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Susan K. Hagle
USDA Forest Service

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Key Points

- Manage for pines, larch, and cedar.
- Precommercial thinning may improve growth and survival of pines and larch
- Thinning for bark beetle control may result in increased mortality if laminated root rot is present.

Management Guide for Laminated Root Rot

Phellinus sulphurascens (Pilat)
[formerly *Phellinus weirii* (Murr.) Gilb. Douglas-fir form]

Hosts: Primarily Douglas-fir and grand fir

All conifers may be infected

Two distinct biological species of this fungus occur in western North America; one primarily on Douglas-fir and true firs (discussed here), and the other, on western redcedar (see Chapter 13.5; Cedar Laminated Butt Rot).

A serious disease in northern Idaho and western Montana.

This fungus kills trees in expanding patches in forests. These patches can be from a few trees to many acres in size. Trees of all ages are killed as their roots are destroyed by the fungus. Many also die through wind-throw following decay of support roots. The fungus spreads underground through tree root systems and survives for many decades in dead roots and stumps.

Laminated root rot should be considered a “disease of the site”. That is, established mycelia of this fungus are essentially permanent, so the best course is to minimize losses by managing tree species that can be

expected to have better survival on infested sites.

Laminated root rot is estimated to infest at least 739,000 acres in Idaho and Montana. Mortality centers occupy over 15,000 acres of federal, state and private forestland on and near the Idaho Panhandle, Clearwater, Lolo, Kootenai and northwestern Bitterroot National Forests in Idaho and Montana. It is rarely seen south of the Salmon River in Idaho. Douglas-fir and grand fir forests on warm, moist site types are most damaged.

OVERVIEW OF LAMINATED ROOT ROT MANAGEMENT

1. **Favor resistant species.** Especially pines and western larch.
2. **Thin early and avoid partial harvests.** Precommercially thin favoring root disease resistant species.
3. **Avoid commercial thinning that will leave susceptible crop trees.** Mortality rates of susceptible leave-trees are likely to remain high, or increase following thinning.

*Nomenclature of *Phellinus sulphurascens**

The name *Phellinus sulphurascens* was officially assigned to the Douglas-fir/ true fir form of *Phellinus weirii* by Larsen and others in 1994. But at least as far back as 1954 the two distinct forms of *Phellinus weirii*, one found primarily on Douglas-fir and the other in western redcedar, were recognized (Buckland 1954). The name change will reduce the confusion that has resulted from using the same name for two dissimilar fungus species, with different hosts, and vastly different disease management methods.

Larsen and Lombard initially proposed separation of the cedar form from the Douglas-fir form into two distinct species in 1989. They found significant differences in cultural characteristics, length of setal hyphae, basidiospore germinating characteristics and host specificity. Cedar and Douglas-fir forms are distinctly intersterile groups in culture (Angwin and Hansen 1988). Despite finding nearly complete genetic isolation of Douglas-fir and cedar forms Angwin (1989) recommended considering them to be intersterility groups rather than distinct species. Intersterility observed in cultures was confirmed by Bae and others (1994) in naturally-occurring populations.

Banik and others (1993) found Douglas-fir isolates to be distinct from the cedar form in ELISA (seriological) tests. They also demonstrated that the Douglas-fir form is serologically related to Siberian isolates of *P. sulphurascens*. It is more closely related to the Asian *P. sulphurascens* than to cedar form isolates of *P. weirii*. They concluded that the Douglas-fir form is probably conspecific with *P. sulphurascens*. Based on results from monocaryon matings, Larsen and others (1994) also concluded that the correct name for the Douglas-fir form is *Phellinus sulphurascens* Pilat. They also described the hyphal system of both Douglas-fir and cedar forms as being monomitic. A monomitic hyphal system is the principle criterion that separates the genus *Inonotus* from *Phellinus*. Therefore the correct generic placement of both *P. weirii* and *P. sulphurascens* is *Inonotus*.

Beginning in 2007 Sturrock and associates adopted the use of *Phellinus sulphurascens* to refer to the Douglas-fir form (Sturrock and others 2007, Lim and others 2008). This is the binomial I will use in the present document.

Ecology of laminated root rot in forest ecosystems

Life Cycle

Phellinus sulphurascens begins life, like most fungi, as a spore. The mostly annual, crust-like sporophores typically form on the moist underside of downed logs. The sporophore matures in late

summer or fall. The simple, single-celled spores are produced en masse within pores of the sporophore. They are wind or water-dispersed; rarely landing on a substrate that is suitable for their survival.

Laminated root rot in Western North America by **Thies and Sturrock, 1995**

This useful publication provides excellent photographs and summarizes the relevant information for the identification and management of this disease.

Phellinus sulphurascens

- **Pathogen of conifers in the northern hemisphere.**
- **Annual sporophore**
- **Douglas-fir and grand fir are the most important hosts.**
- **Does not mate with *Phellinus weirii*.**
- **Monomitic hyphal system suggests genus name *Inonotus*.**

Moisture is required for germination. The mycelium that is subsequently produced must establish on suitable wood and repel competitors or perish. Most perish.

Persistent and expanding mycelial clones

New mycelia from germinating spores probably join with, and exchange nuclei (conjugate) with other mycelia to become heterokaryotic. These mycelia may have ecological advantages over those grown from a single spore Angwin and Hansen (1993). Even so, life is precarious for the new heterokaryote and few will live to establish as a clone.

