

## Management Guide for **Armillaria Root Disease**

*Armillaria ostoyae* (Romagnesi) Herink

### Topics

Introduction	1
<a href="#">Management overview</a>	2
<a href="#">Ecology of <i>Armillaria ostoyae</i></a>	4
<a href="#">Patch dynamics</a>	7
<a href="#">Resistance to <i>Armillaria</i> root disease</a>	8
<a href="#">Mode I disease</a>	9
<a href="#">Mode II disease</a>	10
<a href="#">Mode II mortality rates</a>	12
<a href="#">Mode II site predictors</a>	13
<a href="#">Modes I and II thinning</a>	14
<a href="#">Brush removal</a>	15
<a href="#">Mode III disease</a>	16
<a href="#">Mode IV disease</a>	17
<a href="#">Armillaria and forest succession</a>	17
<a href="#">Armillaria taxonomy</a>	19
<a href="#">Other reading</a>	20
<a href="#">Field Guide</a>	
<a href="#">Management Guide Index</a>	

### Key Points

- Know which type of *Armillaria* root disease you are managing.
- Manage for pines, larch, and cedar.
- Precommercial thinning may improve growth and survival of pines and larch.
- Avoid harvests that leave susceptible species (usually Douglas-fir or true firs) as crop trees.

**Hosts:  
Primarily  
Douglas-fir and  
true firs**

**All conifers may be  
damaged.**

***Armillaria* species are the most  
damaging and broadly distributed forest  
tree pathogens in the world.**

***A. ostoyae* is the most pervasive killer of  
Douglas-fir and grand fir in the northern  
Rockies.**

### *A Disease of the Site*

*Armillaria* root disease should be considered a “disease of the site”. That is, established mycelia of this fungus are essentially permanent, so the best course to minimize losses is to manage tree species that will survive on infested sites. In large areas of northern Idaho and western Montana, this includes most of the potentially best timber-producing sites.

Elsewhere in the northern and central Rockies, *Armillaria* is often less damaging and more easily tolerated. *Armillaria ostoyae* is a native pathogen with a broad host range but is most common and damaging on Douglas-fir, grand fir and subalpine fir. Mortality rates are highest on warm, moist habitats but large disease patches develop on dry sites and cold, high-elevation sites as well.

### *The Four Modes of Armillaria Root Disease*

Most patterns of *Armillaria* root disease can be identified as one of four types. Management options vary according to the mode of disease development.

- I. Distinct, generally large, root disease patches with single or few host species.
- II. Multiple clones merge forming essentially continuous coverage of sites. Grouped as well as dispersed mortality occurs throughout the stand. A mosaic of brushy openings, patches of dying trees, and apparently unaffected trees may cover large areas.
- III. Primary spread of infection from stumps of the previous generation results in clusters of mortality of seedling and

sapling trees, but secondary spread does not occur. By 20-30 years of age, root disease mortality has nearly ceased.

- IV. Root lesions of limited extent, often accompanied by butt rot probably resulting from primary inoculum from dead trees and stumps of a previous generation on the site. Little mortality results until advancing age or environmental stresses trigger extension of root lesions. Small groups of mortality result from limited secondary spread of the disease. Impact is generally low.

## *Management overview*

See 'About the Four Modes of Armillaria Root Disease' on page 9.



Figure 1. A 60-foot wide strip was destumped to control spread of Armillaria root disease at the advancing edge of the disease patch. [Photo by Robert James]

*Armillaria* mycelia can not be practically eradicated from a site but damage can be kept at tolerable levels with appropriate management. Selecting the management options best suited for each situation depends first on understanding the mode of *Armillaria* root disease. Other options may also be considered, depending on the mode of *Armillaria* root disease to be managed. Tables 1 and 2 will help identify the mode of *Armillaria* root disease in your forest.

### Root Disease Resistant Species

The most widely used and successful approach to controlling *Armillaria* root disease damage is through the use of disease tolerant or resistant species that are from a local seed source and are well adapted to the site (Table 2). Pines, western larch, spruces, western redcedar and hemlocks

are more resistant to *Armillaria* root disease than are true firs and Douglas-fir over most of western North America. There are notable exceptions to this rule, depending upon the mode of *Armillaria* root disease encountered and the location. Precommercial thinning, commercial thinning, and site regeneration offer opportunities to change species composition.

### Inoculum reduction

Stumps can be removed by pushing out with a dozer (fig. 1), or pulling up using a grapple. Root-raking removes root fragments from the soil. Push-over or pop-up logging, involves uprooting trees as part of the harvest operation. Roth and others (2000) reported that destumping by push-out logging was only effective in reducing mortality of the subsequent regenerated stand if accompanied by hand-picking of root fragments.

**Table 1. Diseases caused by *Armillaria*. (Exceptions are seen in all locations.)**

Location	Forest conditions	Damage	Management approach
Eastern Montana	Douglas-fir stands	<b>Mode I:</b> Discrete root disease centers, often large and aggressive, but not common.	Mark the locations of centers before harvest; use resistant species; remove inoculum in or around perimeter of center.
Western Montana, Northern Idaho	Broad range of habitat types with Douglas-fir and true fir components	<b>Mode II:</b> Present in most stands. Diffuse mortality and large and small root disease centers.	Highly significant losses usually requiring species conversion. Important consideration in management plans. Favor resistant species.
Northern Idaho	Lodgepole pine	<b>Mode I:</b> Rare, discrete root disease centers, can be large.	Aggressive disease seen in lodgepole pine and associated subalpine fir. Currently unmanaged.
Southern Idaho	Douglas-fir, subalpine fir	<b>Mode IV:</b> Small groups and individual trees killed.	Minor impact overall, Significant decay or defect from butt rot on some sites.
Southern Utah	Ponderosa pine, mature true firs, spruce	<b>Mode III:</b> Primary pathogen killing a few trees at a time	Usually minor impact. Favor resistant species in disease pockets.
Remainder of Utah	Douglas-fir, grand fir, pines, spruce, subalpine fir	<b>Mode IV:</b> Broadly distributed; mostly weak pathogen or saprophyte.	Causes little direct mortality so it is rarely directly managed.
Nevada	Douglas-fir, grand fir pines, spruce, subalpine fir, incense cedar	<b>Mode IV:</b> Broadly distributed; mostly weak pathogen or saprophyte.	Causes little direct mortality. Root disease pockets closely associated with endemic bark beetle populations. Rarely directly managed.

These procedures can significantly reduce the food base available to the fungus and delay infection of susceptible crop trees. On the other hand, direct inoculum removal is temporary in effect (the fungus often re-infests), expensive and may damage soil (Quesnel and Curran 2000) and other site amenities. It has been used to good effect in high value plantations such as orchards and in ornamental plantings.

**Avoiding Hazardous sites.**

Where root disease is limited to discrete patches that occupy relatively small areas, these patches may be excluded from timber management. Where root pathogens are more generally distributed, avoid managing highly susceptible species on the most hazardous sites. Moist grand fir, cedar and hemlock habitat types are hazardous sites in northern Idaho and western Montana. Douglas-fir and true firs may be especially poor risks on most of these sites.

