughout the year but mostly in the spring and fall. Spores do not infect mechanical wounds or old branch stubs as was once thought. Instead, spores infect small (less than 2 millimeters in diameter) exposed branchlet stubs just before the stubs are overgrown. Trees or branches whose growth has been suppressed seal branchlet stubs very slowly, thus allowing more time for infection. Once branchlet stubs are overgrown, the fungal mycelium from germinated spores enters a dormant state, which can last for 50 or more years without causing decay.

Dormant infections are activated immediately by mechanical injuries, frost cracks, or formation of large branch stubs that allow air to enter the trunk interior. The larger the injury, the more likely that one or more dormant infections will be activated and cause decay. Decay first appears as elongated areas of wood that are stained light brown or yellow. Advanced decay appears yellow to reddish-yellow and fibrous or stringy. Extensive decay columns may occur after several dormant infections become active, cause decay, and subsequently coalesce. After extensive decay, conks are produced, often at old branch stubs or wounds. When conks are single and small, decay usually extends 8 feet above and below the conk. When there are two or more conks, decay can extend 20 feet above the highest and 20 feet below the lowest conk on the bole.

Red ring rot

Red ring rot is caused by the fungus *Phellinus pini*. It is the most common cause of stem decay in Douglas-fir in westside forests and affects several

Figure 7.—Wood decay is caused by fungi that enter trees through wounds or small branches as shown here in artificially infected stems of 85-year-old white fir. The specimen on the left is a control tree that received no treatment. The specimen on the right is a tree from a stand that was thinned and fertilized.



conifer species in eastside forests (Table 3). A related fungus, *Phellinus cancriformans*, causes sunken cankers on white fir in southern Oregon.

The fungus forms hoof-shaped to bracketlike perennial conks on stems that often occur at knots or branch stubs. The upper surface of the conk is rough, dull grayish to brownish. The lower surface is a rich brown color with small circular to large sinuous openings. Decay in the early stages varies by tree species. In the late stages, decay appears as spindle-shaped, white pockets with firm wood in between ("white speck"). Decay may be spread uniformly but often forms crescents or rings. Swollen knots or punk knots may form on tree boles. The extent of decay varies with tree age and species; for example, decay is 4 feet above and below the conks for Douglas-firs of 150 years, and 22 feet above and below the conks for 350-year-old trees. Decay extent is less for other tree species.

Although wounds, either natural or human-caused, and branch stubs provide openings for infection by other decay fungi, we do not know how *Phellinus pini* enters trees. Conks are closely associated with old dead branch stubs. It is believed that the fungus does not spread downward through the dead and dying branches but rather that the fungus infects the protective wood in the trunk that forms after branches die. The fungus has no competition from other organisms in the resin-soaked protective wood and continues to grow there as a canker-type fungus. The crescent or ring-type decay pattern allows the tree trunk to retain enough integrity that it does not break, and at the same time allows the fungus to exist in a living tree.

Brown crumbly rot

Brown crumbly rot is caused by the fungus *Fomitopsis pinicola*, commonly called the redbelt fungus. This is the most common cause of decay of dead woody material in Oregon (Table 3). Conks occasionally occur on dead portions of living trees, especially Sitka spruce. They usually are produced at least 2 years after tree death. The conks are hard, woody, perennial, shelf- to hoof-shaped (2 to 10 inches wide), and have a smooth gray to black upper surface often with a wide red margin. The undersurface is white to yellowish.

One way the disease spreads is through windborne spores released from the conks. The fungus also has been isolated from the Douglas-fir beetle, which may transmit the fungus while attacking dying trees. The fungus causes a brown cubical rot of the sapwood and heartwood. Small logs, which are mostly sapwood, can completely decay in 10 years, whereas large logs with mostly heartwood may take 30 or more years for complete decay.

Gray-brown sap rot

Gray-brown sap rot is caused by the fungus *Cryptoporus volvatus*, often called the pouch fungus. The fungus causes a soft grayish sap rot of slash and dead trees. The conks occur on trees that have been dead for 12 to 18 months. They occur on most conifers but are most common on beetle- or fire-killed Douglas-fir, true fir, and ponderosa pine. The conks are white to tan, leathery, round, and annual. The brown pore layer is completely enclosed by a leathery membrane. The fungus has been isolated from bark beetles, which may aid in spreading the disease.

Other stem decays

There are many other stem decays of conifers and hardwoods (Table 3).

- **Annosus decay**, caused by *Heterobasidion annosum* (which also causes annosus root disease) can be very damaging to true fir, hemlock, and spruce.
- **Brown trunk rot**, caused by *Fomitopsis officinalis* or the quinine fungus, occurs in old-growth Douglas-fir, larch, and pine, especially trees with broken tops or other wounds.
- **Brown cubical rot** is caused by *Laetiporus sulfureus* or the sulfur fungus, which is edible. The rot occurs on most conifers and on oak.
- **Brown-mottled white rot** is caused by *Pholiota limonella*, or the yellow-cap fungus, and occurs on several conifers and hardwoods.
- **Dry pocket rot** or **pecky rot**, caused by *Oligoporus amarus*, is the most important stem decay of incense-cedar in Oregon.
- **Brown cubical butt rot**, caused by *Oligoporus sericeomollis*, is the most damaging decay of Alaska-cedar and western redcedar.

- **Juniper pocket rot**, caused by *Pyrofomes demidoffii*, is the chief stem decay of living juniper.
- **Ganoderma rot** is found in many conifers and hardwoods and is caused by *Ganoderma applanatum*, the artist's conk.
- **Yellow-brown top rot**, caused by *Fomitopsis cajanderi*, is the most common decay affecting young, broken-topped Douglas-fir.
- **Yellow pitted rot** is caused by *Hericium abietis* or the coral fungus, which is edible. It attacks fir, spruce, and hemlock.

Many species of fungi cause decay in living hardwoods; other species are found only on dead trees, dead portions of trees, or in stumps. Probably the most important heart rot fungus of Oregon hardwoods is **hardwood trunk rot**, caused by *Phellinus igniarius*. **Inonotus trunk rot** occurs on oak and is caused by *Inonotus dryophilus*.

Many other species of decay fungi have been reported on only one hardwood species. For example:

- *Bondarzewia berkeleyi* and *Oxysporus populinus* on bigleaf maple
- *Sistrotrema brinkmannii, Pholiota adiposa, Trametes* sp., and *Meruliopsis corium* on red alder
- *Fomitopsis cajanderi* and *Perreniporia subacida* on Pacific madrone
- *Perreniporia fraxinophilus* on Oregon ash
- *Fistulina hepatica* on tanoak
- *Collybia velutipes* and *Phellinus tremulae* on quaking aspen
- *Spongipellis delectans* and *Pholiota destruens* on black cottonwood
- *Laetiporus sulfureus, Hydnum erinaceus,* and *Bjerkandera adusta* on California black oak

Ecologic roles

The ecologic roles of stem decays in living trees in Oregon have not been well studied. Much of what we know is mainly from observation.

The living but decayed tree provides habitat for cavity-nesting birds that require a certain degree of wood decay in order to excavate for nesting. The pileated woodpecker in eastern Oregon, for example, requires rotten larch or ponderosa pine for its cavities. Secondary cavity-nesting birds (those that cannot excavate) depend on the primary cavity-nesters such as the pileated woodpecker to make nest sites. Northern spotted owls, and perhaps other owls, use cavities created by decay fungi in living trees. Several mammal species also use cavities vacated by the primary cavity-nesters. Large decay columns and hollows created by the Indian paint fungus in grand fir are used as roosting sites and hiding cover by several bird and mammal species.

Inoculating live trees with fungi to promote decay and create habitat for cavity-nesting birds has been successful in Oregon. The idea is that living, decayed trees should stand longer and provide useful habitat longer than decayed snags.

Stem decays can lead to tree mortality through stem breakage at or below the living crown and therefore may be important in creating small gaps in the forest canopy. As with root-disease-caused gaps, these openings change the light, moisture, and temperature in the forest and thus change the habitat for plants and animals. Some plants and animals require more light than others and so are favored by these openings. Many animals and birds require dead standing trees (snags) and down trees for their habitat. Large woody debris also is important for providing habitat for fish and other animals in rivers and streams and adjacent riparian areas.

The role of stem decay fungi in dead trees or in their broken parts, such as tops and branches, has been well studied. Some stem decay fungi such as *Echinodontium tinctorium* or *Phellinus pini* are adapted to grow in live trees. After those trees die, the stem decay fungi are replaced by other fungi that are more adapted to decaying the wood of dead trees.

Other fungi such as *Cryptoporus volvatus* or *Fomitopsis pinicola*, which infect living trees as they are being killed by other agents, continue to decay wood well after the tree has died. These fungi are important in recycling nutrients to the soil, and the trees they decay provide critical habitat for a variety of plants and animals. For example, ants require decaying logs for habitat, and the ants are an important food source for many bird species. Ants also are important predators of the larvae of defoliating insects such as the western spruce budworm.

Management

Management of stem decays will depend on the objectives for the stand or forest. If timber production is to be optimized, several guidelines will minimize stem decays. Or, if a certain amount of stem decay is desirable both in living trees and as an agent for creating snags and down woody material, then follow the reverse of the following decay-minimizing guidelines.

Manage on short rotations.
Keep rotations short—that is, under 125 years—especially for nonresinous species such as true fir. Stem decay has been shown to increase with tree age.

Do not avoid or delay early thinning out of concern about potential decay losses.
Growth increases due to thinning will outweigh decay losses in most cases. Increased tree vigor will prevent infection by certain decay fungi. Thin early so that if decay columns develop as a result of wounding, they will be relatively small due to compartmentalization.

Select crop trees that are vigorous and undamaged.
Crop trees should be the best in form and height, with good live-crown ratios, good current leader growth, and no wounds or top damage.

When pruning or topping, be sure to make cuts properly.
Cut living or dying branches as close as possible to the branch collar, but do not make flush cuts, remove the branch collar, or leave stubs. All of these may result in stem decay.

Minimize wounding.
Wounds can be prevented when thinning, burning, disposing of slash, or removing the overstory because of potential losses due to decay and other defects associated with injuries. The following actions, taken both in planning and during the actual operation, can prevent decay.

- Avoid spring and early summer logging when bark is soft.
- Match the size and type of operating equipment with topography, tree size, and soil type and condition.
- Mark "leave" trees rather than "cut" trees.
- Plan skid trails before logging.
- Match log length with final spacing.
- Log skid trails first.
- Cut low stumps in skid trails.
- Use directional falling.
- Limb and top trees prior to skidding.
- Remove slash from around crop trees if stands are to be underburned.
- Gain the cooperation of the operator. Explain the effects of tree wounding and ask the operator's help in preventing it.