There is little genetic variation within established clones, indicating that they start from single basidiospores and do not mate with or combine genetic material with new spores or colonies (Bae and others 1994). Vegetative incompatibility among clones effectively prevents initiation of new infections in areas occupied by established clones (Bae and others 1994). This means that established clones effectively block invasion by new clones of *P. sulphurascens*. It appears that once a clone is established on a site, it may survive for centuries by utilizing both living hosts and dead substrates. Generation after generation of trees serve as hosts for the same clone on a site.

Because clones within infection centers are genetically distinct from one another, the approximate extent and age of clones associated with some infection centers have been determined. For example, a clone in a mountain hemlock forest in

Oregon was estimated by Dickman and Cook (1989) to be at least 460 years old.

Saprophytic and parasitic existence

Phellinus sulphurascens has been reported to live saprophytically in the roots and lower stem of fallen trees or in individual cut stumps for as long as 50 years (Hansen 1976, 1979a). Hyphae penetrate the wood, causing a ‘white rot’ type of decay in which both cellulose and lignin are degraded. Dead wood utilized by *P. sulphurascens* also may be from tree and woody-shrub species that were resistant when they are alive.

The mycelium spreads from woody substrates to the roots of live trees through direct root contact with infected wood. Infection intensity may increase with each generation (Tkacz and Hansen 1982). In northern Rocky Mountain forests, Douglas-fir and grand fir are particularly susceptible to infection. 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.

Aggregated and dispersed infections in stands

Laminated root rot occurs in complex patterns in forest stands, with many or most of the infected trees bearing no above-ground symptoms of infection. Distinct infection centers are easily detected as aggregations of dead and diseased trees. Much of the infection in a stand will be within and adjacent to these infection centers.

Laminated root rot is commonly found in the same trees and even the same roots with other root diseases such as *Armillaria*, *Annosus*, and *Tomentosus root diseases*.

Mycelium—

Plural: Mycelia
The vegetative (non-fruiting) part of a fungus consisting of a mass of branching, thread-like hyphae.

Monomitic—

a fungus that has only one kind of hyphae, generative hyphae.

Heterokaryotic—

cells having two distinct nuclei. Generally produced by conjugation of two different mycelia.

Homokaryotic—

cells having one nucleus. Homokaryotic mycelia generally result from germination of sexually produced spores, such as those from sporophores.

Infected trees also are found dispersed throughout stands in locations having no apparent relationship to infection centers (Thies and Nelson 1997, Thies and Sturrock 1995). Thies and Nelson (1997) reported that infection rates at considerable distance from infection centers were as high as they were at the edges of infection centers, near confirmed inoculum sources.

EXPANSION RATES

Bloomberg (1990) monitored the rate of expansion of 62 laminated root rot centers, for 27 years, in three coastal Douglas-fir plantations in British Columbia, Canada.

The overall average rate of expansion was unimpressive: 1-8 cm/yr. **Roughly 0.5 to 4 inches per year.**

This was because most of the perimeter of an infection center had no change at all in most years.

However, where expansion did occur, the average rates were impressive: ranging from 54 to 63 cm/yr. **Roughly 27 to 31 inches per year.**

"In most stands, the pattern of [*P. sulphurascens*] infection was diffuse rather than distinctly aggregated, and in no case did openings accurately portray the distribution of the disease." Thies and Nelson 1997

Trees that were expected to be infected based on proximity to known inoculum sources (within 5 m) and having symptomatic crowns (poor growth, thin crowns, chlorosis) were, in fact, no more likely to be infected than trees that grew far from known inoculum sources and had healthy-appearing crowns (31% of the former compared to 29% of the latter). The dispersed infections may eventually become the focus of new infection centers (Thies and Nelson 1997).

Fire

Fire has historically been an important factor in shaping forest composition and structure in the northern Rocky Mountains. Based on the considerable age achieved by *Phellinus sulphurascens* clones, it is reasonable to conclude that they typically survive forest fires. They may be fragmented by partial destruction of the mycelium or by exhaustion of substrate in spots, but regeneration and growth of suitable hosts has the effect of allowing re-expansion of clones.

The most significant effect of fire may be the opportunity it provides for less-susceptible tree species to establish. Pines and larch are sometimes able to survive fires that kill Douglas-fir and grand fir, and to take advantage of fire-produced openings to regenerate. Historically, fires probably played an important role in reducing laminated root rot by favoring disease-resistant tree species.

Forest structure and composition effects of laminated root rot

As previously discussed, *P. sulphurascens* infections are both dispersed and aggregated in stands. However, most mortality occurs where infections are aggregated, also known as "infection centers". As overstory trees are killed in infection centers, openings in the forest canopy result. This produces a characteristically patchy forest structure. Canopy openings provide opportunities for conifer regeneration, and growth of woody shrubs and forbs. These openings increase structural diversity, and sometimes species diversity, in a forest. (Holah and others 1993, Holah and others 1997). After timber harvest, some vegetation differences between former gaps and previously intact forest persist (Ingersoll and others 1996).

Timber harvest

Following harvest, *Phellinus sulphurascens* readily infects the subsequent regeneration of susceptible trees. Although rapid and nearly complete mortality of Douglas-fir regeneration, due to laminated root rot, has been reported (Ingersoll and others 1996), this is not usually the case.

In fact, *Armillaria ostoyae* is often the more aggressive pathogen in the first two decades after timber harvest (Bloomberg 1990, Thies and Sturrock 1995). In northern Idaho and western Montana, stands regenerated after a harvest are usually 25-40 years old by the time laminated root rot begins causing more mortality than *Armillaria* root disease.