**Chemical Control**

**Chemical soil fumigants that destroy *Armillaria* in root fragments are useful in orchards and vineyards. Stumps are removed to remove most of the large inoculum before fumigation. Chemicals can be protectants, eradicants or curatives.**

**Table 2. Common disease expression by host and location.**

Tree species	Location	Susceptibility	Typical disease
Douglas-fir	Eastern Montana	Highly susceptible at all ages	<b>Mode I.</b> Relatively rare; distinct root disease patches
Douglas-fir	Idaho north of Salmon River and Montana west of the continental divide	Highly susceptible at all ages	<b>Mode II.</b> Diffuse and concentrated mortality involving entire stands and drainages. Often severe by age 40. Most stands affected.
Subalpine fir	Idaho north of Salmon River and Montana west of the continental divide	Between Douglas-fir and grand fir in susceptibility	<b>Mode II.</b> Mortality often diffuse; also commonly large distinct disease patches.
Grand fir	Idaho north of Salmon River and Montana west of the continental divide	Highly susceptible at all ages, though somewhat less so than Douglas-fir	<b>Mode II.</b> Diffuse and concentrated mortality over large areas. Large trees often develop butt rot while root disease progresses slowly.
Pines, western larch	Idaho, Montana, Utah and Nevada	Highly resistant (with rare exceptions)	<b>Mode III.</b> Mortality common in saplings, but rarely significant in mature trees.
Western redcedar	Idaho and Montana	Moderately resistant	<b>Modes III and IV.</b> Some mortality in saplings. Residuals of partial harvests often develop severe infections but are very slow to die.
Engelmann spruce	Idaho, Montana, Utah and Nevada	Moderately resistant	<b>Modes III, and IV.</b> Mortality common in saplings. Old trees may have butt rot.
Engelmann spruce	Southern Utah	Moderately resistant	<b>Mode IV.</b> Mature stands on cool sites at high elevations may develop patches.
Grand fir	Southern Utah	Moderately resistant	<b>Mode IV.</b> Mature stands on cool sites at high elevations may develop small patches.
Douglas-fir	Northwestern Montana (Eureka area)	Moderately resistant	<b>Mode IV.</b> Butt rot often develops in mature trees but mortality is uncommon.
Incense cedar	Nevada	Moderately resistant	<b>Mode IV.</b> Rare in young trees. Older trees may develop butt rot.

### Pay attention to seed zones

Severe root disease is often seen in planting stock that is off-site, regardless of the expected disease resistance of the species.

### Maintain tree vigor

Tree vigor plays a role in *Armillaria* resistance on sites or species where the fungus is not an aggressive pathogen. In these situations, the fungus may be secondary to predisposing events such as insect attack, fire or logging injury, or severe drought.

Where the pathogen is aggressive, tree vigor is probably not an important factor. For example, in young coastal

Douglas-fir where *Armillaria* root disease is often quite damaging, tree vigor, as the growth efficiency of trees, was not a factor in determining later infection and mortality. On permanent plots monitored for at least 10 years, Rosso and Hansen (1998) found that the biggest and fastest growing trees were as likely to die from *Armillaria* as the smallest and poorest trees.

## Ecology of *Armillaria* in forest ecosystems

*Armillaria ostoyae* is commonly found infecting the same trees and even the same roots with other root pathogens.

### Persistent and expanding mycelial clones

New clones result from basidiospore-infection of available substrates. Survival rates of newly established mycelia are probably exceedingly low. This is evident in the relative stability of clones on sites, which are often estimated to be in excess of 1000 years of age (Shaw and Roth 1976, Smith and others 1992, Ferguson and others 2003). An individual derived from a single mating of haploid spores (forming a diploid mycelium) is called a 'genet'.

others (2003) in the Blue Mountains of Oregon ranged from 20 to 965 hectares. This included the largest genet reported to date. It covers 2,200 acres (965 hectares) and spans 3.5 miles (5.6 km). See the '*Armillaria ostoyae* clones' sidebar on page 7.

Established mycelia expand outwardly, provided there is suitable substrate. (Read 'Anatomy of a disease patch' on page 7.) *Armillaria* species are capable of forming specialized structures called rhizomorphs, which grow root-like through soil. However, *Armillaria ostoyae* produces few rhizomorphs (Cruickshank and others 1997), relying, instead, on growth of mycelium along and within tree roots to facilitate spread. Expansion rates have been estimated to be 0.7 to 1.3 m/yr (2.3-4.3 ft./yr) in Douglas-fir plantations in southern interior British Columbia (Peet and others 1996).

A similar spread rate (1 m/yr) was reported for a young, naturally regenerated ponderosa pine stand in Washington (Shaw and Roth 1976).

In mature (110-yr old) Douglas-fir in central interior British Columbia van der Kamp (1993) estimated the average spread rate of *A. ostoyae* to be 0.22 m/yr (0.7 ft/yr), about a third that observed in young stands.

### Genet

A genet is an individual derived from compatible spore pairing; a diploid mycelium.

A single **genet** can produce multiple **clones** (ramets) by fragmentation.

Fragmentation may result from loss of substrate, or replacement by competing fungi in portions of an area occupied by a genet. Isolation of portions of the genet results in multiple clones that are genetically identical.

**New genets originate from a single mating between two haploid spores. The resulting mycelium spreads by vegetative growth of mycelium and rhizomorphs.**

Most recognizable mortality patches are probably a single genet (Dettman & van der Kamp 2001a). Genets vary greatly in size, probably depending on site conditions and history. Dettman and van der Kamp found most genets in southern interior British Columbia to be less than 2 hectares in size (2001b) and those in the central interior ranged from less than one to more than 15 hectares (2001a). Several remarkably large, and potentially very old clones have been found. Those described by Ferguson and

## Saprophytic and parasitic existence

*Armillaria ostoyae* can survive as a saprophyte on dead organic matter such as old stumps and roots for several decades.

Even small debris on a site may harbor significant amounts of the fungus. Komroy and others (2005) isolated *Armillaria ostoyae* from as much as a third of small (<2 cm) woody fragments in the upper layers of soil. They also isolated *A. ostoyae* from a number of deciduous tree and shrub species including aspen, honeysuckle and blueberry. Though not considered a primary parasite of these species, the fungus is clearly capable of utilizing a wide variety of substrates to maintain itself on a site.

Hyphae of *A. ostoyae* penetrate wood, causing a 'white rot' type of decay in which both cellulose and lignin are degraded. The mycelium spreads from these woody substrates to the roots of live trees either through direct root contact with infected wood, or by rhizomorphs.

Once established on a root of a live tree, the fungus invades and kills the cambium of the root and the decays the dead root tissues. The mycelium may eventually travel up the root to colonize the root collar, and girdle the tree.

## Growth declines

Decay and girdling are usually slow processes that can be delayed for extended periods while the fungus remains latent in non-expanding lesions. Growth decline of infected trees has been detected for 30 years or more before death in mature Douglas-fir (Bloomberg and Morrison 1989). The longer the period of decline before death, the greater the cumulative growth loss. Smaller trees typically have shorter detectable periods of decline before death than larger trees (Bloomberg and Morrison 1989).

In young trees, terminal growth decline is often observed for only one or two years before death. Much also depends on the relative aggressiveness of the pathogen and whether bark beetles attack the weakened tree.

## Butt rot may develop

Under some conditions, the fungus establishes infection in the heartwood of the roots and in the lower stem (butt) of the tree. Generally the taproot falls victim at an early age and the pathogen travels from the decayed taproot directly into the butt heartwood. The fungus can produce a large cavity of decay in the heartwood of the stem, usually extending less than three feet above the ground. Grand fir, cedars, hemlocks and spruces commonly develop this mode of disease which can persist for many decades without killing the tree.

## Spores abundant but rarely successful

Honey-colored mushrooms may be produced at the base of infected trees during late summer or early autumn. The role of spores from these mushrooms in disease development is not well understood.

At times the fungus seems to invest tremendous energy in the production of these sporophores. Hundreds of pounds of mushrooms can be collected from a site at one time. Each mushroom is capable of producing hundreds of thousands of spores. And yet, no evidence of direct infection of living trees by basidiospores exists. No inoculations of live hosts using basidiospores have succeeded.

Most likely, *Armillaria* spores establish on woody debris initially. The survival rate of even those spores that manage to land on a suitable substrate are probably exceedingly low. Thus, the great abundance of basidiospores may be necessary to ensure the occasional success.

## Rhizomorphs

*Armillaria* root disease has been called

"shoestring root rot"

because of the appearance of rhizomorphs. These root-like structures are used to seek out new substrates.\*

Rhizomorphs are highly differentiated aggregations of hyphae surrounded by a dark cortex of protective cells. They grow from a tip that resembles the meristem of a root.