Rust Diseases

Rust diseases are so named because the fungi that cause them often give a rusty color to infected stems or foliage. Some rust diseases cause abnormal stem swellings called galls; for example, western gall rust of lodgepole pine. Several important rust diseases affect Oregon trees: white pine blister rust, western gall rust, comandra blister rust, stalactiform rust, and several foliage and broom rusts.

Stem rusts

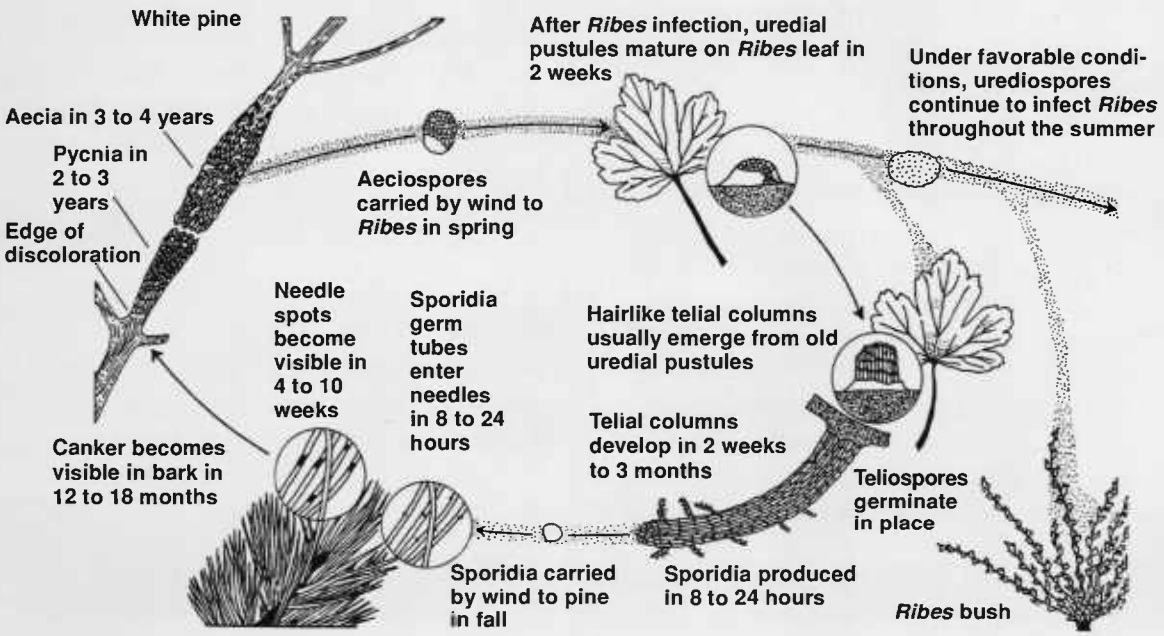
Several rust fungi attack the stems of Oregon pines, true firs, and Engelmann spruce. No other tree species are affected by stem rusts.

White pine blister rust

This is the most destructive stem rust in Oregon. It affects five-needle pines: western white pine, sugar pine, and whitebark pine. It is caused by the fungus *Cronartium ribicola*. Since its introduction from Europe in the early 1900s, white pine blister rust has killed millions of five-needle pines throughout western North America. White pine is desirable because it is highly resistant to laminated root rot and is a preferred species for planting in root disease centers.

White pine blister rust serves as an example of the complex life cycle typical of many, but not all, rust diseases (Figure 8). The disease cycle begins when windborne spores infect the needles. The fungus then grows within the needle and into the main stem where it kills the bark and produces orange to yellow masses of spores. These spores are wind-dispersed and can infect leaves only of *Ribes* (gooseberry and currant plants). These plants are called alternate hosts. The fungus then produces spores on the undersides of *Ribes* leaves. These spores are windblown and, under the right environmental conditions, infect pine needles, completing the cycle. Spores from pine cannot infect other pines; they can infect only *Ribes*, and only spores from *Ribes* can infect pines.

Figure 8.—Life cycle of white pine blister rust *(Cronartium ribicola)*.



Despite early concerns about extinction of white pine, it is once again a viable species provided that disease management is rigorous. Tree improvement programs have developed resistant trees that can tolerate infection by the fungus.

Western gall rust

This disease affects two- and three-needle pines, especially lodgepole, knobcone, and ponderosa pine. The disease is caused by the fungus *Endocronartium (=Peridermium) harknessii*, and is probably the most common disease of lodgepole pine in Oregon. Severe infection causes stem malformation, breakage, and tree mortality, especially in seedlings. The disease causes round to pear-shaped galls on branches or stems. Galls on main stems may continue to grow for years, forming large burls. Yellow-orange spore pustules (aecia) are produced each spring. Spores are windborne and infect other two- or three-needle pines; *no alternate host is required*. Moist conditions stimulate spore release and favor infection on succulent stem tissue. The fastest growing trees are more susceptible than suppressed trees. Moist sites, such as those at the lower edge of a slope, can have extremely high rates of infection.

Comandra blister rust

Like western gall rust, this disease also affects two- and three-needle pines but is more serious on ponderosa pine. The disease gets its name from its alternate host, bastard toadflax (*Comandra umbellata*), which is required to complete the disease cycle. The disease is caused by the fungus *Cronartium comandrae*. It produces spindle-shaped swellings on pine stems and branches. The swellings break open and produce a cracked and pitted canker with abundant resin flow. Dead branches with red foliage and dead tree tops are common. The fungus grows slowly down infected stems causing progressive dying.

Figure 9.—Comandra blister rust has killed the crown of this ponderosa pine.

Tree mortality can occur, especially in seedlings and saplings.

In the spring, dark orange spore pustules (aecia) are produced on the bark of infected pines. The spores are windborne and infect the leaves of bastard toadflax. Infections appear as yellow spots and brown hairlike structures throughout the summer. Pines are infected by spores produced on the toadflax leaves in late summer or fall.

Stalactiform rust

Stalactiform rust affects mainly lodgepole pine and is caused by the fungus *Peridermium stalactiforme* (=*Cronartium coleosporiodes)*. Infections cause stem malformation and breakage but seldom kill the tree. Damage can be severe in very young trees. New infections appear as spindle-shaped swellings on stems and

branches. Older infections appear as diamond-shaped cankers that can reach 30 feet in length. Cankers are resin soaked and yellow. Bark on the cankers frequently falls off, leaving ridges. Yellow spore pustules (aecia) form on the edges of active cankers from May through August. The spores infect the alternate hosts, Indian paintbrush and other members of the plant family *Scrophulariaceae*. Orange spore pustules are produced on leaves of the alternate host, and the spores infect pines in late summer and fall.

Broom rusts

Broom rusts are so called because they cause large, conspicuous, dense branches known as witches' brooms.

Fir broom rust affects true fir and is caused by the fungus *Melampsorella caryophyllacearum*.

Spruce broom rust affects spruce and is caused by the fungus *Chrysomyxa arctostaphyli*.

On both spruce and fir, the needles on broomed twigs are yellow, and the broomed foliage dies in the fall, causing the broom to appear dead in the winter. The brooms often are confused with those caused by dwarf mistletoes; however, the brooms contain no mistletoe plants. Severe broom rust infection results in stem malformation, growth loss, and occasionally tree mortality. The fungi cause a leaf or shoot blight of chickweed (for fir) and a purple-brown leaf spot of kinnikinnick (for spruce), which are their alternate hosts.

White to yellow spore pustules (aecia) occur on foliage of brooms in the summer. Orange to brown spore pustules (uredia and telia) occur on the alternate host's leaves in the spring and summer. Spores are windborne and require moist conditions for infection.

Foliage rusts

Although several species of foliage rust occur in Oregon, their economic importance in native forests is insignificant. However, their impact can be great on agricultural, landscaping, nursery, or Christmas tree businesses.

Needle rusts

Several species of needle rust affect true fir: *Uredinopsis* spp., *Melampsora* spp., *Milesina* spp., and *Pucciniastrum* spp. The fir-fireweed rust, *Pucciniastrum epilobii*, probably is the most damaging needle rust of true fir, causing discoloration and death of current year's needles.

Spruce is affected by two species of needle rust: *Melampsora* spp. and *Chrysomyxa* spp. Infection results in needle discoloration, death, and premature defoliation. Cones also are affected, resulting in damage to seeds.

Pine is affected by two species of needle rust: *Coleosporium* spp. and *Melampsora* spp. Both cause needle discoloration and premature defoliation.

Hemlock is infected by *Pucciniastrum vacinii*, the hemlock-blueberry rust; *Melampsora epitea*, the hemlock-willow rust; and *Melampsora medusae*, the aspen rust.

Douglas-fir and larch are infected by several species of poplar rust—*Melampsora medusae, Melampsora albertensis*, and *Melampsora abietis-canadensis* on quaking aspen; and *Melampsora occidentalis, Melampsora medusae*, and *Melampsora larici-populina* on black cottonwood. The disease causes needle discoloration and death. The fruiting bodies of the rust fungus form on the current year's needles and occasionally on the cones. Several of these rusts have caused severe damage in hybrid poplar plantations.

Table 4.—Selected rust diseases in Oregon trees.

Host tree species	Stem rusts	Broom rusts	Foliage rusts
Conifers			
Cedar	None	None	Incense-cedar rust, Yellow cedar rust
Douglas-fir	None	None	Poplar rust
Fir, true	Fir broom rust	Fir broom rust	Fir–fireweed rust
Hemlock	None	None	Hemlock–blueberry rust, Hemlock–willow rust, Aspen rust
Juniper	None	None	Juniper rust
Larch	None	None	Poplar rust
Pine			
Jeffrey	Western gall rust, Comandra rust, Stalactiform rust	None	Pine needle rust
Knobcone	Western gall rust, Comandra rust, Stalactiform rust	None	Pine needle rust
Lodgepole	Western gall rust, Comandra rust, Stalactiform rust	None	Pine needle rust
Ponderosa	Western gall rust, Comandra rust, Stalactiform rust	None	Pine needle rust
Limber	White pine blister rust	None	Pine needle rust
Sugar	White pine blister rust	None	Pine needle rust
Western white	White pine blister rust	None	Pine needle rust
Whitebark	White pine blister rust	None	Pine needle rust
Spruce	Spruce broom rust	Spruce broom rust	Spruce needle rust, Spruce cone rust
Hardwoods			
Alder	None	None	Alder leaf rust
Aspen	None	None	Aspen rust
Birch	None	None	Birch leaf rust
Buckthorn	None	None	Buckthorn leaf rust
Cottonwood	None	None	Poplar rust
Madrone	None	None	Madrone leaf rust
Oak/Tanoak	None	None	Cronartium rust
Willow	None	None	Willow leaf rust

Incense-cedar often is infected by *Gymnosporangium libocedri*, Alaska-cedar by *Gymnosporangium nootkatense*, and juniper by several species of *Gymnosporangium*. Serviceberry is the alternate host for incense-cedar and juniper rust, and mountain ash is the alternate host for yellow cedar rust.