Temporal patterns of mortality and re-growth: wave pattern

Patches of laminated root rot exhibit slow expansion into surrounding forest; a rate of about one foot per year (Thies and Sturrock 1995). In addition to expansion at the perimeter of patches, trees within the disease patch regenerate, grow and die in a pattern described by Dickman and Cook (1989) as waves of infection.

The initial mortality wave occurs as the pathogen moves outward along the margins of the disease patch into the non-diseased portion of the stand. The resulting canopy opening is eventually filled with tree seedlings. When this new cohort of trees contacts inoculum from infected roots from the previous stand, they in turn are killed; the second wave of mortality.

Subsequent waves follow as new cohorts of trees regenerate, reach enough biomass to fuel a pulse of fungal growth, and die to once again open the canopy. 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 of the disease-affected area to the perimeter.

Mortality wave patterns on harvested sites

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 first 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.

Dispense with the vigor myth

Contrary to popular belief, superior vigor does not confer improved resistance of Douglas-fir to laminated root rot. Hansen and Goheen (2000) refer to this as “the widespread but false belief that pathogens in natural ecosystems only kill weakened trees”. In reality, faster growing trees have an equal or somewhat higher probability of dying from laminated root rot compared to slower growing trees (Miller and others 2006) This is probably just a matter of their larger root systems contacting more inoculum.

The wave phenomenon was described by Dickman and Cook (1989) as “outwardly-spreading rings within rings.”

WAVE PATTERN

As a result of the temporal pattern of tree death and subsequent regeneration, ranks of trees of similar ages are observed in concentric circles.

Recognizing this pattern in stands will aid in identifying the extent of an infection center.

THE VIGOR MYTH:

The widespread but false belief that pathogens in natural ecosystems only kill weakened trees (Hansen and Goheen 2000).

On sites where a mature stand with laminated root rot has been harvested, *Armillaria* root disease often becomes the primary pathogen in young conifers that regenerate following harvest.

It may take several decades for laminated root rot to again become the most prevalent disease.

INFECTION DETECTION

Ectotrophic mycelium



Thies and Nelson (1997) found that the presence of ectotrophic mycelium identified 82 percent of the infected trees in a mature stand.

Wallis and Bloomberg (1981) found that only half of the infected Douglas-fir trees 25 to 60 years of age have above-ground symptoms.

Impact of laminated root rot on forest productivity

Infection rates

Although nearly all conifers are occasionally infected, Douglas-fir, mountain hemlock, grand fir and white fir are most susceptible (Hansen and Goheen 2000). In the northern Rockies, Douglas-fir and grand fir are the most common hosts.

The disease is best known and probably most damaging in coastal Douglas-fir forests of Oregon, Washington and British Columbia. Surveys indicate that 8.6% of the Douglas-fir forest area in western Oregon is occupied by laminated root rot centers in which half of the Douglas-fir have been killed (Hansen and Goheen 2000). The disease is present in 80% of second-growth Douglas-fir stands on Vancouver Island, British Columbia (Bloomberg and Reynolds 1985).

A recent analysis in Idaho and Montana, estimated that 739,000 acres are infested with this disease. Of these, 156,000 acres have mortality rates of at least 25% of the basal area every 15 years. Large canopy gaps, with over 75 percent canopy loss caused by laminated root rot occupy 15,400 acres of the most productive forest land types in these two states.

Growth decline

Decay and girdling can be slow processes and considerable infection can develop in root systems before trees begin showing obvious crown symptoms. The first symptom to develop is usually growth decline. The cumulative growth depends on the age of trees. Bloomberg and Wallis (1979) detected reduced

radial increment in infected trees lasting 30 years or more before death in mature Douglas-fir. Smaller trees typically had shorter detectable periods of decline before death. The longer the period of decline before death, the greater is the cumulative growth loss. Diameter growth is more affected by laminated root rot than height growth in coastal Douglas-fir (Harrington and Thies 2007). As expected, trees with the highest rates of root decay exhibit the largest declines in growth.

Mortality

In coastal forests, grand fir is the most susceptible species, followed by Douglas-fir (Table 1). Nelson and Sturrock (1993) measured grand fir mortality rates averaging about 31% in 17-20 years for trees planted around infected stumps in coastal Oregon and British Columbia. Rates for coastal Douglas-fir averaged about 26%. Western hemlock and ponderosa pine had moderate mortality rates of 12% and 11%, respectively. Other pines and western redcedar mortality rates lowest, around 5%.

Similar rates of mortality were measured in inland forests on randomly-placed plots. In this case, Douglas-fir had the higher mortality rate compared to grand fir. A net loss in basal area of 35% for Douglas-fir and 20% for grand fir was measured. The plots, in northern Idaho, monitored trees that were at least 5 inches dbh, at the start of the study, over a 20-year period (Hagle, in preparation).

Perhaps the relative rates of mortality can be inferred from a survey reported by Filip and Schmitt (1979). They selected locations with at least 15% mortality and *Phellinus* as the primary cause. They found 20% of the Douglas-fir dead and 23% of

grand fir. This may suggest similar mortality rates for these two species; but the relatively longer needle retention of dead grand fir could give a false impression of a higher mortality rate.

Table 1. Relative susceptibility to *Phellinus sulphurascens* in the northern Rockies .