Rhizomorphs facilitate movement of the fungus through soil and under bark. They grow from inoculum, usually roots or stumps of killed trees, to suitable substrates.

*Armillaria ostoyae*

produces relatively few rhizomorphs compared to many other species of *Armillaria*. They have a dichotomous branching pattern (Morrison 1989).

Probably because of inoculum potential, rhizomorph-originated infections are less successful than are infections spread from root contact with inoculum (Robinson and Morrison 2001).

\*There is evidence rhizomorphs also provide water, nutrients and oxygen to the mycelium.



Figure 2. Fire probably has little direct effect on *Armillaria* inoculum in roots that are more than a few inches below the ground surface.  
(Photo from USFS files)

### Fire

Fire has been a historically important factor in shaping forest composition and structure in northern and central Rocky Mountains. Based on the considerable age achieved by *Armillaria* clones, it is reasonable to conclude that they typically survive forest fires. There is little direct evidence of the effects of fire on established *Armillaria* mycelia but abundant anecdotal evidence suggests that the fungus is capable of surviving and thriving in subsequent regenerated stands. This is to be expected

because most of the inoculum resides deep in root systems.

Filip and Yang-Erve (1997) tested survival of *Armillaria ostoyae* in buried wood on sites broadcast burned in fall and spring. In general, burning had no effect on inoculum survival although a reduction of viability was observed in inocula buried nearest the surface in the fall-burning treatment. The burn treatments were not accomplished according to plan, so it remains unclear whether slash burning affects shallow inocula.



Figure 3. Primary spread of infection from stump or snag roots to trees following fire or harvest.  
(Photo by John Schwandt)

## Primary vs. secondary spread

### Primary spread (Figure 3)

In inland western forests, root disease of stands established after harvest or severe fires generally results from infections spreading from primary inoculum (stumps or buried woody material from the previous stand). Inoculum potential slowly declines as the roots of stumps and snags deteriorate. Although the disease can be severe in the first few years after stand establishment, mortality of resistant species, such as pines and larch, generally declines after two or three decades, often with little lasting impact on stands.

Primary and secondary spread of disease is common in Douglas-fir stands in inland forests; however in coastal forests of the Pacific Northwest from Oregon to British Columbia, Douglas-fir acts more like inland ponderosa pine. The disease may be severe in young stands for a decade or two after harvest but subsides thereafter. Secondary spread of the disease does not play a significant role.

Similarly, while ponderosa pine is relatively resistant to secondary spread in most inland western forests, in some natural pine stands in central Washington both primary and secondary disease spread is common (Shaw and Roth 1976). On these sites, Douglas-fir is considered to be relatively resistant to root disease.

### Secondary spread (Figure 4)

Also in inland western forests, root disease of Douglas-fir, grand fir and, probably, western redcedar results from both primary and secondary spread of the fungus. Secondary inoculum is produced on roots of *Armillaria*-killed trees. The fungus is capable of maintaining very large mycelia for at least several centuries by means of secondary inoculum.

The ability of trees to wall off root infections and, eventually slough those infections, limits secondary spread of *Armillaria*.

**Secondary disease spread varies by location**



Figure 4. Secondary spread from tree to tree may continue throughout the life of the stand. (Photo by Susan Hagle)

***Armillaria* often becomes the primary pathogen in regeneration after a fire or harvest, even though laminated or annosus root diseases were dominant in the pre-harvest stand.**

## *Waves of mortality and regeneration in root disease patches*

Wave patterns of mortality and regeneration are most clearly observable on sites that have no cutting history and large infection patches. At the margins of the disease patch, the slow advance of the fungus into the non-diseased portion of the stand produces the initial wave of mortality (See ‘Anatomy of a root disease patch’ below). This is followed by a wave of regeneration in the canopy opening, then slow mortality of seedlings and saplings. When the survivors of this cohort of regeneration reaches roughly pole size, the food base will be sufficient to fuel a second wave of mortality in what is now the older portion of the disease patch.

The result is a stand with zones representing the temporal sequence of root disease development in the stand. These zones form concentric rings from the center to the perimeter of the disease-affected area .

The primary difference between the wave pattern in root disease infected stands on sites with no cutting history and that on sites that have been cut is that the harvest sets the timing of the wave by stimulating the regeneration that will reach pole size at about the same time.

Primary spread from stumps results in new infections in these young trees. As in the uncut stand, the young trees die a few at a time until the survivors are large enough to provide a substantial food base for the fungus. At this point, secondary spread will accelerate the rate of mortality. As trees die, they are replaced by abundant regeneration. Mortality then slows until sufficiently large root systems have been produced to fuel another wave of mortality. Recognizable root disease patches eventually re-emerge in cutover stands as groups of trees are killed and openings are regenerated.



[Photo by James Byler]

In the older portions of the patch (shaded), some trees reach sufficient size to produce secondary inoculum. This leads to clusters of mortality (B) in older portions of the patch.



### **Anatomy of a root disease patch**

Trees from the original stand die at the margins of the disease patch (A).

A zone of open canopy, heavy fuel-loading and new regeneration can be seen just inside the patch margin, where recent mortality has expanded the patch area (yellow).

### **Armillaria clones are the largest organisms known to man.**

#### **Big**

The original “humongous fungus” is 38 acres (15 hectares), 1500 to 10,000 years old, weighs about 100 tons. It is *A. gallica*. Found living in the upper peninsula of Michigan and was widely covered in the press; even on a TV talk show (David Letterman).  
(Smith and others 1992)

#### **Bigger**

1,500 acres (600 hectares), *A. ostoyae*, Southeast Washington (Shaw and Roth 1976)

#### **Biggest**

2,400 acres (965 hectares) and estimated to be at least 2,400 years old, *A. ostoyae*, in the Blue Mountains of eastern Oregon. It stretches 3.5 miles (5.6 km) wide and covers an area larger than 1,600 football fields.  
(Ferguson and others 2003)

## Inoculum Potential

**Energy is required for *Armillaria* to overcome a tree's defenses. When the fungus is able to establish on a large root mass, it has a large food base to provide energy, which means it has a large inoculum potential.**

Low inoculum potential of rhizomorphs may render them less effective in establishing infections than mycelium growing directly from a more substantial food base.

**The main differences in host resistance appear to be determined by the frequency and longevity with which lesions are restricted by secondary periderms.**

Secondary periderms may have several layers of phellem. The tree root may have several successive necrophylactic periderms that have been produced as each previous periderm was breached.

Most lesions are halted in the bark or cambium of roots. If prevented from expanding long enough, periderm-surrounded infections will eventually be sloughed (as the root grows) and the root will heal.

## *Mechanisms of Resistance to Armillaria root disease*

*Armillaria ostoyae* occurs throughout Europe and north America and is primarily a pathogen of conifers. It is also known to attack and kill some hardwoods growing in association with conifers including birch (Morrison and others 1985) and aspen (Pankuch and others 2003). Although all native forest conifers in the Rocky Mountains are considered susceptible, differences are readily observed. The effectiveness of resistance mechanisms of conifers vary by host and pathogen species and by the age of trees.

Bear in mind that there is a world of difference between a pathogen simply being capable of infecting a host, and that pathogen causing sufficient disease in that host to present a management challenge.

### Primary Defense—Chemical

Root bark of conifers contains phenolic compounds that are inhibitory to *Armillaria ostoyae*. Larch has much higher concentrations of phenols in the root bark, compared to Douglas-fir or grand fir (Entry and others 1992). Ponderosa pine and western white pine were intermediate. These chemical defenses probably prevent most infections. These authors also suggest that the relatively higher sugar content in Douglas-fir and grand fir bark contributes to the success of the pathogen.

Resin production in response to infection of the cambium presents at least a temporary physical barrier as well as biochemical. Time and energy are required to digest resins in order for hyphae to penetrate resin-soaked tissues.