Leaf rusts

Leaf rusts caused by *Melamsoridium* spp. and *Pucciniastrum* spp. have been reported on red alder. *Puccinia* sp. occur on buckthorn. *Pucciniastrum* spp. have been found on Pacific madrone, *Cronartium* spp. on oak and tanoak, and *Melampsora* spp. on willow, aspen, and cottonwood. Hybrid poplars (black cottonwood X eastern cottonwood) are highly susceptible to leaf rusts caused by *Melampsora larici-populina* (alternate host, larch), *Melampsora medusae*, and *Melampsora occidentalis*. (Alternate hosts for the latter two fungi include several conifer species.)

No rusts have been reported on yew, redwood, western redcedar, Port-Orford-cedar, cherry, dogwood, maple, ash, myrtlewood, or chinkapin in Oregon.

Ecologic roles

White pine blister rust, unlike most other conifer rusts in Oregon, is an introduced disease and therefore has dramatically altered the ecology of the five-needle pines throughout western North America. Because most five-needle pines have not developed resistance to the disease, tree mortality has been so widespread that the five-needle pines are no longer part of the ecosystem in many areas. These areas have succeeded to climax species in many cases. At low elevations, sugar pine has been replaced by white fir. At middle elevations, white pine has been replaced by white or grand fir. At upper elevations, white pine has been replaced by Shasta red fir or Pacific silver fir, and whitebark pine has been replaced by subalpine fir or mountain hemlock. Dead pines provide habitat for birds and mammals as standing snags and as down woody debris.

Ecologic roles of the native rust diseases are much more subtle. Mortality usually is not so widespread as to affect tree species' abundance or to alter forest succession. Individual dead trees or affected tree parts provide habitat for wildlife.

Management

Probably no other forest disease in western North America has received as much attention as white pine blister rust. Millions of dollars were spent to eradicate the alternate host in order to control the disease, but eradication proved impossible. Chemical control also failed. Recent efforts have focused on breeding resistant trees and relying on natural resistance to manage the disease. Pruning infected branches or uninfected lower branches (see page 48) or excising around stem cankers also helps reduce impact of the disease. *Ribes* removal on a small scale, such as in pine plantations or near ornamentals, is useful in reducing disease incidence. Some attempts have been made to rate sites for hazard of rust infection based on presence or absence of various species of *Ribes* plants. Low-hazard sites are planted with susceptible pines, medium-hazard sites are planted with pine of low or questionable resistance, and the most resistant stock is planted in high-hazard sites.

Attempts to control western gall rust have focused on developing genetically resistant stock. For all other rusts, management has focused on removing infected trees during normal silvicultural operations. Foliage rusts can be controlled with chemical sprays in agricultural, nursery, landscape, or Christmas tree settings.

Other Fungal Diseases

Several other tree diseases occur in Oregon that affect the foliage, stems, or entire seedlings. These diseases are important in high-yield forestry, Christmas tree plantations, ornamental landscaping, or seedling nurseries.

Foliage diseases

Several fungi attack the foliage of conifer and hardwood trees (Table 5, page 28). Infected leaves have reduced photosynthetic efficiency and drop from the tree prematurely. The net effect is reduced tree growth and vigor as well as an unappealing appearance. Foliage diseases often are most severe in offsite plantings or following years when wet weather continues through spring into the summer.

Brown felt blight

This disease is caused by two species of fungi: *Herpotrichia juniperi* on conifers other than pine, and *Herpotrichia coulteri* on pine. The disease is common at high elevations. Lower branches are covered with dense cobwebby growths of brown to black mycelium that kill foliage. The fungi develop on foliage under snow where high humidity and relatively mild temperatures provide conditions required for growth. Fruiting bodies form on the mycelium and produce spores that are windborne and infect susceptible foliage.

Rhabdocline needle cast

This disease of Douglas-fir is caused by the fungi *Rhabdocline pseudotsugae* and *Rhabdocline weirii*. Windborne spores are released in May and June from fruiting bodies on the undersurfaces of infected needles. Only the current year's needles are susceptible and they are not cast until the following year. Coastal Douglas-fir is less susceptible than inland Douglas-fir, but considerable variation in resistance occurs within natural stands.

Swiss needle cast

Swiss needle cast of Douglas-fir is caused by the fungus *Phaeocryptopus gaumanni*. It is very common in western Oregon and is especially damaging along the north Oregon coast. Premature needle loss and tree growth retardation are common in severely affected plantations, and mortality occasionally has occurred. Spores released from fruiting bodies on 1-year-old needles in April and May infect the newly emerged needles. Succulent foliage, dense stocking, moist conditions, and offsite plantings favor infection. The disease is recognized by the yellowing and browning in the late winter or early spring of infected previous year's needles shortly before the current year's needles emerge. Casting of the 1- and 2-year-old needles usually begins in the lower portion of the crown and progresses upward.

Elytroderma needle blight

This disease is caused by the fungus *Elytroderma deformans*. It affects ponderosa, Jeffrey, knobcone, and, rarely, lodgepole pine. It causes the 1-year-old needles to turn red in the spring. The disease also affects the twigs and causes a witches' broom with upward-turning branchlets. Windborne spores are produced on infected needles in the spring and infect current year's needles during periods of high humidity and cool temperatures. The fungus grows from infected needles to woody tissues and remains active for many years by infecting new needles. In severely infected trees, entire tree tops may be misshapen, growth retardation occurs, and, occasionally, entire trees die or are predisposed to bark beetles. This disease often is confused with dwarf mistletoe, but *Elytroderma*-broomed branches do not have mistletoe plants.

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Table 5.—Foliage and canker pathogens (except rusts) in Oregon trees.

Host tree species	Foliage pathogens	Canker pathogens
Conifers		
Cedar	*Lophodermium juniperi* *Didymascella thujina*	None
Douglas-fir	*Herpotrichia juniperi* *Rhabdocline* spp. *Phaeocryptopus gaumanni* *Rhizosphaera kalkhoffi*	*Cytospora abietis* *Diaporthe lokoyae* *Dermea pseudotsugae* *Sclerophoma pythiophila*
Fir, true	*Herpotrichia juniperi* *Lirula abietis-concoloris* *Virgella robusta* *Lophodermium decorum* *Phacidium abietis*	*Cytospora abietis* *Grovesiella abieticola* *Nectria fuckeliana*
Hemlock	*Herpotrichia juniperi* *Epipolaeum tsugae*	*Sirococcus strobilinus*
Juniper	*Lophodermium juniperi* *Didymascella thujina*	None
Larch	*Hypodermella laricis* *Meria laricis*	*Lachnellula* sp.
Pine		
Jeffrey, Knobcone, Lodgepole, Ponderosa	*Herpotrichia coulteri* *Elytroderma deformans* *Lophodermella* spp. *Lophodermium* spp. *Mycosphaerella pini* *Davisomycella* spp.	*Atropellis* spp. *Sclerophoma pythiophila*
Limber, Sugar, White, Whitebark	*Herpotrichia coulteri* *Lophodermella* spp. *Lophodermium* spp. *Bifusella* spp.	*Atropellis* spp. *Sclerophoma pythiophila*
Spruce	*Herpotrichia juniperi* *Lophodermium* spp. *Rhizosphaera kalkhoffii*	*Sirococcus strobilinus*
Yew	*Lophodermium juniperi* *Macrophoma taxi* *Mycosphaerella taxi* *Phoma hystrella* *Sphaerulina taxi*	*Diploidia taxi*
Hardwoods		
Alder		
Red	*Cercosporella alni* *Gnomonia alni* *Gnomoniella tubiformis* *Hyposila californica* *Microsphaera alni* *Septoria alnifolia* *Taphrina japonica*	*Didymosphaeria oregonensis* *Hymenochaete agglutinans* *Nectria* spp. *Cytospora* sp.
White	*Gnomonia alni* *Phyllactinia guttata* *Taphrina occidentalis*	*Nectria cinnabarina*

Table 5.— continued.

Host tree species	Foliage pathogens	Canker pathogens
Hardwoods—continued		
Ash	*Apiognomonia errabunda* *Mycosphaerella* spp. *Phyllactinia guttata*	*Cytospora ambiens* *Hysterographium fraxini* *Nectria cinnabarina*
Aspen	*Erysiphe cichoracearum* *Marssonina populi* *Uncinula salicis* *Venturia macularis*	*Ceratocystis fimbriata* *Cryptosphaeria populina* *Cytospora chrysosperma*
Birch	*Cylindrosporium betulae* *Phyllactinia guttata* *Septoria betulicola* *Taphrina* spp.	*Cytospora* sp. *Nectria* sp.
Buckthorn	*Phyllosticta rhamnigena* *Septoria blasdalei*	*Nectria cinnabarina*
Cherry	*Taphrina flectans*	*Nectria cinnabarina*
Chinkapin	*Coronellaria castanopsidis* *Dothidella janus* *Microsphaera alni* *Sphaerulina myriadea* *Taphrina castanopsidis*	None
Cottonwood	*Venturia populina* *Taphrina populisalicis* *Marssonina* spp. *Septoria* spp.	*Cytospora sordida* *Dothichiza populea* *Fusarium* spp. *Hypoxylon mammatum* *Nectria galligena* *Septoria musiva*
Dogwood	*Phyllactinia guttata* *Discula destructiva*	*Nectria* spp.
Madrone	*Ascochyta hanseni* *Cryptostictus arbuti* *Didymosporium arbuticola* *Disaeta arbuti* *Elsinoe mattirolianum* *Exobasidium vaccinii* *Mycosphaerella arbuticola* *Phyllosticta fimbriata* *Rhytisma arbuti*	*Botryosphaeria dothidea* *Hendersonula toruloidea* *Phytophthora cactorum*
Maple	*Microsphaera alni* *Phyllactinia guttata* *Rhytisma* spp. *Uncinula* spp. *Phyllosticta* sp.	*Nectria galligena* *Verticillium albo-atrum*
Myrtlewood	*Botryosphaeria* sp. *Capnodium tuba* *Chaetasbolisia falcata* *Colletotrichum gleosporiodes* *Kabatiella phoradendri* *Mycosphaerella arbuticola* *Phaeosaccardinula* spp. *Vertixore atronitidum*	*Nectria galligena*

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Table 5.—continued.