Species	Susceptibility	Reported mortality rates (infested sites)	On northern Rocky Mountain sites
Douglas fir	High	35% /20 yrs ID ¹ 20% E. OR/WA ² 26% /17-20 yrs Coastal ³	This is the most damaged species in the Rocky Mountain and west coast portions of the range of laminated root rot in North America. Both coastal and inland varieties of Douglas-fir are highly susceptible.
Grand fir	High	20% /20 yrs ID 23% E. OR/WA 31%/17-20 yrs Coastal	Grand fir is very susceptible to infection but dies somewhat more slowly than Douglas-fir.
Subalpine fir	High	43% /20 yrs ID	Subalpine fir can be very susceptible when growing in association with grand fir on the warm/ moist end of the range of subalpine fir. Cold and dry habitats appear to inhibit development of the disease.
Mountain Hemlock	High	32% E. OR/WA	We know little about root diseases in this species in the northern Rocky Mountains but it is considered highly susceptible on the eastern slopes of the Cascades.
Western larch	Moderate	6%/20 yrs ID 27% E. OR/WA	Damage is uncommon, usually occurring in older, unthrifty, trees growing with Douglas-fir or grand fir.
Western hemlock	Moderate	4% /20 yrs ID 12%/17-20 yrs Coastal	We rarely see significant damage in this species in inland stands.
Western redcedar	Low	>1%/20 yrs ID 5%/17-20 yrs Coastal	Western redcedar is reported as either immune or highly tolerant.
Ponderosa pine	Low	>1%/20 yrs ID 11%/17-20 yrs Coastal	Damage is rare in this species; has been observed in individuals growing with Douglas-fir and grand fir.
Lodgepole pine	Low	>1%/20 yrs ID 9% E. OR/WA 5%/17-20 yrs Coastal	Damage is rare, usually occurring in older, unthrifty, trees growing with Douglas-fir and grand fir.
Western white pine	Low	>1%/20 yrs ID 5%/17-20 yrs Coastal	Damage is rare but has been observed in older trees.
Engelmann spruce	Low	Insufficient data	Damage is rare; usually occurring in trees grown with Douglas-fir and grand fir.

1 Percent of trees killed in 20 years with *Phellinus* as an identified cause; on permanent plots that had known *Phellinus* inoculum within 30 feet (Unpublished data, Hagle 2008.) Plots are in northern Idaho.

2 Percent of trees that were dead at the time of the survey; stands were east of the Cascade range in Oregon and Washington (Filip and Schmitt 1979.) Plots were selected on the basis of having at least 15% mortality with *Phellinus* as the primary cause.

3 Percent of trees killed in 17-20 yrs in plots with known *Phellinus* infection; coastal forests of Oregon and British Columbia (Nelson & Sturrock 1993.)

Sites that are prone to laminated root rot in Idaho and Montana

Laminated root rot

Is found mostly on western hemlock or grand fir potential vegetation types.

Laminated root rot causes problems on a narrower range of habitat types than do *Armillaria* or *Annosus* root diseases. Cool, moist types typified by western hemlock potential vegetation type (PVT) and warm moist habitats with grand fir PVT make up the majority of the infested sites. Likewise, a high proportion of these PVTs may be infested by this pathogen within its range. Byler and others (1990) found sites on the Lolo NF in western

Western hemlock and grand fir PVTs had the highest constancy and highest severity of laminated root rot in 312 randomly placed permanent plots in the Coeur d'Alene Basin of northern Idaho (Figure 1). Of 73 plots in western hemlock PVT group, 62% had confirmed laminated root rot. Of 88 plots with grand fir PVTs 54% had the disease (Hagle, unpublished.) Overall, 48% of the 312 plots had laminated root rot. In contrast Douglas-fir PVT plots

Western Montana

Frequency of *Phellinus sulphurascens* by habitat types on the Lolo National Forest: [Byler and others 1990]

Western hemlock PVT— 87%

Grand fir PVT— 17%

Western redcedar PVT— 11%

Douglas-fir PVT— 4%

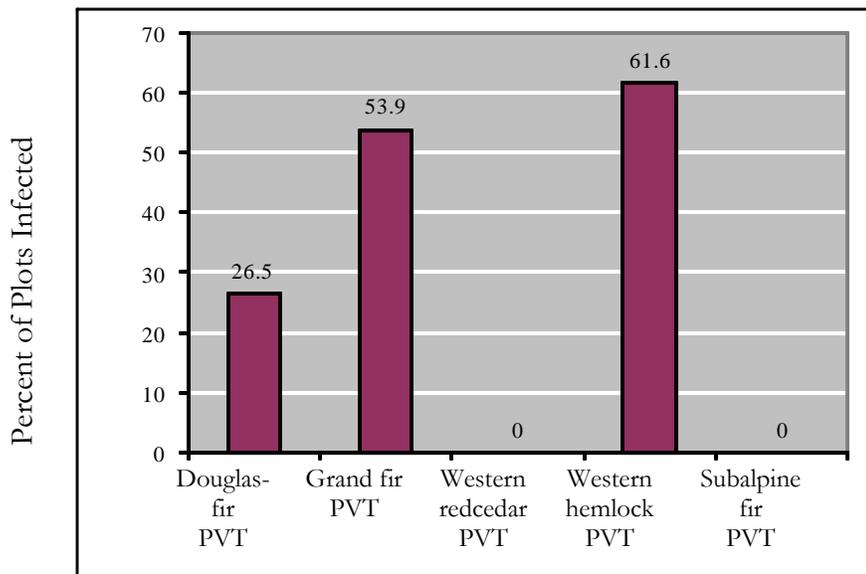


Figure 1. Percent of 312 plots with confirmed laminated root rot within five sampled habitat type groups. [Coeur d'Alene Basin, Hagle 2008 unpublished.]