### Secondary Defense—Physical Barriers

As a secondary defense, a tree under attack will attempt to

“compartmentalize” a wound or infection (Tippet and Shigo 1981). This is a relatively rapid, generic, response of the tree to wounding or pathogenic infection. In effect, the cells near the infection site, that were present at the time of infection, are fortified. The cell walls become lignified and suberized. The cells also are made toxic to invading hyphae through the deposition of secondary metabolites (phenolic compounds, mostly). Within months, trees will produce an organized periderm to wall off the infection.

Referred to as necrophylactic periderms, they consist of single or multiple phellem layers which wall off infected tissue. These periderms delay extension of the infection but they may be eventually breached by hyphae. The main differences in host resistance appear to be determined by the frequency and longevity with which infections are restricted by secondary periderms.

Two important studies compared a relatively resistant species (western larch) to a highly susceptible species (Douglas-fir) at three ages (Robinson and Morrison 2001, Robinson and others 2004). At 6-11 years of age both tree species were readily infected, girdled and killed by *Armillaria ostoyae*.

Resistance was more effective in 18-19 year-olds of both species but differences between larch and Douglas-fir were evident. Necrophylactic periderm formation had delayed the advance of nearly half of the infections in larch and only a fourth of those in Douglas-fir (Robinson and Morrison 2001).

At 25-27 years of age, the necrophylactic periderms formed by larch roots were far more impervious to breaching (55% intact) than those in Douglas-fir (none intact) (Robinson and others 2004).

Larch periderms also were more often replaced if breaching did occur. These studies suggest that resistance develops in larch somewhere between 8 and 15 years of age but much later in Douglas-fir, probably closer to 35 years.

Most of the lesions on older trees are contained in the bark or outer portions of the root near infected cambium (Robinson and Morrison 2001). If prevented from expanding long enough, these infections will eventually be sloughed and the root healed. If the pathogen manages to girdle the root cambium, the distal portions of roots are killed and decayed by the pathogen (Shaw 1980). At some point the inoculum potential developed by the fungus on these killed roots may become sufficient to allow it to breach the periderm. The host is often seen to lay down repeated periderms following each breaching. Larger roots, nearer the

root collar, react more aggressively and are more successful at containing infections.

### Timing is everything

Young trees have small root systems that provide little inoculum potential when overcome by the pathogen (figure 5). Therefore, the early susceptibility of larch and pines probably contributes little to secondary spread of *Armillaria* root disease. The converse is also true; as older, larger trees are killed the mass of each root system contributes significantly to the inoculum potential of the pathogen. As inoculum potential increases, secondary spread of the disease also increases. Therefore, the delayed resistance of Douglas-fir probably contributes greatly to the increases in inoculum potential and to the secondary spread observed in Douglas-fir forests. This may hold for true firs as well.



Figure 5. Although susceptible at a young age, the surviving western white pine will become resistant before they are large enough to contribute significantly to inoculum potential on this site. With little potential for secondary spread, *Armillaria* root disease will decline. [Photo by James Byler]

## About the Four Modes of *Armillaria* root disease

### MODE I — DISTINCT ROOT DISEASE PATCHES

The etiology (pattern of development) of *Armillaria* root disease mode I is best understood. This disease mode is typified by more or less round patches of root disease-killed trees (figure 6). They are ringed with newly dead and dying trees and usually have advanced regeneration in their centers, where young trees have taken advantage of the disease-caused opening.

These disease patches increase in radius at a rate of about 0.2–1.3 m (0.7–4 feet) per year. As susceptible trees at the margin are encountered, the pathogen infects, girdles and kills these trees and then uses the root system to spread to adjacent live trees. In the interior of the disease patch, susceptible trees regenerate and grow in the opening. They are in turn killed as their roots encounter inoculum in the decaying roots of the previous generation.

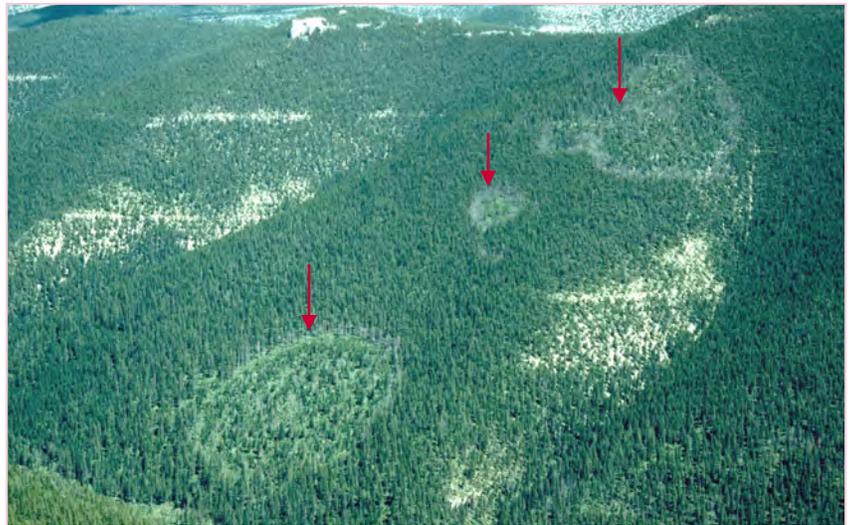


Figure 6. These three root disease patches in a Douglas-fir forest in eastern Montana form distinct fairy-ring patterns when observed from a distance. Discrete patches with clearly defined edges are typical of Mode I *Armillaria* root disease. [Photo by Ralph Williams]

## Managing Mode I

### Resistant species

If there are resistant tree species that are suited and economically feasible to grow on the infected site, this is usually the most effective and efficient means of reducing damage.

### Avoid infected sites

This may be an option for this type because the infected area may be fairly limited and distinct. The fungus is likely to continue to expand at the margins of the disease patch if susceptible trees are present.

### Reduce inoculum

Stump removal, especially around the perimeter of the disease patch may contain the fungus. Removal within the disease patch can be expected to reduce but not eliminate the disease.

## Root disease patches in eastern Montana

Root disease patches east of the Continental divide in Montana appear to fit this etiology very well. *Armillaria* is nearly always the only disease of consequence in these patches. They are typically seen in Douglas-fir stands of uniform age, and density. The disease patches are strikingly uniform in shape (round or oval) and profile. The profile (figures 6 & 7) shows the largest advanced regeneration in the center of the patch. The size and age of regenerated trees declines toward the margins of the patch where an increasing number of still-standing dead trees are seen. In the margins, recently dead, red trees are mingled with thin-crowned dying trees and those showing little or no apparent decline.



Figure 7. Mode I *Armillaria* root disease patches have a distinct margin. The interior of the patch has successively older regeneration nearer the center of the patch. [Photo by Ralph Williams]

These patches probably represent single successful genets established hundreds or thousands of years ago. They survive on one generation of trees after another as wildfires sweep through on long intervals to restart stands on the site. Douglas-fir generally occurs as single-species stands in this situation.

## Disease patches on dry sites

Dry site types, such as those with Douglas-fir habitat types (Dry Biozone in British Columbia) generally have a lower incidence and smaller extent *Armillaria* genets than do moist sites (Byler and others 1992, Morrison and others 2000). They commonly form distinct patches in a forest typifying Mode I root disease.

## Lodgepole pine and subalpine fir in northern Idaho

Large, apparently single-genet *Armillaria* disease patches are uncommonly seen in lodgepole pine stands and mixtures of lodgepole pine and subalpine fir in northern Idaho. Here, as in the Douglas-fir stands of eastern Montana, the fungus aggressively eats away at the margins of the disease patch while infecting the trees that regenerate in the older, central portions of the disease patch.