Host tree species	Foliage pathogens	Canker pathogens
Hardwoods—continued		
Oak	*Gnomonia quercina* *Microsphaera* spp. *Septoria quercicola* *Sphaerotheca* spp. *Taphrina caerulescens*	None
Tanoak	*Capnodium coffeae* *Ceathocarpum conflictum* *Chaestasbolisia falcata* *Limacinia lithocarpi* *Pestolotia castagnei* *Phaeosaccardinula anomola* *Protepeltis lithocarpi*	None
Willow	*Cylindrosporium salicinum* *Rhytisma salicinum* *Septoria* spp.	*Nectria* spp. *Cytospora* spp.

Lophodermella needle casts

These needle casts affect several species of pine in Oregon. The most common are caused by the fungus *Lophodermella morbida* in ponderosa pine, *Lophodermella arcuata* in five-needle pines, and *Lophodermella concolor* in lodgepole pine. New needles are infected by windborne and rain-splashed spores in the early summer. Infected needles turn brown the following year. Damage can be spectacular in offsite ponderosa pine, especially in young or small trees. Onsite trees are not seriously damaged or killed. Lophodermella needle cast is a serious problem on the knobcone-Monterey pine hybrid (the KMX pine).

Lophodermium needle casts

These diseases affect all species of pine in Oregon and are caused by several species in the *Lophodermium pinastri* complex of fungi. Another species, *Lophodermium decorum*, causes a needle cast of true fir. *Lophodermium juniperi* occurs on juniper and incense-cedar, *Lophodermium crassum* on Brewer spruce, and *Lophodermium piceae* on Sitka and Engelmann spruce. The diseases caused by these fungi are quite common but cause little damage except in offsite trees. The fruiting bodies of the fungi appear on needles 3 years old or older. They are dull to shiny black, elliptical structures that occur in rows or lines. Infected needles remain on the tree several years after dying.

True fir needle diseases

Three species of fungi cause needle diseases in true fir: *Lirula abietis-concoloris*, *Virgella robusta*, and *Phacidium abietis* (snow blight). Infections develop on young expanding needles. For *Lirula abietis-concoloris* and *Virgella robusta*, elongate dark brown or black fruiting bodies form in either one or two rows on needles 2 years old or older. *Phacidium abietis* attacks needles of all ages while they are under the snow. In the summer and fall, dark brown oval fruiting bodies appear on the undersides of needles. These diseases are of little economic importance except in ornamental settings, Christmas tree plantations, or nurseries.

Red band needle blight

Red band needle blight is caused by the fungus *Mycosphaerella* (=*Dothistroma*) *pini*. It affects ponderosa, Jeffrey, knobcone, KMX, and lodgepole pine in Oregon. Windborne spores are released from May

to November and can affect needles of all ages. The disease is recognized by yellow to tan spots and bands that appear on needles in July. Infected needles drop in late summer, fall, or in some cases spring of the following year. Twigs may have only a few healthy needles concentrated at the outer ends of branches or near tops.

Davisomycella needle blight

Several species of pine are affected by this blight. *Davisomycella lacriformis* occurs on knobcone pine, *Davisomycella montana* on lodgepole pine, and *Davisomycella medusa* on ponderosa, Jeffrey, and lodgepole pine. This disease can be locally serious, resulting in tree growth retardation. The elongate, black, raised fruiting bodies mature on infected needles in June, and spores are released in the summer and fall. Spores infect the current year's needles. The fungus remains dormant for 2 to 4 years before symptoms appear.

Bifusella needle cast

This disease occurs in the five-needle pines in Oregon. It is caused by several species of fungi in the genus *Bifusella*. The fungus forms shiny, black, elongate fruiting bodies of variable lengths on 2- to 3-year-old needles. Because the disease occurs on older needles, it is not considered serious.

Larch needle blight and larch needle cast

Two common needle diseases of western larch are the needle blight, caused by the fungus *Hypodermella laricis*, and the needle cast caused by the fungus *Meria laricis*. Spores from previously killed needles infect new foliage in the spring during moist weather. Needle cast affects the tips or some other part of the needles; in contrast, needle blight affects the whole needle as if scorched by fire, and all needles on a spur are affected. Needles infected by *Meria* are cast early; needles affected by *Hypodermella* are retained for 1 year or more. Infected crowns usually refoliate, but repeated infection may cause growth retardation.

Cedar leaf blight

This blight, caused by the fungus *Didymascella (=Keithia) thujina*, affects western redcedar seedlings and saplings. Infected foliage, especially lower foliage in dense stands, appears scorched in the spring. In the fall, infected leaf twigs drop, leaving the branches bare. In the spring following infection, circular to elliptical brown to black fruiting bodies are formed on the upper surfaces of infected leaves.

Many species of fungi cause leaf spots, lesions, or leaf mortality in Oregon hardwoods (Table 5). Except perhaps for the leaf rusts and anthracnose diseases of ash and maple, most leaf diseases are of little economic importance.

No serious foliage diseases have been reported in Oregon on redwood or Port-Orford-cedar.

Canker diseases

Canker diseases are caused by fungi that attack tree stems and branches. Canker diseases usually are indicated by dead branches or tops along with localized dead areas on the stem or branches. These dead, often sunken areas of infected bark are referred to as "cankers." Branch death occurs when the fungus penetrates and kills inner bark tissues, cutting off the flow of water and nutrients to the rest of the branch. Most canker diseases occur sporadically and usually affect trees on unfavorable sites or trees weakened by water stress or low-temperature injury.

Atropellis canker

Two species of fungi, *Atropellis piniphila* and *Atropellis pinicola*, cause this disease of pine. The disease occurs in many lodgepole pine stands, but relatively few trees are severely infected. The disease also affects western white, whitebark, ponderosa, and sugar pine. Infection can result in wood quality reduction, stem breakage, and occasionally tree mortality. Windborne spores infect throughout the growing season through unbroken bark in the internodal region, though some infection occurs through branch stubs. Small black or dark brown fruiting bodies form on dead bark in the cankers. The disease is best identified by the bluish stain in the wood behind the canker.

Cytospora canker

Cytospora canker is caused by several species of fungi in the genus *Cytospora: Cytospora abietis* on true fir and sometimes on Douglas-fir; *Cytospora sordida* on black cottonwood; *Cytospora chrysosperma* on quaking aspen; *Cytospora ambiens* on Oregon ash; and an unidentified species on alder, birch, and willow. The disease usually is associated with some sort of environmental stress or wounding and seldom causes serious damage except when associated with dwarf mistletoe. Spores are carried by wind, insects, birds, or splashing rain. New hosts are infected through wounds, dead twigs, or dwarf mistletoe infections. The fungi produce slightly sunken cankers that result in branch or stem mortality.

Phomopsis canker

This disease of Douglas-fir is caused by the fungus *Diaporthe (=Phomopsis) lokoyae*. Infection can result in top-killing and mortality of small trees, but damage usually is not serious. Spores infect small shoots and cause distinct round or oval sunken cankers with brown bark. This bark sloughs off in the following growing season. Small dead branchlets frequently are in the center of cankers. Fruiting bodies appear as fine black pimples on dead bark. The disease is associated with drought.

Dermea canker

Dermea canker occurs on young Douglas-fir in Oregon. It is caused by the fungus *Dermea pseudotsugae*. The trunk cankers often girdle the tree resulting in top-kill and tree mortality. Trees are predisposed to Dermea canker by drought or frosts. The disease is recognized by sunken cankers that have tiny, black fruiting bodies that appear on the dead bark a year after infection.

Sclerophoma canker

This canker affects all species of pine and Douglas-fir. The causal fungus is *Sclerophoma pythiophila*. Infections cause twig and top dieback with some tree mortality. The fruiting bodies are small, round, and embedded in the needles or bark. The infected wood is stained a bluish black.

Grovesiella canker

Grovesiella canker affects true fir and is caused by the fungus *Grovesiella (=Scleroderris) abieticola*. Infections result in annual cankers and twig dieback. Small, black fruiting bodies form on the dead wood of the canker.

Nectria canker

This disease affects true fir and hardwoods. Cankers are caused by the fungus *Nectria fuckeliana* in true fir; by *Nectria galligena* in bigleaf maple, myrtlewood, willow, dogwood, and black cottonwood; by *Nectria cinnabarina* in ash, white alder, cherry, and buckthorn; by *Nectria coccinea* in dogwood; by *Nectria corylis* in willow; and by an unidentified species of *Nectria*

in red alder and birch. Fruiting bodies are formed at the margins of conspicuous trunk cankers.

Sirococcus tip blight

The fungus *Sirococcus strobilinus* causes a tip blight in hemlock and spruce seedlings and saplings. Small grayish-green to black fruiting bodies form on shoots, needles, or cones. Spores may be seedborne.

Other canker diseases

Other canker diseases of hardwoods are caused by several species of fungi.

- *Verticillium albo-atrum* causes branch flagging in bigleaf maple.
- Several fungi cause cankers in red alder: *Didymosphaeria oregonensis* and *Hymenochaete agglutinans*.
- Three species of fungi cause cankers in Pacific madrone: *Botryosphaeria dothidea, Phytophthora cactorum,* and *Hendersonula toruloidea.*
- *Hysterographium fraxini* causes a canker in Oregon ash.
- *Dothichiza populea, Fusarium* spp., *Hypoxylon mammatum,* and *Septoria musiva* cause cankers in black cottonwood.
- Cankers in quaking aspen are caused by *Ceratocystis fimbriata* and *Cryptosphaeria populina.*

No fungi have been reported to cause serious cankers in cedar, juniper, redwood, tanoak, oak, or chinkapin in Oregon.

Seedling diseases

All root, rust, foliage, and canker diseases discussed previously can affect seedlings and more frequently kill seedlings than more mature trees. In addition, the following diseases typically affect only seedlings in nurseries or greenhouses.

Damping-off

Several genera of fungi cause damping-off: *Phytophthora, Pythium,* and *Fusarium.* All species of conifers and some species of hardwoods are susceptible. Damping-off fungi are soil inhabitants and survive either as dormant spores or as mycelium in organic matter. Seedlings may fail to emerge due to infection or decay of seeds, or infection may occur in succulent stem tissue at or just below the ground line. The disease is spread by movement of infested soil or seedlings.

Fusarium root rot

The rot caused by the fungus *Fusarium oxysporum* is the most serious disease in Oregon bareroot nurseries. The disease can affect all conifer seedlings. Spores infect succulent young roots in warm, moist conditions. In older seedlings it causes typical root rot symptoms: rootlet deterioration, foliage yellowing, general decline, and death. Soil and seedling movement spread the disease.