Montana on western hemlock PVT to have far higher probability of laminated root rot than other sites. They found the pathogen on seven of eight stands (87%) sampled in this PVT group compared to the second highest, 17% of 106 samples, in grand fir PVTs. In contrast, they detected laminated root rot on only 4% of the sample in Douglas-fir habitat types and 11% in western redcedar habitat types.

had a lower than average probability of infection, only 26%. None of the 13 western redcedar PVT plots showed infection, but they all had redcedar forest composition, a resistant tree species. Only four plots with subalpine fir PVTs were sampled and none had evidence of *P. sulphurascens* infection. The pathogen has been observed on subalpine fir habitats, as well as on this host, but not often.

Assessing Laminated Root Rot

Disease intensity on infested sites

Root Disease Severity Rating

Root disease severity is a measure of the cumulative impact of root disease in a stand or on a site. It is visually rated based on relative effect of root disease on forest canopy density (*see sidebar*). At the most severe end of the scale, no trees of the original overstory remain alive. At the low end of the scale, little evidence of the disease will be seen and little or no canopy loss will have occurred. Laminated root rot is more likely to cause severe loss of forest cover than are the other major root diseases in northern Idaho and west-central Montana.

Loss of forest cover, or canopy is reflected in the site root disease severity (Hagle 1993). Disease severity can be assessed using aerial photography or on the ground. It is a rapid and efficient method to assess the relative impact of the disease on a site and to predict future impacts (Hagle and others 1992, Hagle 1993, Hagle 2000).

In 312 permanent plots in northern Idaho, monitored for 18 to 24 years, root disease severity assigned at the beginning of the monitoring period was the best predictor of tree mortality over the entire period. Severity was a highly significant indicator ($P > .0001$) for subsequent Douglas-fir mortality, in particular.

At the highest levels of disease severity, stands commonly have only a few surviving trees by 80 to 100 years of age. Large openings in the tree canopy permit growth of shrubs, forbs, and conifer regeneration. Douglas-fir and

grand fir regeneration in these openings seldom reaches maturity. However, if disease-resistant species, western hemlock and western redcedar in particular, establish in the openings, they are likely to survive and eventually return the forest canopy to full closure.

Figure 2 illustrates the relatively greater severity of root disease impact with laminated root rot compared to other root diseases on permanent plots in northern Idaho. The proportion of plots falling in the highest disease severity levels was nearly double compared to all plots. A similar trend was found in permanent plots on the Kootenai NF in Montana. *Phellinus sulphurascens* was seldom the only root pathogen detected on a plot, with *Armillaria ostoyae* or *Heterobasidion annosum* commonly also present.

ROOT DISEASE SEVERITY CLASSES

0—No evidence of root disease.

Low—Ranges from minor evidence up to 20 percent canopy reduction.

Moderate—Between 20 and 75 percent loss of canopy.

High—at least 75 percent loss of canopy.

[Guidelines for rating root disease severity: see Hagle 1993 or on the web Hagle 2009.]

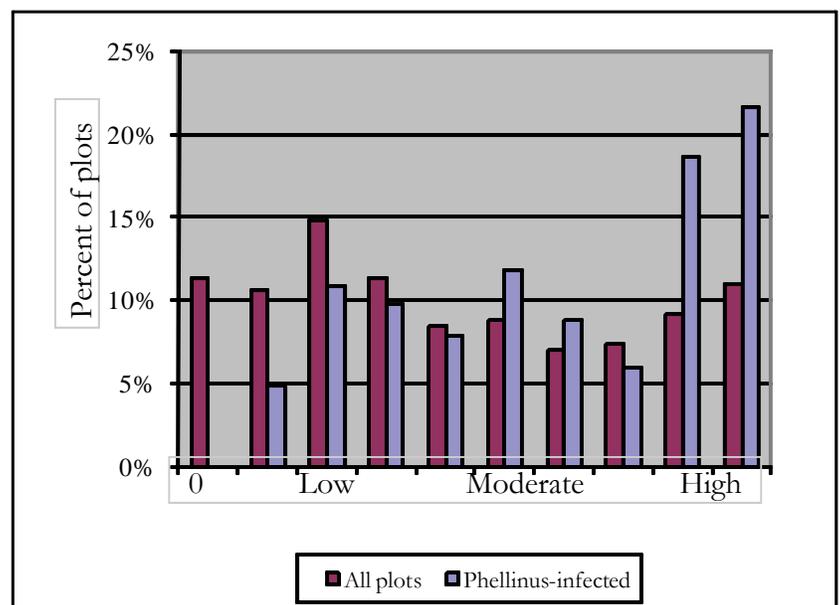


Figure 2. Distribution of root disease severities among 213 plots (all plots) compared to 102 plots with confirmed *Phellinus sulphurascens* (*Phellinus*-infected.) Coeur d'Alene Basin, Idaho Panhandle NF



The characteristic brown stain in the sapwood of recently cut stumps is used to identify many of the

Mechanisms of spread

Thies and Westlind (2005) recently concluded that there is "a need to examine more closely the epidemiology of laminated root rot."

...the infection process is more complex and needs to be better understood."

Root to root spread has long been the accepted model for laminated root rot and fits most field observations of the disease. However several recent discoveries seem inconsistent with this mechanism of spread.

Thies and Westlind cite:

1. scattered infected trees,
2. the appearance of laminated root rot in replacement stands where it had not been present before harvest, and,
3. nearly a third of plots that had laminated root rot before harvest, having no disease-killed trees in the replacement stand.

However, the presence of *P. sulphurascens* was significantly associated with more severe root disease.