## MODE II — PATCH AND DIFFUSE MORTALITY

Mode II *Armillaria* root disease is by far the most common mode in Idaho and western Montana. The disease probably has a similar etiology to Mode I but there may be many more clones, perhaps larger individuals and, sometimes, more variety of both host and root pathogen species involved. The profile of a diseased stand is much more complicated than that of Mode I, but the development of the disease may not be. As in Mode I disease,

primary spread from killed trees and stumps following disturbance such as stand-replacement fires and regeneration harvests establish the pathogen in the subsequent generation of trees on the site.

Secondary spread from roots of infected trees to adjacent trees results in mortality beyond the reach of infected stump roots and maintains the fungus long after the stumps have rotted away.

**66% of acres in northern Idaho and western Montana have significant root disease impacts.**

**Most is due to Mode II *Armillaria* alone or in combination with *Annosus* root disease or laminated root rot.**

A wave-like pattern of mortality is evident but made more difficult to observe as patch boundaries merge.

Mode II *Armillaria* is most typical of root disease in Idaho and western Montana. Douglas-fir is typically the most-damaged host with grand fir, white fir and subalpine fir also very susceptible. As individual clones

of *Armillaria* merge they produce extensive disease patches (figure 8). Studies of naturally occurring *Armillaria* colonies have shown minimal overlap of individual clones of the species indicating that they are capable of excluding other individuals of the same species (Smith and others 1992, Ferguson and others 2003).



Figure 8. Dispersed and concentrated groups of mortality and irregular clusters of regeneration mark this hillside on the Nez Perce forest in Idaho with a long history of Mode II *Armillaria* root disease. The boundaries of patches are indistinct with continuous root disease symptoms extending for several miles in all directions in this forest type. [photo by Susan Hagle]

Dettman and van der Kamp (2001) studied the distribution of *Armillaria ostoyae* in moist forests (Interior Cedar-Hemlock) of southern interior British Columbia (presumably similar to that across the border in northern Idaho). They found 88% of the forested area to be infested, with genets less than 2 hectares (5 acres) in size. The genets were uniformly pathogenic and probably mostly very old and stable.

Root infection is common, even in young trees. Morrison and others (2000) estimated that about 38% of 13-24 year old trees were infected on moist sites in southern interior British Columbia

(comparable to grand fir habitat types in Idaho and Montana). Thirty two percent were infected on wet (cedar and hemlock habitat types) if there were dead trees present. Even where no dead trees were present, 25% on moist and 15% on wet types were infected. As stands age, these infection rates increase steadily. By comparison, only 10% of trees on dry sites were found to be infected even with mortality present. On dry sites, in southern interior British Columbia, Idaho and Montana, *Armillaria* root disease tends to fit the Mode I pattern. Genets are fewer leaving most of the forest uninfected (Byler and others 1992, Morrison and others 2000).

## Managing Mode II

### Resistant species

For Mode II *Armillaria* root disease, conversion to disease resistant species is usually the only practical method to control damage.

Increasing proportions of less-susceptible species to 60% may greatly reduce damage to susceptible trees by interfering with root transfer of the fungus on site.

Species diversity can be enhanced with *Armillaria ostoyae* resistant hardwoods such as birch or mountain maple.

The presence of many overlapping clones, and nearly continuous distribution over large areas limits the application of inoculum reduction and hazardous site avoidance.

Partial harvests that leave a large composition of susceptible species may produce the undesirable outcome of losing most of the remaining trees within a few years.

**HOW FAST DO THEY DIE?**



**Douglas-fir 17 to 23 years of age took an average of one to three years to die after basal resinosis was detected.**

**(Morrison and Pellow 1974)  
(Photo by Susan Hagle)**

**Sources of data for graph:**

- Byler, Marsden and Hagle 2006, Root disease-caused tree mortality in northern Idaho stands: 22 year monitoring results and implications. (In preparation)
- Hagle, Marsden and Welborn, 2006. Indicators and patterns of conifer mortality caused by root disease. (In preparation).
- Hagle, 2005. Root disease and associated bark beetle mortality estimation for intermountain west risk map. Summary of results from 758 permanent plots in northern Idaho and western Montana, monitored 8-18 years. In-house report.
- Morrison and others 1988
- Morrison and Pellow 1994

In mixed conifer forests of the Blue Mountains in northeast Oregon, Ferguson and others(2003) measured genets ranging from 20 to 965 hectares (49-2,385 acres) in size. They estimated the age of genets to range from 1,900 to 8,650 years. Although similar in most ways to *Armillaria* root disease in northern Idaho, in these stands true fir species appear to be more susceptible to the disease than Douglas-fir.

**Expected mortality rates in Mode II disease**

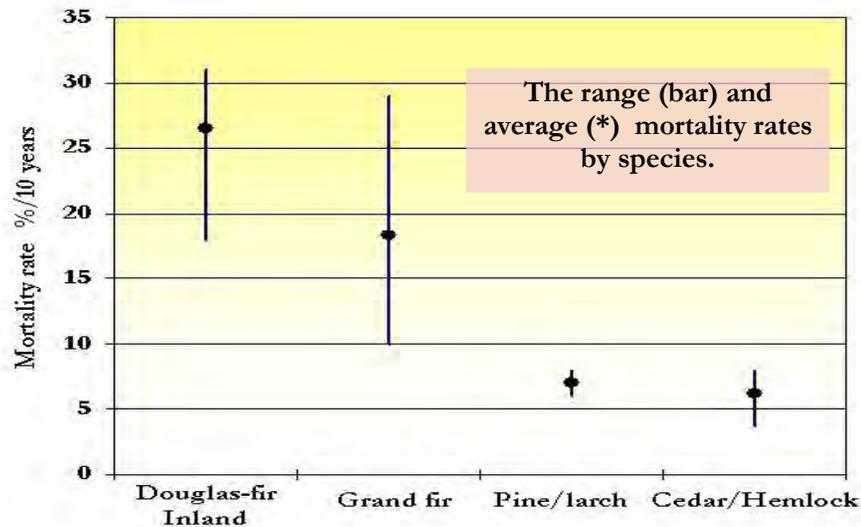
Mortality rates vary by stand age, species composition and site quality. In the northern Rocky Mountains, Douglas-fir and grand fir mortality rates are considerably higher than those of pines, western larch, western redcedar and western hemlock (figure 9). From nine studies that have employed permanent plots to monitor root disease mortality of forest trees, results are remarkably consistent. The primary causes of mortality on plots in the northern Rockies were usually a combination of *Armillaria ostoyae* and *Phellinus weirii* (fir type). Elsewhere, it was almost entirely

*Armillaria ostoyae*.

In unthinned stands, mortality rates of ponderosa and lodgepole pines, western redcedar and western hemlock averaged 1.5% to 10% per decade. Likewise, mortality rates for coastal Douglas-fir were less than five percent per decade. Young western larch averaged 18% mortality in one study (Morrison and others 1988). The rate of mortality of larch declined throughout the 14-yr monitoring period. Grand fir was variable, ranging from 10 to 30% per decade. Douglas-fir mortality averages ranged from nearly 20% to a little more than 30% per decade in the various studies. Little is known about mortality rates of subalpine fir but they appear to fall between those of Douglas-fir and grand fir.

In general mortality rates of pines, coastal Douglas-fir and western larch declined with age and thinning. The opposite was the case for inland Douglas-fir and grand fir. Mortality rates ranged from 13%/decade in 5-19 yr old Douglas-fir (Morrison and others 1988) to 30%/decade at 100 yrs of age (Byler and others, in preparation).

**Figure 9. Root disease mortality rates of trees at least 20 yrs old.**



**Site Predictors of Mode II Armillaria root disease**

Site factors such as moisture and temperature show promise as indicators of disease-proneness.

**Disease incidence**

Incidence of *Armillaria* root disease is highest on hemlock, grand fir and cedar habitat types. On the Lolo National Forest, Byler and others (1990) reported a high of 80-88% of stands with root disease (mountain and western hemlock habitat types) to a low of only 13% (ponderosa pine habitat type). Half of the stands on grand fir habitat types had root disease. A third or fewer were afflicted on other habitat types (figure 10). Species composition interacts significantly with habitat type to influence root disease prevalence. In this case, many stands on cedar habitat types were dominated by western redcedar and grand fir with few live Douglas-fir remaining whereas Douglas-fir was a primary component on grand fir, Douglas-fir and western hemlock habitat types. Species composition alone was not a good predictor of root disease because of the strong influence of habitat type.