Stem cankers

Cankers caused by *Fusarium* sp. and *Phoma eupyrena* are particularly damaging to bareroot Douglas-fir. *Diaporthe lokoyae* and *Sirococcus* spp. also cause cankers and tip dieback on Douglas-fir and spruce respectively. Damage usually is noticed when seedling tops turn brown. Closer inspection reveals dead portions on the stem where the fungus killed the cambium.

Gray mold

This disease, caused by the fungus *Botrytis cinerea,* is especially common in container nurseries and in dense 2-year-old and older beds in bareroot nurseries. All conifer species, especially Douglas-fir, hemlock, and cedar, are affected. The fungus is a common soil inhabitant that infects by windborne or water-splashed

spores, especially during cool, moist conditions. The disease is recognized by a brownish-gray mycelium on infected portions of seedlings. Spores are released when seedlings are agitated. Infection turns foliage yellow and brown, causing seedling dieback and death.

Phytophthora root rot

Several species of fungi in the genus *Phytophthora* cause this rot. Most conifer and some hardwoods are susceptible. The fungus invades roots through the soil. Mycelium and spores require high soil moisture to spread and can be moved on plants, machinery, or animals to uninfested areas. The disease often is confined to wet, low-lying areas, along drainages, or where drainage is poor. Stunting, yellowing, and wilting usually precede seedling mortality. Red to brown discoloration of root cambium is a common symptom.

Management

Management of foliage, canker, and seedling diseases usually is not warranted nor practical in forest settings. In many cases, environmental conditions contribute to disease expression. To reduce disease impact, maintain vigorous plants through spacing, thinning, balancing nutrients and pH, maintaining adequate soil moisture, preventing wounds, increasing biodiversity, and especially by avoiding offsite planting. In forest nurseries, Christmas tree farms, or agricultural, greenhouse, or ornamental settings, foliar sprays may help prevent or reduce infection.

Mistletoes

All mistletoes are flowering, seed-bearing plants. They have stems, roots, and foliage and reproduce and spread by seeds (Figure 10). Dwarf mistletoes do not have enough chlorophyll to produce their own food. They rely totally on the host tree for nutrients and water, which they extract through rootlike structures that penetrate into the bark and wood of stems and branches. When the host tree dies, the mistletoe plant also dies. Infections reduce tree growth and vigor, lower wood quality, and cause mortality.

Dwarf mistletoes (*Arceuthobium* spp.) are one of the most damaging diseases of Oregon's conifer forests. On the other hand, they provide habitat for several owl species and for other birds and mammals. True mistletoes (*Phoradendron* spp.) are not as damaging as dwarf mistletoes.

General biology

Dwarf mistletoes

Dwarf mistletoes complete their life cycle in about 4 to 6 years. In late summer, fruits of female plants mature. Small sticky seeds are explosively ejected from fruit and may travel as far as 40 feet from the tree. A few of these seeds land on and adhere to needles. Rain allows the seed to slide down the needle and lodge against the stem at the needle base. The following spring or summer, the seeds germinate and sink roots into the stem. There are no visible symptoms at this time.

During the second year, the first visible symptom—swelling of infected branches—appears. This swelling continues to enlarge in the third year when the first (sterile) shoots of the dwarf mistletoe plants appear. The characteristic aerial shoots, complete with flowers, usually appear in the fourth year and produce seed by the end of the fifth year. Shoots may not appear if, as in dense understory stands, there is insufficient light. These are called "latent" infections and will not form shoots until overstories are killed or removed. Severe, long-term dwarf mistletoe infection often stimulates host trees to produce dense clumps of branches called "witches' brooms" (Figure 11). The brooms serve as "sinks" for nutrients that otherwise would be used to grow wood and foliage.

Figure 10.—Dwarf mistletoe stems and seeds on an infected ponderosa pine.

True mistletoes

True mistletoes bear seeds in white fruits. Seeds are not forcibly discharged as in dwarf mistletoes. Instead, birds eat the fruits, digest the pulp, and excrete the living seed on branches where birds perch. The seeds germinate, and infection occurs in the young, thin bark. True mistletoes make most of their food through photosynthesis but get water and nutrients from their hosts.

Douglas-fir dwarf mistletoe

Douglas-fir dwarf mistletoe (*Arceuthobium douglasii*) probably is the most damaging disease of Douglas-fir in central, southern, and eastern Oregon. The primary host is Douglas-fir; infection also has been reported in true fir (Table 6, pages 36-37).

Table 6.—Occurrence of mistletoes on Oregon trees.

Tree species	Primary host for (> 90% infected)	Secondary host for (50-90% infected)	Occasional host for (5-50% infected)	Rare host for (< 5% infected)
Conifers				
Cedar				
Alaska-	None	None	None	None
Incense-	Incense-cedar mistletoe	None	None	None
Western red	None	None	None	None
Fir				
Douglas-	Douglas-fir dwarf mistletoe	None	None	None
Grand or White	White fir dwarf mistletoe	None	None	Hemlock,[1,2] Douglas-fir, and larch dwarf mistletoes.
Noble	Hemlock dwarf mistletoe[1,2]	None	None	None
Pacific silver	Hemlock dwarf mistletoe[1,2]	None	None	White fir and Douglas-fir dwarf mistletoes
Shasta red	Red fir dwarf mistletoe	None	None	None
Subalpine	Hemlock dwarf mistletoe[1]	None	Larch dwarf mistletoe	Douglas-fir dwarf mistletoe
Hemlock				
Mountain	Hemlock dwarf mistletoe[1]	Limber pine dwarf mistletoe	None	Hemlock dwarf mistletoe[2]
Western	Hemlock dwarf mistletoe [2]	None	Hemlock dwarf mistletoe[1]	None
Juniper	Dense mistletoe Juniper mistletoe	None	None	None
Larch	Larch dwarf mistletoe	None	None	None
Pine				
Jeffrey	Western dwarf mistletoe	None	None	Knobcone and white pine dwarf mistletoes
Knobcone	Knobcone pine dwarf mistletoe	Western dwarf mistletoe	None	None
Limber	None	None	None	None
Lodgepole	Lodgepole pine dwarf mistletoe	None	Larch and western dwarf mistletoes	Hemlock dwarf mistletoe[1]
Ponderosa	Western dwarf mistletoe	None	Lodgepole pine dwarf mistletoe	Knobcone pine dwarf mistletoe
Shore	None	None	Knobcone pine dwarf mistletoe	None
Sugar	None	White pine dwarf mistletoe	None	None

Table 6.—continued.

Tree species	Primary host for (> 90% infected)	Secondary host for (50-90% infected)	Occasional host for (5-50% infected)	Rare host for (< 5% infected)
Conifers—continued				
Pine—continued				
Western white	White pine dwarf mistletoe	None	Hemlock dwarf mistletoe[1]	None
Whitebark	Limber pine dwarf mistletoe	Hemlock dwarf mistletoe[1]	None	Larch dwarf mistletoe
Redwood	None	None	None	None
Spruce				
Brewer	None	White fir dwarf mistletoe	White pine dwarf mistletoe	Hemlock dwarf mistletoe [2]
Engelmann	None	None	None	Hemlock dwarf mistletoe [2]
Sitka	None	None	None	Hemlock dwarf mistletoe [2]
Yew	None	None	None	None
Hardwoods				
Oak	Oak mistletoe	None	None	None

[1] Mountain hemlock subspecies

[2] Western hemlock subspecies

Douglas-fir dwarf mistletoe has rather small (about 0.25 to 0.5 inch long) leafless, olive-green shoots near the ends of infected branches in witches' brooms. Usually only a small portion of a witches' broom has visible mistletoe plants. Witches' brooms usually are the last portion of the tree to die, and death may take several decades even in severely infected trees. Growth losses of 50 to 60 percent have been reported in severely infected trees.

Hemlock dwarf mistletoe

Hemlock dwarf mistletoe (*Arceuthobium tsugense*) causes substantial economic damage on several other species of conifer besides western and mountain hemlock. There are two subspecies: *Arceuthobium tsugense* subspecies *tsugense* attacks mainly western hemlock and true fir, and *Arceuthobium tsugense* subsp. *mertensianae* attacks mainly mountain hemlock and the true fir. Pacific silver fir and noble fir are severely damaged by both subspecies of hemlock dwarf mistletoe. In true fir, several species of canker fungi, especially *Cytospora abietis*, infect near the mistletoe infections and cause twig and branch mortality. Eventually, if enough branches are affected, the weakened trees are attacked by bark beetles and die.

Hemlock dwarf mistletoe has green to reddish shoots about 2 inches long. Witches' brooms are common on older trees. Infected trees with good crown ratios grow better than trees with poor crowns. Severely infected trees have volume growth rates about half those of uninfected trees.

Figure 11.—Dwarf mistletoe causes large witches' brooms in Douglas-fir, as in the fir at left.

Larch dwarf mistletoe

Larch dwarf mistletoe (*Arceuthobium laricis*) probably is the second most economically destructive mistletoe in Oregon. Its principal host is western larch; subalpine fir and lodgepole pine are occasional hosts, and whitebark pine is a rare host.

Larch dwarf mistletoe has dark purple shoots 1.5 to 4 inches long. Witches' brooms are common and frequently break off under snow and ice in the winter. Severely affected trees often resemble telephone poles because of the loss of infected branches. Occasionally, severely infected and suppressed trees are broken or bent by the snow and ice.

Pine dwarf mistletoes

Pine dwarf mistletoes include *Arceuthobium americanum* primarily on lodgepole pine; *Arceuthobium campylopodum* on ponderosa, Jeffrey, and knobcone pine; *Arceuthobium monticola* on white pine, *Arceuthobium cyanocarpum* on whitebark pine; and *Arceuthobium siskiyouense* on knobcone pine.

Lodgepole pine dwarf mistletoe has olive-green shoots that are about 2.5 inches long. Large witches' brooms are common. This is economically the most important disease of lodgepole pine. *Arceuthobium campylopodum*, also called western dwarf mistletoe, has olive-green to yellow shoots that are very large (3 to 8 inches long). Witches' brooms generally are not as large as in Douglas-fir or lodgepole pine. They often are confused with witches' brooms caused by Elytroderma needle blight, but the latter do not have the mistletoe plants.

Arceuthobium monticola, white pine dwarf mistletoe, occurs only in southern Oregon. Its dark brown shoots are 3 to 4 inches high. *Arceuthobium cyanocarpum*, limber pine dwarf mistletoe, has yellow-green shoots 1 to 2 inches high. *Arceuthobium siskiyouense*, knobcone pine dwarf mistletoe, has yellow to brown shoots 2 to 3 inches high.