Surveying stands for location and size of infection centers

Most of the laminated root disease in a replacement stand will be where it occurred in the previous stand. Before stand harvest, openings or gaps caused by root disease are easily observed. These infection centers can be mapped by walking parallel transects and marking the beginning and end of each infection center using GPS (or a sketch map). This method is especially appropriate in coastal forests where the disease is more

often concentrated in discrete infection centers. See Thies and Sturrock (1995) for more information.

Stump survey

After harvest, cut stump surfaces can be examined for symptoms of laminated root rot. A portion of the stumps with infected roots will have decay or stain visible at the stump surface. The locations and number of visibly infested stumps are a good indicator of the biomass of the root pathogen on the site (Thies and Westlind 2006). Such information can be used to direct planting of disease resistant trees or other efforts to reduce damage in the replacement stand.

Considerations for managing laminated root rot

Some important characteristics of laminated root rot to be considered for management of the disease include:

- 1) Laminated root rot is a 'disease of the site' because the pathogen, *Phellinus sulphurascens*, persists on a site from one generation of trees to the next.
- 2) The fungus is both an efficient parasite and saprophyte, meaning that both live and dead wood is utilized by the fungus.
- 3) The fungus biomass on a site is limited by the biomass of host tissue. So if there is an abundance of large host trees, the fungus can achieve very large biomass on a site sufficient for survival for many decades, perhaps hundreds of years after the host has died. However, if host biomass

declines, so will the fungus biomass.

- 4) The effective host range (species on which the fungus can build biomass) is limited.
- 5) The pathogen is probably naturally limited by competing fungi, especially saprophytic species that are capable of growing rapidly in dead wood.
- 6) The pathogen is rarely successful in establishing new infection centers so infestations are fairly stable.
- 7) Scattered infections, unassociated with disease centers, on roots of apparently healthy trees, are common. This probably accounts for the disease appearing in locations in replacement stands which were judged to be disease-free in the previous stand.
- 8) Laminated root rot is rarely the only root disease present in stands in Idaho and Montana.

Managing laminated root rot

Management objectives will guide the choice of methods to be used, but it is also important to consider the presence of root disease when developing the objectives for a stand or group of stands. Established root pathogens can be as limiting as other features that determine site capability such as latitude and altitude, soils, moisture regimes, and exposure. Realistic management objectives will take into account the laminated root rot considerations outlined in the previous section.

Methods for managing laminated root rot fall into two categories, silvicultural control of damage and direct inoculum removal. Silvicultural methods for managing root diseases are nearly always more successful and cost-effective than are chemical fumigation or physical removal of inoculum. However, for high-value trees there is some indication that chemical fumigation can rid live trees of infections. Inoculum removal by de-stumping infested sites also may have some applicability.

BOTTOM LINE

- **Root disease is permanent**
- **You may be able to reduce the mass of the fungus but you won't eliminate it.**

Usually, you will need to change tree composition to more resistant species.

Silvicultural control methods

Regeneration Systems

Armillaria and annosus root diseases are commonly found on sites that have laminated root rot. Fortunately, silvicultural control methods used to manage laminated root rot should be equally effective against these other major root pathogens of Douglas-fir and true firs.

Even-aged silvicultural systems, are aimed at producing a replacement stand that is better suited to resist or tolerate laminated root rot. Whether clearcut or seedtree methods are used, the target replacement stand should be comprised of less than one-third Douglas-fir and grand fir (combined). Pines (western white, ponderosa and lodgepole), western larch, Engelmann spruce and western redcedar are good choices for disease-resistance. Mixed-species stands are nearly always more resilient than single-species stands.

Shelterwood or uneven-aged systems should be avoided because they usually result in too much Douglas-fir, grand fir or subalpine fir regeneration. Disease intensification in partially-harvested stands appears to be due to the rapid colonization of the stumps and roots after infected trees are cut. Infested stumps then serve as effective food bases for the pathogens enabling them to infect and kill other nearby trees.

Western redcedar, normally considered a root disease-tolerant species, may develop a progressive decline after a partial harvest. If western redcedar is an important component of the residual stand, it would be prudent to avoid removing more than about one third of the overstory canopy to minimize risk of *Armillaria* root disease losses in the residual cedar. This does not apply to western redcedar that regenerates after the harvest.



Western larch (above) and ponderosa pine (below) seed-trees are used to regenerate root disease infested sites.





Photo above shows uneven-aged stands often provide ideal habitat for root pathogens as Douglas-fir and grand fir regenerate in partial shade.

Utilization of stumps by *P. sulphurascens* is about the same whether the stand is clearcut or partially harvested. What makes the difference in subsequent impact is the composition of the stand on the site following harvest. Therefore, when possible, resistant or disease-tolerant species should be favored when harvesting in root-disease afflicted stands. Douglas-fir and grand fir often die within a few years after thinning, having produced little or no additional growth. Douglas-fir and grand fir left as shelterwood or seed or crop trees, are likely to exhibit rates of mortality around 30 to 40 percent per decade following a partial harvest (figure 3).

Commercial thinning

Impacts of root diseases following commercial thinning, like other partial harvests, mostly depends on the composition of the residual stand. As discussed with respect to shelterwood and uneven-aged systems, higher rates of mortality in residual Douglas-fir and grand fir can be expected (figure 3). To minimize the effects of mortality and, perhaps, slow the spread of the disease, reduce Douglas-fir and grand fir to less than one third of the basal area. Even so, you can expect high rates of mortality in residual Douglas-fir and grand fir.