Surveys of Douglas-fir stands on moist cedar-hemlock in southern interior British Columbia found root disease on 88% of hectares (Dettman and van der Kamp 2001b). An assessment of the Coeur d'Alene Basin National Forest (figure 11) uncovered root disease in 98% of stands with Douglas-fir and grand fir cover types, across all habitat types, but the highest severity classes were most common on grand fir and cedar/hemlock habitat type groups (Hagle and others 1994).

**Disease severity** is highest on grand fir and moist subalpine fir habitat types with Douglas-fir, grand fir or subalpine fir forest types. The most severe condition, root disease patches in which few

trees survive, have been reported to occupy 5.1% of the Coeur d'Alene Forest (Williams and Marsden 1982). This agrees well with a later assessment of root disease on the Coeur d'Alene Basin Forest in which 5.8% of stands had severe root disease, defined as having 75% or greater loss of canopy to root disease (Hagle and others 1994). Approximately one third of the Coeur d'Alene Basin Forest has Douglas or grand fir cover type. Williams and Marsden (1982) reported finding most of the disease patches on grand fir or western hemlock habitat types. Hagle and others found severe disease most common on grand fir, western hemlock or moist subalpine fir habitat types. Mortality rates of Douglas-fir and grand fir are highest at a relatively younger age on highly productive sites such as western redcedar and western and mountain hemlock habitat types (Hagle and others, in preparation).

Visually estimated root disease severity (Hagle 1992) is a measure of cumulative root disease impact in a stand. Recent analysis of permanent plot data from north Idaho found the root disease severity assigned at the time of plot establishment was the best predictor of mortality in the subsequent 20 years. Figure 12 illustrates this relationship (Hagle and others, in preparation).

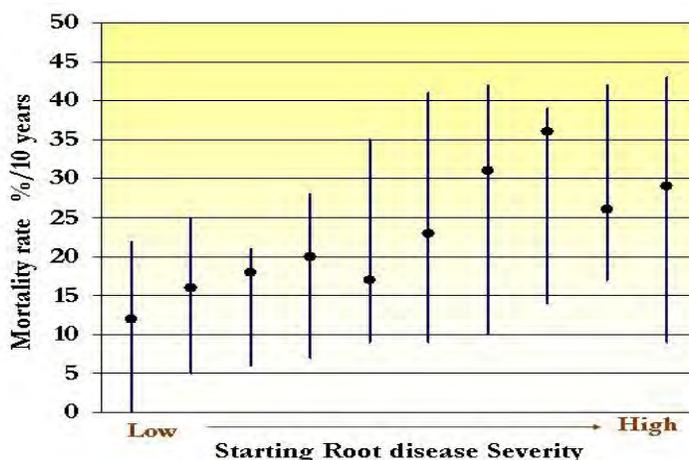
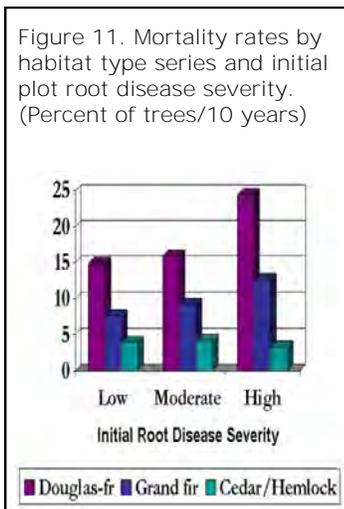
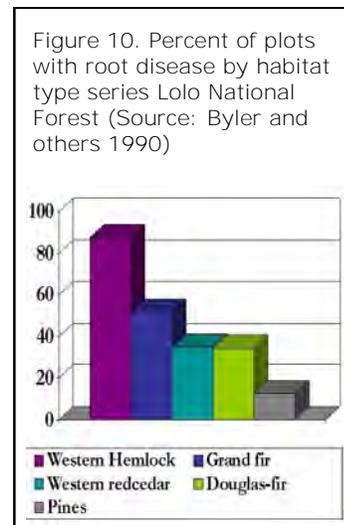


Figure 12. Mortality rates (all tree species) from permanent plots in Idaho and Montana.

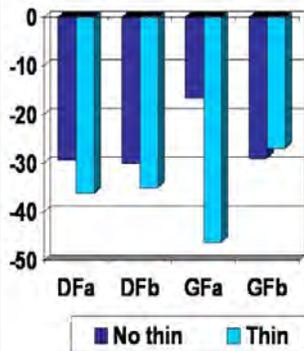
## *Thinning in Modes I and II Armillaria root disease*

### *Commercial thinning*

#### Comparing commercially thinned and unthinned stands

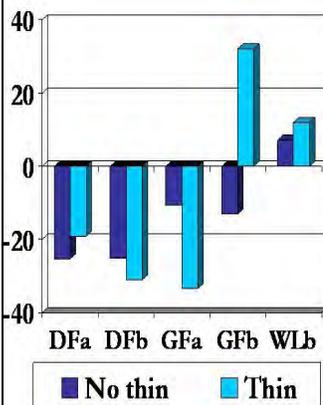
**Figure 13. Root disease mortality; trees**  
Mature Douglas-fir and grand fir

Percent change: stems/10yrs



**Figure 14. Root disease mortality; volume**  
Mature Douglas-fir, grand fir and western larch

Percent change: volume/10yr



#### Data sources:

- (a) Hagle and others (in preparation) 213 permanent plots monitored 20 years.  
 (b) Byler and others (in preparation) 12 permanent plots monitored 22 years.  
 Both studies located on the Idaho Panhandle National Forest in northern Idaho.

It is fairly safe to say that *Armillaria*-afflicted stands will not respond favorably to commercial thinning if susceptible species are left. Mortality rates vary greatly by location and through time. By the time stands reach commercial size many have high mortality rates. If mortality rates in a stand are already high, increases in mortality after thinning tend to be small or none. However, where mortality rates were initially moderate or low, increases can be dramatic following thinning.

Most reports are from retrospective studies comparing thinned stands to uncut stands. For example, comparing selectively harvested stands to uncut stands, Morrison and others (2001) observed a trend in infection and mortality rates, subsequent to harvest, that was associated with site quality. Rates were low on dry sites, moderate on wet sites, and high on moist sites. All site types had slightly higher mortality rates if stands were selectively harvested compared with the uncut stands. Differences were statistically significant in two out of four comparisons.

Byler and others demonstrated differences in response between Douglas-fir and grand fir where laminated root rot and *Armillaria* root disease were both primary causes of mortality (unpublished report). The permanent plots, in northern Idaho, were monitored for 22 years after commercial sanitation thinning (with unthinned controls). Mortality rates (percent of trees) for Douglas-fir remained fairly consistent between the two time intervals regardless of whether stands were thinned or not, and regardless of the time following thinning (Figure 13). Grand fir, however, had a low initial rate of mortality after thinning. This rate nearly doubled between 15 and 22 years after

harvest, compared to the first 15 years. Also, in the Byler study, Douglas-fir timber volume decreased at both 15 and 22-year remeasurements post-treatment (figure 14). However, grand fir volume increased in the first 15 years following thinning then declined rapidly. Grand fir appeared to respond positively to sanitation thinning for at least the short term, leading the authors to suggest that grand fir may be an acceptable leave tree choice when a regeneration harvest is to follow within 10-15 years.

Hagle and others (unpublished) found the grand fir decline to begin sooner, between 8 and 10 years after thinning with a net loss of 67% in 20 years in the thinned stands compared to 22% loss in the unthinned stands (figures 13 & 14). Douglas-fir mortality rates were slightly higher if measured as proportion of stems, or not significantly different, if measured as proportion of volume following thinning. In both Byler and Hagle studies, the largest and most vigorous-appearing trees were retained during the thinning. Thinning did not improve survival of remaining Douglas-fir or grand fir.