Although limber pine dwarf mistletoe has been reported on whitebark pine, it has not been found on limber pine in Oregon.

True fir dwarf mistletoe

True fir dwarf mistletoe is caused by *Arceuthobium abietinum*. One variety infects white and grand fir, and another

variety infects Shasta red fir. True fir mistletoe occurs only in the Cascade Range and in southern Oregon. As with noble and Pacific silver fir that are infected with hemlock dwarf mistletoe, trees infected with true fir mistletoe frequently are attacked by canker fungi that enter through old mistletoe infections. Branch mortality leading to bark beetle attack and tree mortality are common especially in central Oregon. Old true fir mistletoe infections also serve as entry points for stem decay fungi.

True fir mistletoe is identified by the yellow-green shoots that are 3 to 8 inches long. Witches' brooms generally do not form, but branch dieback in true fir usually is a symptom of true fir dwarf mistletoe. Trees with good live crowns are better able to withstand infection than trees with poor crowns.

"True" or leafy mistletoes

"True" or leafy mistletoes, *Phoradendron* spp., are found on conifers and hardwoods. "Dense" mistletoe (*Phoradendron densum*) and juniper mistletoe (*Phoradendron juniperinum*) infect western juniper. Incense-cedar mistletoe (*Phoradendron libocedri*) attacks incense-cedar. *Phoradendron villosum* attacks both Oregon white oak and California black oak. Oak mistletoe often is collected and used during Christmas.

Ecologic roles

The destructive role of dwarf mistletoes from a timber management perspective has been recognized for decades in Oregon. Tree growth retardation, lower wood quality, and tree killing are the result of severe dwarf mistletoe infections. Other roles that dwarf mistletoes play in forest ecosystems are not well known, and only very recently have we begun to recognize the importance of dwarf mistletoes from a nontimber viewpoint.

Wildfire probably is the primary natural ecological factor governing the distribution and abundance of dwarf mistletoes in Oregon. Wildfire suppression during this century has led to an increased frequency of dwarf mistletoes especially in central, southern, and eastern Oregon. Increased infections create mistletoe brooms that eventually die and fall to the ground. This creates a fuel ladder for fire and increases fire around the bases of trees, resulting in more tree mortality even in thick-barked species such as ponderosa pine.

Associations between dwarf mistletoe and vertebrates in Oregon are now being realized and increasingly reported. During bad weather, porcupines often use Douglas-fir that are infected with dwarf mistletoe; the witches' brooms offer protection from snow and wind. The northern spotted owl, the great gray owl, and the long-eared owl have been shown to select dwarf-mistletoe-infected Douglas-fir for nest building. Other bird species also use the witches' brooms for roosting. Hemlock mistletoe brooms may be used by the marbled murrelet.

The ecologic roles of true mistletoes are not well known, except that they provide a food source for birds.

Management

To aid timber production and wildfire prevention, dwarf mistletoe eradication treatments have been used for decades in Oregon. Clearcutting or selectively removing or girdling infected overstory trees is very effective because dwarf mistletoes spread most rapidly from tall trees to nearby small ones. Dwarf mistletoes require a living host to survive; once a tree dies, the dwarf mistletoe dies.

Infected green trees that are left in clearcuts can and will infect susceptible regeneration.

Another strategy to reduce the impact of dwarf mistletoes is to favor resistant or nonhost species in mixed-species stands during thinning or partial cutting. Except for hemlock dwarf mistletoe and western dwarf mistletoe, each mistletoe seriously affects only one host species; for example, Douglas-fir dwarf mistletoe will not infect ponderosa pine.

Thinning also can be used to reduce dwarf mistletoe infections to acceptable levels consistent with management objectives. Thinning has been shown to improve growth rates even in moderately infected ponderosa pine, Douglas-fir, and larch in Oregon. The key is to remove infected overstory trees to prevent further infection of crop trees, and then space residual trees to improve growth and vigor. Crop trees should not be thinned until at least 5 years after overstory removal in order to allow enough time for latent infections to appear.

Branch pruning also is a mistletoe control option. This has been done with young trees, ornamental trees, and trees in developed recreation areas. However, as the economic benefits of pruning are realized in Oregon, pruning to remove mistletoe-infected branches will become more practical as a regular operational control strategy.

As we realize the beneficial role of dwarf mistletoe in wildlife habitat, managing for dwarf mistletoe has become more acceptable. Infected trees can be protected as wildlife trees with the trade-off being reduced tree growth and vigor and an infection source for adjacent susceptible trees.

Abiotic Diseases

Abiotic (nonliving) factors of the environment can directly injure trees, or they can predispose trees to damage by other pests. The most important abiotic diseases in Oregon are caused by water stress (for example, resulting from drought), soil compaction or site disturbance, and low temperatures. Air pollution damage, sun scald, and damage from nutrient deficiencies are examples of other important abiotic diseases.

Water stress

Water stress in trees usually results from soil moisture deficiency caused by periods of below-normal precipitation (drought). The severity of water stress is influenced by soil types, competing vegetation, soil disturbance, and other factors. Symptoms of water stress include reduced growth, yellowing needles, loss of foliage, and dead branches. Symptoms generally progress from the top of the tree downward and from the outside to the inside of the crown. Symptoms usually appear first on tree parts most distant from the water-absorbing roots.

Although water stress can kill trees, its most important effect is that it predisposes trees to injury by insects and fungi. Stressed trees have reduced vigor and are less able to produce defensive chemicals to ward off attacks by many pests. Many canker diseases damage trees that are experiencing water stress. As a result, the occurrence of pest-caused damage rises dramatically during and for a few years following drought.

Soil compaction and site disturbance

Soil compaction can reduce water penetration and impede gas exchange in roots, which increase mortality of the fine, nutrient-absorbing roots. As a result, trees on compacted soils or with damaged roots have many of the symptoms previously described for water stress. Regardless of tree size, most water-absorbing roots occur in the uppermost layers of the soil, many within a few inches of the surface. In fact, 90 percent of the fine roots in trees are in the top 2 feet of soil. Livestock and heavy equipment easily damage roots and compact soils in this zone.

Low-temperature injury

Unseasonably low temperatures can directly kill tree tissues. Late spring frost can kill succulent new foliage soon after it emerges. Very low temperatures following an unseasonably warm period during winter can cause top kill in large trees. Winter drying occurs when warm winds blow over very cold or frozen soil. The warm winds cause foliage to lose water rapidly, while cold soil slows water uptake by roots. The result is dehydration and needle browning, often affecting trees over large areas.

Damage reduction

Prevention is the key to reducing damage from abiotic factors. This can be accomplished by maintaining proper tree densities, managing competing vegetation, and proper livestock management. Avoid unnecessary disturbance and soil compaction. Compacted soil can be tilled, but living tree roots may be damaged. Plant only tree species that are adapted to the site and from the appropriate seed zone. This will ensure that trees are better able to resist extreme changes that may occur.

Effects of Forest Practices on Disease

CHAPTER 8

Precommercial thinning

Precommercial thinning—removing trees too small to have commercial value in order to achieve better spacing in the rest of the stand—has been practiced for many years in Oregon, especially in Douglas-fir and ponderosa pine. Scientific research to date has shown some advantages and disadvantages to precommercial thinning as a disease-control measure.

Advantages include:

- Wounded and infested trees can be eliminated.
- Residual trees, if wounded during early thinning, will develop small decay columns rather than the large columns created if thinning is done when trees are larger.
- Excellent growth response will result if live-crown ratios and previous height growth are good.
- Shorter rotation ages can be used.
- Disease-tolerant species can be favored.
- Residual trees are more resistant to certain diseases because of increased vigor.

Some disadvantages are:

- Some species (for example, true fir) can be sunscalded on certain sites if spacing is too wide.
- Slash creation increases risk from fire, stem-wounding, and bark beetle attack.

In central Oregon, precommercial thinning increases volume growth in ponderosa pine and Douglas-fir that is moderately infected with dwarf mistletoe. Douglas-fir mortality after 10 years occurs in less than 1 percent of the thinned trees. Less infected trees show a significant increase in radial growth following thinning, but more heavily infected trees do not.

In eastern Oregon, precommercial thinning increases vigor of true fir which decreases tree susceptibility to infection and stem decay caused by the Indian

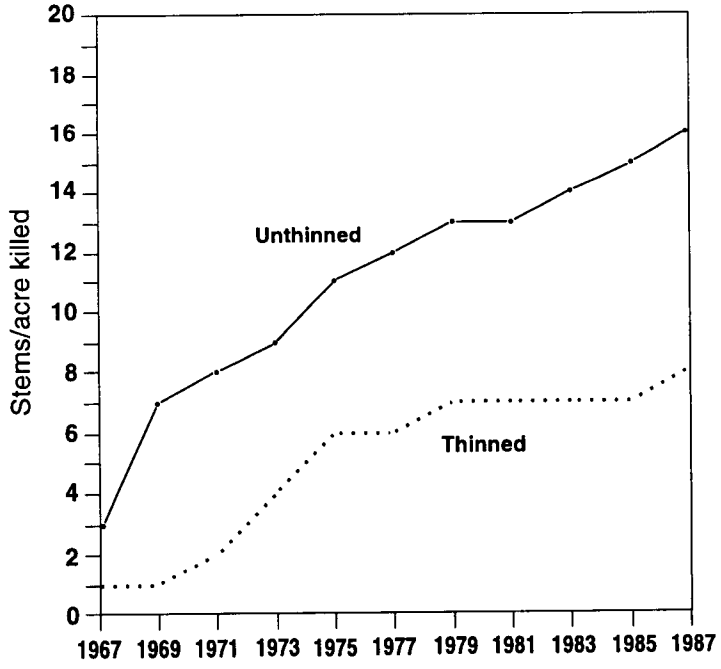


Figure 12.—Cumulative mortality of crop trees caused by Armillaria root disease in thinned and unthinned plots of 30-year-old ponderosa pine in central Oregon.

paint fungus. In the Cascade Range, precommercial thinning neither increases nor decreases mortality from Armillaria root disease in ponderosa pine (Figure 12), Douglas-fir, true fir, or hemlock. Precommercial thinning in Douglas-fir stands with black stain root disease may increase root disease.

Commercial thinning

Commercial thinning is selectively removing trees that can be used commercially. As utilization standards improve, trees that were precommercially thinned yesterday may be of commercial value today.