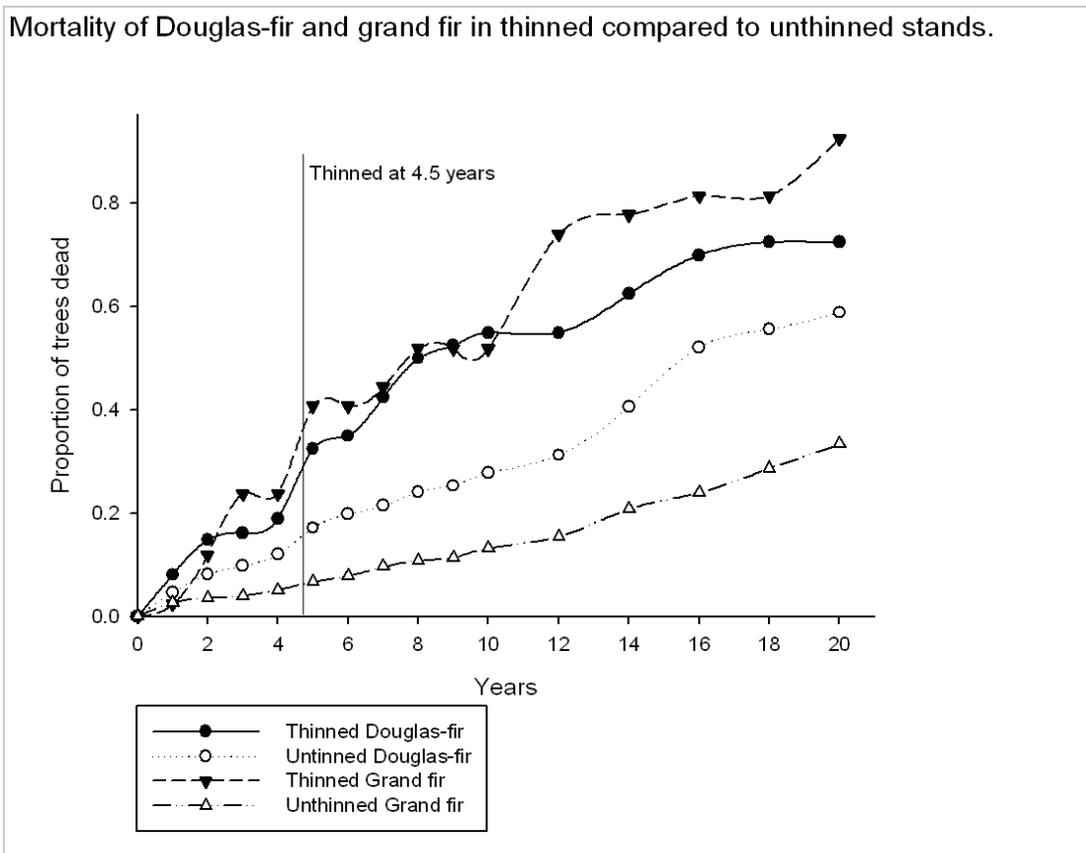


Figure 3. The rates of mortality in commercially thinned (filled symbols) compared to unthinned (hollow symbols) Douglas-fir and grand fir stands. The already-high rate of mortality of Douglas-fir was increased somewhat by thinning. Grand fir mortality was much higher in the thinned stands compared to those left unthinned. The stands have a combination of laminated, *Armillaria* and annosus root diseases .

Removals for bark beetle control

Douglas-fir beetle (*Dendroctonus pseudotsugae*), fir engraver beetle (*Scolytus ventralis*) and western balsam bark beetle (*Dryocoetes confusus*) are all associated to some extent with root disease-weakened trees. In most cases, they can utilize weakened trees to maintain endemic populations. Epidemic populations are not so closely tied to weakened trees and may attack many healthy trees.

Removal of attacked trees

Root pathogens almost certainly utilize the roots of bark beetle-killed trees in much the same way they use stumps of harvested trees. Efforts to control bark beetles by removing beetle-infested material from the woods may be of benefit in controlling the beetle populations and would be unlikely to increase root disease impacts. The roots of killed trees would be utilized by the fungus whether the stem was removed or not.

Bark beetle prevention thinning

Thinning to reduce stand density and thereby reducing the attractiveness of a stand to bark beetles is, in effect, a commercial thinning. If the species composition of the residual stand can be shifted by thinning to leave mostly disease resistant species, it will benefit both beetle and root disease management.

If most of the after-thinning stand will be Douglas-fir and true firs, laminated root rot and other root diseases can be expected to increase. The increase in mortality is fairly rapid, more than doubling rate of mortality in 10 years, to kill over half the Douglas-fir and

grand fir in a stand in the first decade after thinning. After a decade or so of poor growth, and high mortality rates, the residual stand volume often is so low as to render a regeneration harvest no longer economically feasible.

Precommercial thinning can be used to push the stand species composition toward disease resistant or tolerant species, especially western larch and pines. If possible, a reduction of the combined composition of Douglas-fir and grand fir to less than one third will minimize laminated root rot impact. By maturity such stands are likely to have even less Douglas-fir and grand fir and root pathogen biomass should be fairly low.

Laminated root rot can be problematic in western larch. Observation suggests that most of the problem occurs in poor growing trees. Periods of crowding or severe dwarf mistletoe infection may compromise the natural resistance of western larch to root infections. Precommercial thinning can enhance resistance of western larch by maintaining the tree's ability to produce phenols and necrophylactic periderm to limit the spread of *P. sulphurascens* infections. Larch dwarf mistletoe infection levels can also be reduced by selective removal of obviously infected trees during precommercial thinning. For other strategies to reduce dwarf mistletoe infection, see Hoffman (2004).

If the disease severity is moderate or higher, precommercial thinning leaving Douglas-fir crop trees may be uneconomical because of poor subsequent survival.