Morrison and others (2001) found that, on moist sites, as much as 90% of live, codominant trees in undisturbed stands had *Armillaria* root lesions. Root systems remain alive for a time after the tree is severed, during this time they can prevent colonization by saprophytic fungi but the *Armillaria* lesions are able to expand (Shaw 1980). During this time the stumps may become extensively colonized by pathogenic fungi. This increase in inoculum potential allows the pathogen to spread from roots of stumps to residual trees and post-harvest regenerated trees.

### *Precommercial thinning in modes I and II*

Even small stumps, from precommercial thinning have been found to be colonized by *Armillaria* spp. Cruickshank and others (1997) reported finding up to 77% *Armillaria* infection in precommercial thinning stumps in British Columbia. Infection rates were highest on interior cedar/hemlock sites, where rates ranged from 28 to 77% and averaged 51%. Inland Douglas-fir sites averaged about one third. Lowest rates were in the coastal forests (average 12-22%).

About half of the individual stump root infections lead directly to infection of Douglas-fir crop trees; again the rates were a little higher in the interior cedar/hemlock type. These sites develop Mode II disease so these early infections have the potential to lead to considerable long term impacts. Resulting mortality has not, as yet, been monitored.

Although little can be said, as yet, about mortality rates of Douglas-fir, grand fir or subalpine fir after thinning, we have fairly reliable data suggesting that resistant species improve growth and survival following precommercial thinning.

In stands or portions of stands with low root pathogen incidence, precommercial thinning may provide satisfactory results. The increase in growth of the residual stand may offset subsequent mortality enough to justify the thinning investment. However, careful examination of the candidate stand is in order because even a moderate rate of infection may not be obvious in young stands. Young trees die and lose their foliage quickly. Close inspection of small openings in young stands may reveal more mortality than is otherwise apparent. Precommercial thinning, especially in sapling-size trees may not greatly increase

the long-term losses but the money spent thinning may prove to be poorly spent if few of the residual trees reach maturity.

**If less susceptible species are favored in thinning and the resultant stand has an improved species composition, thinning can be an excellent investment.**

Western larch and lodgepole pine are particularly sensitive to lateral competition (crowding). Thinning may be required to maintain these species even where root disease mortality rates are expected to be fairly high among competing Douglas-fir and grand fir. Large root pathogen biomass may be maintained by dying Douglas-fir and grand fir. *Armillaria* attacks on larch and pines may be more frequent under these conditions, with subsequently higher rates of mortality and lower rates of growth in these species as well.

Also a factor, is the delay between crown closure and the highest rates of root disease mortality. It may take decades after crown closure before sufficient root disease mortality has occurred to open the canopy. In many cases, the easing of crowding occurs too late to benefit intolerant species; larch and lodgepole pine, in particular.

The infection rates detected in young, unthinned stands that have few above-ground symptoms suggests that new infections resulting from thinning may not greatly alter the impact on stands. As stated earlier, Morrison and others (2000) found 32-38% of saplings to have root infections on moist and wet sites in interior British Columbia. Mortality rates were low in these stands at this age but could be expected to increase as the infections spread across the tree root systems.



Figure 15. Precommercial thinning and weeding, removed mostly Douglas-fir and grand fir while retaining mostly western larch. These released larch can be expected to become increasingly resistant to *Armillaria* root disease.

To the extent that thinning increases growth, it probably also increases infection rates as the larger root systems contact more inoculum. In trees of resistant species with good growth, this increase in contact with inoculum is presumably offset by the ability of the tree to contain and eventually shed infections. This is demonstrated through lower

mortality rates of resistant species following thinning (Filip and others 1989). However, species with inferior resistance, such as Douglas-fir and grand fir have higher mortality rates among the faster-growing trees.

Precommercial thinning in mixed-species stands is discussed in more detail under Mode III—Disease of young trees.

### *Brush removal improves conifer growth and may increase Armillaria root disease*

Removal of hardwood shrubs and trees (by cutting) to release young conifers is a common practice in the intermountain west, especially on private timber land. Early seral hardwood shrubs and trees often grow rapidly in the first decades after disturbance. In many cases, hardwood trees and shrubs endure long-term where root disease prevents conifer canopy closure or produces openings by killing trees.

Several recent studies in the interior cedar-Hemlock and Interior Douglas-fir forest types of southern interior British Columbia have revealed significant increases in *Armillaria ostoyae* activity following cutting of competing hardwood trees. Paper birch, in particular were removed by various means to release young Douglas-fir (Baleshta and others 2005, Simard and others 2005) and lodgepole pine (Simard and

others 2005). Although the birch thinning significantly improved residual tree growth, Douglas-fir and lodgepole pine mortality due to *Armillaria* root disease also increased with increasing hardwood thinning intensity. In general, a 1.5 to several times increase in Douglas-fir mortality rates were measured following hardwood thinning compared to unthinned stands. The increased mortality of lodgepole pine was temporary, lasting only 3 years after thinning (Simard and others 2005).

Increased growth of crop tree roots probably accounts for at least some of the mortality increase. Larger root systems contact inoculum more readily. Also, although birch is considered tolerant to *Armillaria*, severed root systems may have been utilized by the fungus to increase inoculum potential.

#### **Brushing can increase mortality rates**

There is mounting evidence that increased growth following brush cutting may lead to several fold increases in crop tree mortality rates.

## MODE III — DISEASE OF YOUNG TREES

*Armillaria* root disease commonly develops in young pines, and western larch regenerated on harvested sites. Young spruces, cedars and hemlocks are sometimes killed as well. Primary spread of the pathogen occurs from large stumps of the previous generation. Young trees are infected and killed as their roots contact infected stumps.

Two or three decades later, as the inoculum dies out or recedes into the interior of the old stumps, the mortality rate of young trees declines. Secondary spread is minimal, probably due to increasing resistance of the young pines, larch, spruces, hemlock and redcedar to *Armillaria*. Therefore, these species should be favored over inland Douglas-fir and grand fir in silvicultural treatments.

In young stands with extensive root disease mortality, thinning should be delayed, but lightly affected stands can be thinned. Mortality due to mode III *Armillaria* root disease declines after about 15 to 20 years and is minimal after 25-30 years of age. There is usually little lasting effect from this early mortality. The small canopy openings are usually insignificant in a mature stand.

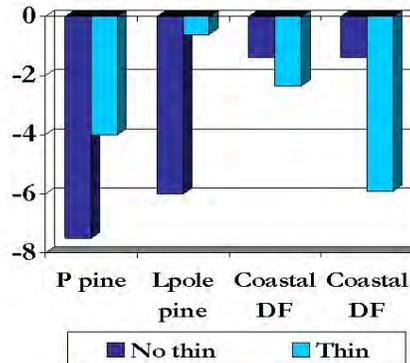
In contrast to inland Douglas-fir which nearly always exhibits mode I or mode II disease, coastal Douglas-fir generally follows the mode III pattern. Little damage is seen after the first two or three decades after stand establishment.

### *Precommercial thinning in Mode III Armillaria root disease*

Among tree species exhibiting mode III disease, response to precommercial thinning varies significantly. Therefore, species composition is an important factor in realizing benefits from precommercial thinning in *Armillaria ostoyae*-affected

stands.

**Figure 15. Mortality after precommercial thinning**  
Percent change; stems/10yrs



Thinning is generally intended to improve growth of crop trees by reducing inter-tree competition and, in some cases, to alter species composition. Douglas-fir stands in Western Cascades realized no significant differences in growth or mortality Filip and Goheen (1995) following precommercial thinning. In contrast, precommercial thinning may have been an effective method of increasing both growth and survival of ponderosa pine stands with *Armillaria* root disease (Filip and others 1989).