Advantages of commercial thinning are similar to those for precommercial thinning, but there are additional disadvantages because the trees are larger. Those include:

- Wounded trees lead to larger decay columns.
- Stumps can become inoculum sources for annosus root disease.
- Windthrow can increase, especially in stands with root rot.

Volume growth in larch decreases as severity of dwarf mistletoe infection increases. Thinning from above (removing dominant and codominant trees) increases mortality from snow and ice damage to infected residual trees. Thinning from below (removing intermediate and suppressed trees) is recommended to increase volume growth and vigor in lightly to moderately infected residual larch trees and to reduce new infections by removing severely infected trees. The effect of commercially thinning mistletoe-infested stands of other tree species has not been studied but probably is similar to larch.

Commercial thinning can increase the incidence of stem decay if measures are not followed to reduce tree wounding. On the other hand, the *percentage* of decay in thinned trees is less because of the extra volume growth added after thinning. Commercial thinning has been thought to increase the incidence of windthrow in stands with root disease, but few data exist to support this claim.

Sanitation–salvage cutting

The term "sanitation–salvage" cutting is a combination of several closely related terms. Salvage cuttings are made for the chief purpose of harvesting trees that have been or are in imminent danger of being killed or damaged by injurious agents other than competition among trees. Sanitation cuttings are to remove trees that have been attacked or appear in imminent danger of attack by pests to prevent spread from one tree to another. These operations are not necessarily confined to the removal of merchantable trees.

There are several economic benefits to sanitation–salvage cutting, benefits especially but not exclusively related to timber values. Sanitation–salvage cutting reduces fire hazard by removing dead and dying trees. Stands can be regenerated to a more healthy condition. Infested and high-risk trees are removed. The economic loss of dead and dying trees is partially recovered.

It has been suggested, though not conclusively demonstrated, that sanitation–salvage cutting increases other pest problems such as root diseases. Living trees have defense mechanisms that prevent root pathogens from advancing along root systems to the root collar, which results in tree mortality. Dead trees lack these defense mechanisms, and infection from *Armillaria* or annosus quickly spreads throughout the entire root system after living infected trees are killed or

Figure 13.—Advance Douglas-fir regeneration in an area where trees died of laminated root rot.

harvested. This causes an increase in inoculum potential and can result in increased infection and mortality of adjacent living trees.

Partially harvested stands, including seed-tree and shelterwood harvests and those with sanitation–salvage cutting, may have increased mortality due to root diseases, especially annosus root disease. This occurs when living trees are harvested with dead trees. Spores from the annosus root disease fungus, *Heterobasidion annosum*, require freshly exposed living wood, such as in a freshly cut stump top or fresh trunk wound, to germinate and infect. In forests with repeated defoliation by insects, many trees thought dead and marked for removal actually are still alive, and the freshly cut stumps are ideal infection courts for windblown spores of *Heterobasidion annosum*, especially stumps of true fir and hemlock.

Clearcutting and regeneration

For pest management, clearcutting usually presents fewer problems than other types of regeneration harvesting, because it leaves few trees to be windthrown, to infect regeneration, or to damage regeneration upon subsequent removal. However, susceptible regeneration can become infested with dwarf mistletoe from adjacent border trees or unmerchantable residuals, or root disease may spread from infested stumps to susceptible regeneration within the unit.

The type of regeneration—planted, natural, or advance—will determine the amount of potential pest damage. Planting allows the establishment of pest-resistant species, but seedlings need to be from appropriate seed transfer zones or else severe damage from disease may result. Natural regeneration may foster the spread of certain diseases if susceptible species are allowed to regenerate. Advance regeneration (Figure 13) may already be infested with pathogens, such as the Indian paint fungus, before the overstory is harvested and therefore poses the greatest risk of future pest-caused losses. Also, new practices that retain living trees and snags within clearcuts will influence disease populations and the amount of new host trees in the future stand.

Uneven-age management

Uneven-age management, in the strict sense, has not been widely practiced in Oregon. Although many forests have an uneven-age appearance, many actually are uneven in size rather than uneven in age. This is especially true for shade-tolerant species such as true fir, where suppressed understory trees may be the same age as their overstory.

From a pest management perspective, uneven-age management is more appropriate in some forest types, such as pure ponderosa pine, than in others, such as true fir dominated forests, because of the fewer pests associated with pine. Root diseases, stem decays, and dwarf mistletoes are affected by stand structure and composition. Silvicultural systems that produce and maintain multistoried stands and climax tree species (especially true fir) generally will allow these forest pests to increase.

All the important root pathogenic fungi, the principal causes of root disease, spread underground through root contacts or grafts. In addition, annosus root disease spreads quite effectively by airborne spores, and black stain root disease is spread by insects. Live healthy trees eventually contact infected stumps. Harvesting large, live, infected trees may aggravate root disease on a site. If uneven-age management creates stands by means of repeated harvesting and the establishment of susceptible regeneration, root disease may be perpetuated and made worse.

Stem decay fungi spread via airborne spores that either enter fresh wounds or are stimulated by wounding if already present in infected stems. True fir that have been suppressed are more prone to infection by the Indian paint fungus than are vigorous trees. Therefore, if uneven-age management increases tree wounding through increased stand entry, or increases tree suppression, then stem decay may increase.

Dwarf mistletoe affects most conifer species in Oregon. Spread is by seeds that rely on forceful ejection, wind, and gravity to contact susceptible hosts. Multistoried and single-species stands foster effective mistletoe spread. Therefore, if uneven-age management creates these kinds of stands, dwarf mistletoe severity may increase.

Measures can be taken to reduce pest-caused damage in multistoried stands that result from uneven-age management:

- Nonsusceptible tree species can be favored and regenerated.
- Tree vigor can be improved and maintained through thinning.
- Tree wounding can be reduced by properly planning harvest operations.
- Freshly cut stumps can be treated to prevent infection from root pathogens.

Uneven-age management in most cases will require more care than even-age management. That may be impractical in severely diseased stands, but can be effective in many stands to meet land-use objectives and still prevent and reduce the adverse affects of forest diseases. More research and testing are needed to determine the short- and long-term effects of uneven-age management in several plant communities in Oregon.

Prescribed burning

Prescribed burning has been a silvicultural practice for many years, especially in eastern Oregon. It has been used to reduce fuel loads and to remove unwanted understory vegetation. However, more research is needed in Oregon regarding the use of prescribed fires and their effects, both positive and negative, on the incidence of root and stem decays

Figure 14.—Root diseases may be managed by excavating infected stumps with heavy machinery.

and associated bark beetles in residual trees and in subsequent regeneration.

In central Oregon, ash leachates from prescribed burns in ponderosa pine decrease the growth of *Armillaria ostoyae*, cause of Armillaria root disease. These leachates increase the growth of *Trichoderma* spp., which in turn reduces the growth and rhizomorph formation of *Armillaria ostoyae*. However, the effects of fire on infection and mortality from Armillaria root disease still is largely unknown.

In southern Oregon, underburning white fir stands causes sufficient scorch to cause cambial death that is associated with stained and decayed wood even 2 years after burning.

Nitrogen fertilizing

Some research on the effects of fertilizer on forest diseases has been reported in Oregon. Fertilizing with urea significantly improves tree growth and vigor. This should shorten rotation ages and decrease decay volumes by increasing sound wood volumes. Although wound closure and cross-sectional area of decay are not affected by fertilizing, the *percentage* of decay is significantly less in trees that have been both thinned and fertilized.

Effects of fertilizing on dwarf mistletoes and host are mostly unknown. In one study with artificially infected ponderosa pine seedlings, height growth of fertilized and infected seedlings was significantly greater than that of unfertilized and infected seedlings.

Most of the research on fertilizer has involved root diseases. Low levels of certain soil nutrients are associated with infection and decay caused by Armillaria root disease. Where fertilizer has been applied to infected stands, results have been conflicting. In some cases, Armillaria root disease has increased; in others, it has decreased. Differences may depend on whether trees already are infected when fertilizer is applied.

Stump treatments for root disease

Stump treatments have been used both experimentally and operationally to prevent and reduce damage caused by root pathogens in Oregon. Two types of treatments have been tried: removing the stump from the soil (Figure 14) and treating the stump with chemicals or biological agents. By removing the principal inoculum sources in infected areas—

primarily, infected stumps from harvested or dead trees—incidence of root disease on an infested site should decrease with time, even in highly susceptible tree species. Some studies have demonstrated this and even have shown increased seedling growth after stump excavation.

Applying borax (sodium tetraborate) has been demonstrated to protect stumps from root pathogens. Operationally, it is used only for annosus root disease in Oregon. However, more recent studies in northeastern Oregon have suggested that freshly cut stumps of true fir already may have annosus root disease, and the effectiveness of borax in preventing *additional* stump infection may be questionable, especially in stands that have been harvested previously. Also, the effects of boron on nontarget organisms, such as ants and mammals, is being questioned.

Applying biological control agents, such as antagonistic fungi, to stumps looks promising but needs further testing to become operational. More research is needed in Oregon to assess the frequency of live tree root infections and the effectiveness of treating previously harvested stands with chemicals or biologicals. Using fungicides to protect living trees from underground infection by other root pathogens such as *Armillaria ostoyae* has been shown to be ineffective.

Fumigation to eradicate some root pathogens from infected stumps and even from living trees shows more promise than protectant fungicides. Fumigants such as chloropicrin, methyl bromide, Vapam, Vorlex, or carbon disulfide can eliminate *Armillaria ostoyae* from small stumps of ponderosa pine. Chloropicrin, allyl alcohol, Vapam, or Vorlex can eliminate *Phellinus weirii* from Douglas-fir stumps. Experiments have had some success in controlling laminated root rot with antagonistic soil fungi such as *Trichoderma* spp. instead of with fumigants.

Artificial branch pruning

Natural or self-pruning is a slow process. The presence of saprophytic fungi, which hasten decay of the dead branches, is the most important factor in determining the rate of natural branch shedding. Artificially pruning lower crown branches, both living and dead, has been used to improve wood quality and value. Pruned live branches seal faster than pruned dead branches. Artificial pruning usually is combined with stand thinning to increase tree growth and sealing of branch stubs.

Advantages of artificial pruning are:

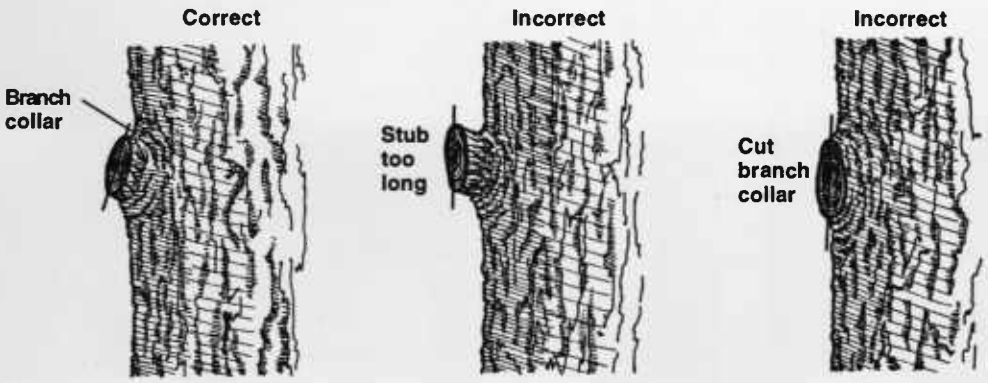
- Increased wood quality and value, because pruning creates tight knots or no knots in outer wood rather than the loose knots that form from dead branches in the natural pruning process
- Improved stand access during thinning operations
- Removal or prevention of infection from stem rusts and dwarf mistletoe

Disadvantages of pruning are:

- Risk of increased stem decay, ring shakes, frost and sun cracks, wetwood, cankers, bark and pitch pockets, and insect attack because of improper pruning
- Tree growth reduction if too many live branches are removed
- Sunscalding of thin-barked species
- Formation of epicormic branches

From a disease standpoint, pruning has been used most successfully for prevention of white pine blister rust. Basal and stem cankers most often are the cause of mortality of blister-rust-infected pines. These infections occur in small trees, 2 to 5 feet high, where the lower branches are infected. If the lower half of the live crown is pruned, the likelihood of infection is drastically decreased. All infected branches in the remaining crown also should be pruned. Trees with cankers on the main stem should not be pruned,

Figure 15.—Prune branches properly to minimize damage to the branch collar.



because these trees probably will die within the next few years.

For other stem rusts and for mistletoe infections, it usually is necessary to remove only infected branches.

Improper pruning can cause serious damage to trees from decay, cracks, and cankers. Branches should not be cut flush with the stem but flush with the branch collar (Figure 15). Stubs beyond the branch collar also should be removed because, on both living and dead branches, these provide entry courts for decay fungi. After pruning, cuts do not need to be painted; in fact, wound dressings have been shown to increase decay in some cases.

Conclusions

Tree diseases generally are much easier to prevent than to correct. By integrating disease management with forest resource management, the overall health and productivity of Oregon's forests can be improved and maintained. Many forest practices are used in Oregon, but their long-term effects on forest diseases are not well known. Natural enemies of pathogens and associated insects also are influenced by forest practices. Healthy forests cannot be maintained solely through preservation. Intelligent, active forest management can manipulate forest diseases to improve and maintain the quality of Oregon's natural resources.

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Glossary

Abiotic—Nonliving.

Abiotic diseases—Diseases caused by nonliving factors such as water stress, temperature extremes, or soil compaction.

Advance regeneration—Regeneration that occurs before any special measures are undertaken to establish new growth.

Aecia—Yellow-orange spore pustules of a rust fungus.

Agent—Something that produces or is capable of producing an effect, such as disease. For example, the fungus *Armillaria ostoyae* is the agent for Armillaria root disease. (See also **abiotic diseases** and **biotic diseases**.)

Alternate host—An organism that serves as a host for a parasite during part of the parasite's life cycle. (See also *host*.) For example, plants in the *Ribes* family (gooseberry and currant) are alternate hosts for the fungus that causes white pine blister rust.

Antagonistic fungi—Fungi that are damaging to other fungi. For example, the fungus *Trichoderma* is damaging to the root pathogen *Armillaria*.

Biological control—Controlling pests through the action of living organisms (whether naturally occurring or brought in by humans) rather than by the application of chemicals. For example, certain butterfly larvae feed on mistletoe plants, causing them to die.

Biotic—Living.

Biotic diseases—Diseases caused by living organisms such as fungi or parasitic seed plants (mistletoes).

Cambium—The thin layer of tissue in a plant that gives rise to new cells and is responsible for secondary growth.

Chlorosis (adj. **chlorotic**)—An abnormal yellowing or graying of plant parts, such as leaves, due to partial destruction of their chlorophyll. Chlorosis can be a symptom of plant disease.

Climax species—Those species of trees found in dominant positions in a fully mature forest.

Commercial thinning—Selectively removing trees that have commercial value. (See also **precommercial thinning**.)

Conk—The sporophore of a wood-invading fungus; usually, conks appear woody or leathery and grow on tree trunks or branches.

Disease—A sustained disturbance to the normal function or structure of a tree as provoked by biological (biotic), chemical, or physical (abiotic) factors of the environment.

Distress cone crop—An abundance of undersized cones produced by diseased trees as a symptom of advanced stages of disease and decline.

Ectotrophic mycelium—A white to grayish crust of fungal growth on root surfaces, a sign of laminated root rot.

Elongate—Stretched out; lengthened.

Entry court—An opening in a host organism, such as a wound on a tree, through which disease organisms can enter.

Epicormic branches—Branches that form on the bole of a tree after increased light allows dormant buds in the bark to sprout. These branches can create knots that degrade lumber value.

Forest succession—The predictable pattern of forest regeneration after a disturbance such as a fire or a clearcut.

Fruiting body—See **sporophore**.

Fungus (pl. **fungi**)—A single- or many-celled organism that, lacking chlorophyll and therefore unable to make its own food, feeds on dead or living plant or animal matter.

Gall—Abnormal stem swelling. A sign of disease, such as western gall rust of lodgepole pine.

Genus (pl. **genera**)—A class, kind, or group of organisms marked by one or more characteristics. For example, pine belong to the genus *Pinus*. (See also *species*.)

Group-selection harvest system—A tree harvesting system designed to create an uneven-age stand by repeatedly cutting groups or patches of mature trees at short intervals over an indefinite period of time.

Host—An organism, such as a tree, on or in which a parasite is living and from which it is obtaining its food. (See also **alternate host** and **secondary host**.)

Hyphae—The individual, microscopic, threadlike filaments of fungi.

Inoculum—The pathogen or pathogen part (such as spores or mycelium) that infects plants.

Internodal—Between the points on the stem at which a leaf or leaves emerge. *Internodal length* is the space between each year's twig bud scars.

Laminated decay—A symptom of laminated root rot in which decayed wood separates into sheets along annual growth rings.

Live crown ratio—The proportion of a living crown height to total tree height, usually expressed as a percentage.

Mycelial fans—White to cream-colored sheets of fungus found inside the bark on the lower main stem of a tree or in roots. They are a sign of Armillaria root disease.

Mycelium (pl. **mycelia**)—The mass of interwoven threads (hyphae) making up the vegetative body of a fungus.

Offsite planting—Trees that are transplanted to a geographical area different from the one in which they originally grew, especially if the site characteristics are different.

Pathogen—An entity, usually biological such as a fungus, that can cause disease.

Pathogenic—Causing or capable of causing disease.

Precommercial thinning—Removing trees too small to have commercial value in order to achieve better spacing for the rest of the trees in the stand. (See also **commercial thinning**.)

Prescribed burning—The use of regulated fires to reduce or eliminate the unincorporated organic matter of the forest floor or low, undesirable vegetation.

Rhizomorph—An aggregation of hyphae into a cordlike or rootlike strand.

Salvage cutting—Harvesting trees that have been or are in imminent danger of being killed or damaged by injurious agents other than competition from other trees.

Sanitation cutting—Harvesting trees that have been or appear to be in imminent danger of being attacked by pests, in order to prevent spread of disease from one tree to another.

Sanitation–salvage cutting—See **salvage cutting** and **sanitation cutting**.

Saprophyte (adj. **saprophytic**)—An organism that lives on dead or decaying organic matter.

Secondary host—A tree or plant that is infected to a much lesser degree than a primary host.

Seed-tree harvest system—Removal of mature trees in one cutting except for a small number of seed-producing trees left singly or in small groups.

Seed zone—A geographical area containing trees that produce seed requiring the same site conditions as found in that area for optimum seedling growth and survival.

Setal hyphae—Small, wiry, reddish-brown hairs found between sheets of decayed wood. The presence of setal hyphae is proof of laminated root rot.

Shelterwood harvest system—Removing mature trees in a series of cuttings over a relatively short portion of the rotation. This practice encourages establishment of essentially even-age reproduction under the partial shelter of seed trees.

Sign—The actual parts of the pathogen present or near the diseased plant. For example, mushrooms at the base of a tree may be a sign of Armillaria root disease. (See also **symptom**.)

Sp., Spp.—Abbreviations. Sp. means one species, spp. means two or more species within the genus named.

Species—A subcategory of organisms with one or more characteristics in common. For example, a species of pine is lodgepole pine, *Pinus contorta*. (See also **genus**.)

Spore—The principal reproductive units of fungi and other lower-order plants, containing one or more cells.

Sporophore—In a fungus, a mass of hyphae in a specialized structure for producing spores.

Susceptible—Likely to become damaged as a result of contact with a disease agent.

Symptom—The reaction of the host plant to disease. For example, yellowing foliage may be a symptom of root disease. (See also sign.)

Telia—The final-stage spores in the life cycle of a rust fungus.

Uredia—An orange to brown mass of hyphae and spores of a rust fungus, forming pustules that break the outer surface of the host plant.

Witches' broom—An abnormal development of many brushlike, weak branches or shoots at one point on a tree. A sign of disease, such as broom rust or dwarf mistletoe.

Zoospore—A spore, such as from a fungus, that can move independently through water.

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Common and scientific names of trees and plants mentioned in the text, and the page numbers on which they are discussed.

All figures are by Gregory M. Filip except the following: **Fig. 4**, p. 7, Walter Thies, USDA Forest Service, PNW Station, Corvallis, OR; **Fig. 5**, p. 10, Borys Tkacz, USDA Forest Service, Forest Pest Management, Flagstaff, AZ; **Fig. 6**, p. 13, Richard Mason, USDA Forest Service, PNW Station, LaGrande, OR; **Fig. 8**, p. 21, Douglas Miller, James Kimmey, and Marvin Fowler, USDA Forest Service, Forest Pest Leaflet No. 36; **Fig. 10**, p. 35, Jack Thompson, USDA Forest Service, Forest Pest Management, Missoula, MT; **Fig. 15**, p. 49, Debby Sundbaum-Sommers, Philomath, OR.

Extension Service, Oregon State University, Corvallis, Lyla Houglum, interim director. This publication was produced and distributed in furtherance of the Acts of Congress of May 8 and June 30, 1914. Extension work is a cooperative program of Oregon State University, the U.S. Department of Agriculture, and Oregon counties.