In another study in the Cascades (Rosso and Hansen 1998) *Armillaria* root disease in Douglas-fir was described as more consistent with Mode II behavior in that mortality continues well beyond the first two to three decades. Here, a positive increase in growth was measured following precommercial thinning but incidence of *Armillaria* root disease was also higher in thinned stands compared to unthinned controls. The most significant finding in this study was that tree vigor was not a factor in root disease. Vigorous and non-vigorous Douglas-fir were equally likely to be killed by *Armillaria* root disease.

## Managing Mode III

### Tolerate losses

Damage from primary inoculum is short-term and usually not significant in at the final harvest.

### Reduce inoculum

Stump removal can significantly reduce primary inoculum on sites and prevent most damage from this inoculum source. The economics of stump removal depends on anticipated damage from primary inoculum and potential damage to the site.

### Match planting stock to the site

Resistance does not hold up if the planting stock is not appropriate for the site. Check seed zones and potential vegetation or habitat types.

### Precommercially thin

Thin favoring resistant species, and to maintain growth of resistant trees.

**Disease Types I or II in Douglas-fir and true firs may occur on the same site with type III in other tree species if a variety of tree species are present.**

## Managing Mode IV

### Maintain tree vigor

Initial infection is probably not preventable because the fungus can establish small lesions and maintain quiescent infections for prolonged periods on healthy trees. Therefore the goal of preventing the development of severe root disease is accomplished by scheduling harvest to avoid tree senescence; proper timing of thinning to help trees resist drought affects; and prevention of bark beetle and defoliator outbreaks.

**“Douglas-fir is a short-lived species on the better white pine sites; root-rotting fungi often cause heavy mortality beginning in the sixth or seventh decade of life.” Watt, 1960**

Precommercial thinning in dense lodgepole pine stands in Alberta, British Columbia, did not change mortality rates during 11 years after thinning compared to unthinned controls, but did improve growth (Blenis 2000). Post-thinning mortality rates were

generally less than 0.5%/year but ranged as high as 1.5%/yr on a few plots. From this it is reasonable to conclude that precommercial thinning is probably of benefit to species that normally develop Mode III *Armillaria* root disease.

## MODE IV — WEAK OR SECONDARY ROOT AND BUTT DECAY

*Armillaria* root disease is typically manifest in aging or stressed trees. Root lesions probably establish from primary spread early in the process of stand establishment following harvest or fire. Lesions progress little, perhaps even become quiescent for some time. Lesions may begin to expand as trees become less efficient through aging or as a result of stressful

conditions such as drought, fire or logging injuries, fir engraver strip attacks, budworm defoliation, etc. Some infections spread enough to contribute to tree mortality. Killed roots may provide sufficient inoculum to support secondary spread of the fungus to adjacent live trees. Small groups of mortality may result. The overall stand impact is minor.

## *Armillaria root disease and forest succession*

The status of Douglas-fir, at least in the white pine type, has undoubtedly changed greatly in the past 80-100 years. Early in the 20th century, Douglas-fir was not generally a major stand component; certainly not like it is now. For example, based on 400 permanent plots established in the white pine type in northern Idaho and western Montana between 1909 and 1927, Davis (1942) described the average mature stand as having only 15% larch and Douglas-fir (combined). He reported 22% grand fir, 49% white pine and 14% western redcedar in the stands.

*Armillaria* root disease was recognized as an important factor in

limiting Douglas-fir in these forests as far back as at least the 1940's. Analysis of results from the above plots led Haig and others to conclude,

*“Douglas-fir fails to keep up sufficiently rapid height growth to maintain its position in the dominant canopy and is not sufficiently tolerant to thrive in an intermediate position. Its susceptibility to attack by fungi, particularly the root rot fungus (*Armillaria mellea*), removes individuals from the stand at a comparatively early age. – according to consistent records from permanent sample plots on the better white pine sites this weeding-out process may begin as early as 40 years.” [Haig and others 1941]*

Watt, 1960 also observed the temporary nature of Douglas-fir importance in stands but noted the relatively longer tenure of grand fir. Based also on permanent plot results, Watt stated,

*"At 75 years (oldest age class), grand fir was still hanging in and increasing its proportion of the stands."*

Larch in these stands grew well in young age classes on the better sites but, in the absence of thinning, declined in the sixth or seventh decades because of lateral competition.

A recent study in forests of northern Idaho and Montana further examined effects of root disease on forest succession (Hagle and others 2000). Composition and structure changes were analyzed for 25,670 acres of western redcedar habitat type over a 40-year period, from 1935 to 1975. This was a good time to look at forest change in the Northern Rockies. Early seral tree species (pines and western larch) were still abundant in 1935 although white pine blister rust (*Cronartium ribicola*) had recently invaded many white pine forests, and fire control was just beginning to be effective.

Stands that were well-stocked, pole size (6-14 inch average diameter at breast height) Douglas-fir on western redcedar site types (habitat types) averaged 42 years of age in 1935. They were actually mostly mixed species stands with Douglas-fir making up the majority of the stand volume. Regional yield tables would project these to be larger diameter, well-stocked Douglas-fir stands 40 years later,

when they would average about age 82. However, only 9% of these stands met this expectation.

Most of the sample stands were dominated by cedar in 1975, most of the Douglas-fir had already died out. Many other stands had become low density grand fir or Douglas-fir stands with severe root disease. They had less than 20,000 board foot volume per acre; very low stocking for 80 year old stands on these productive sites.

In most of these stands, *Armillaria* root disease is still active in what Douglas-fir remains on the site, and has become increasingly active in the grand fir component. The Douglas-fir wasn't harvested; there was no evidence of tree cutting in the sample. This represents a natural course of succession on cedar habitat types where root diseases are an important feature of most sites.

Root disease was the primary driver of succession in 83% of these acres. Douglas-fir beetle was also active on 14% of acres. Between the two, they directed succession on 86% of acres. Only those stands with no detectable root disease activity followed the expected successional pathway by retaining Douglas-fir forest type while growing to large tree and well-stocked stand structures.

The pervasive influence of root disease in northern Rocky Mountain forests is easily overlooked because change happens slowly. It is also less obvious, in many cases, because the mortality and brushy openings are so consistently present as to appear unremarkable to the untrained eye.

### Succession mediated by root disease on a western redcedar site

**Time period: 43 years**

A mixed stand of 45-yr old Douglas-fir and grand fir had understory of grand fir and western redcedar in 1962. The owner of this forest projected culmination of the stands and expected harvest of mature Douglas-fir at age 90 (in 2007). Today, the Douglas-fir can be seen on the forest floor under a 40-60 year-old stand of western redcedar and grand fir.

[Photos by Susan Hagle]



## *Identity Crisis Averted*



Originally recognized for its common and edible mushroom, *Armillaria* species have been known since the early 1700's (Watling et al 1991). *Armillaria mellea* was, until relatively recently, the name assigned several of these pathogens of trees throughout much of the northern hemisphere.

Mating reaction studies in Finland starting in the early 1970's contributed to unraveling the *Armillaria* species confusion

which has led to better understanding of *Armillaria* species ecologies (Wargo and Shaw 1985). By the late 1970's, the species *Armillaria mellea* was recognized as an amalgam of at least 10 distinct "biological species". Among these, *Armillaria ostoyae* emerged as the primary conifer pathogen in western North American forests (Morrison and others 1985, McDonald and others 1987).

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### Forest Health Protection and State Forestry Organizations

Assistance on State  
And Private Lands

Montana: (406) 542-4300

Idaho: (208) 769-1525

Utah: (801) 538-5530

Nevada: (775) 684-2500

Wyoming: (307) 777-  
5659

Assistance on  
Federal Lands

US Forest Service Region  
One

Missoula: (406) 329-3605  
Coeur d'Alene: (208) 765-  
7342

US Forest Service Region  
Four

Ogden: (801) 476-9720  
Boise (208) 373-4227

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