Tree Resistance

Necrophylactic periderms in roots produced in response to injury or infection. Trees may produce several successive necrophylactic periderms as each previous periderm is breached.

Most lesions are halted by defense chemicals (phenolic compounds) 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.



Photo shows fire is an important natural control for root disease by favoring the survival and regeneration of pines and western larch.



Saprophytic fungi such as *Antrodia heteromorpha* (shown on a Douglas-fir stump above and below) quickly establish on stumps and dead trees and effectively exclude root pathogens from the resource. They probably play an important role in limiting the buildup of root diseases on harvested or burned sites.



Mortality rates of Douglas-fir remain about the same whether stands are precommercially thinned or not and growth response of this species is both minimal and temporary.

Management by fire

Fire can be very effective in creating large mosaics typical of the natural fire patterns on habitats where laminated root rot is prevalent. The outcome with respect to laminated root rot will depend on the species that are regenerated. Burning does not harm *Phellinus sulphurascens* nor the other major root pathogens because they reside well below ground.

The roots of trees killed or damaged by fire will be utilized by root pathogens to increase biomass and spread to the residual trees and regeneration. So, like a regeneration harvest, expect higher mortality rates among residuals and

adjacent to the burn-treated area. Be prepared for a short flush of bark beetle mortality in fire-damaged trees.

Fertilization

Improvements in Douglas-fir growth may (Miller and others 2004) or may not (Nelson and others 1994) be realized through nitrogen and potassium fertilization. However, laminated root rot mortality is unaffected by fertilization (Thies and Westlund 2005, Miller and others 2006). Fertilization of Douglas-fir with nitrogen alone may increase the probability of *Armillaria* root disease-cause death (Moore and others 2004). Faster growing trees are likely to contact more inoculum which may increase mortality rates relative to slower growing trees (Miller and others 2006)

(Continued on page 15)

Managing laminated root rot: Biological control

Antagonistic fungi have been used successfully as biological pesticides in forest nurseries and in other agricultural settings. A common mold-type fungus, *Trichoderma viride* Pers., is an aggressive fungal antagonist in culture (Nelson 1964). When inoculated in holes drilled into *Phellinus*-infested stumps, *T. viride* was successful in killing *Phellinus* near the inoculated sites (Nelson and Thies 1985, 1986). Unfortunately, *Trichoderma* had limited ability to penetrate the woody substrate, which limited its ability to interact directly with the root pathogen.

Many other naturally-occurring saprophytic fungi invade conifer roots shortly after tree

death or harvest. These organisms have the potential to limit the development of root disease fungi. Among the most common fungi seen fruiting on stumps and snags in the northern rockies are *Fomitopsis pinicola* (Swartz:Fr.) Karst., *Trichaptum abietinum* (Dicks.:Fr.) Ryv., *Gloeophyllum sepiarium* (Fr.) Karst., and *Antrodia heteromorpha* (Fr.) Donk. In addition, several are routinely isolated from dead roots of trees, in particular *Perenniporia subacida* (Pk.) Donk, *F. pinicola*, *Resinicium bicolor* (Alb.& Schw. ex Fr.) Parm. and *T. abietinum*. These fungi probably naturally control laminated root rot and other root diseases to some extent by competing for woody substrates.

Managing laminated root rot: Direct Inoculum Removal

(Continued from page 14)

The amount of inoculum in the soil significantly influences the transmission and subsequent disease severity in the regenerated stand (Miller and others 2006). Methods and results of reducing the biomass of pathogen inoculum has been studied extensively in western coastal Douglas-fir forests where site values are high and alternative tree species have much lower timber value and productivity (Thies and Sturrock 1995).

Root removal

Stump and root removal can be accomplished either by digging out the stump and roots after the harvest or by push-falling trees and then raking the roots from the soil. An excavator is used for digging out stumps and roots and can also be used for push-falling trees. After push-falling, the trees are shaken to remove soil from the root wad. Roots still need to be raked from the stump site after push-falling. As many of the large roots as possible are removed from the soil. Once deposited above ground the roots become too dry to support the pathogen.

Either method is expensive and would likely only be used on high-value sites where Douglas-fir or grand fir were the preferred species. In the interest of operator safety, slopes should be less than 30 percent (Thies and Sturrock 1995).

Sanitizing the infested area

Destumping infection centers has reduced laminated root rot

mortality by over half in the first two decades after site regeneration (with Douglas-fir) in coastal forests (Morrison and others 1988, Thies and others 1994). Perhaps a third of the treated area had no laminated root rot. Infected stumps are identified by the presence of stain or decay at the stump surface. All infected stumps are removed along with as much of the root system as possible.

Isolating the infested area

Lateral expansion of root disease centers may be halted or slowed by removing root systems in a buffer surrounding an infection center. Although the infection center itself is still unsuitable for growing Douglas-fir and grand fir, it is somewhat contained in this way. Bear in mind that some infections will be present in non-symptomatic trees outside the obvious infection center and may become new expanding centers.

We have little experience with physical removal of inoculum in inland forests (see sidebar). These approaches probably have limited application in Idaho and Montana where at least two other aggressive root pathogens are also likely to be present both within and outside of obvious infection centers.

Chemical control

Most of the early research efforts were aimed at fumigating around or in stumps to kill inoculum. A recent study has also shown efficacy in killing infections in living trees.

ISOLATING THE INFESTED AREA



Stumps were removed to restrict spread of the root pathogen at the margin of the infection center (at left in the above photo). All stumps were removed and wind-rowed in a 200-foot buffer (see photo below). This control effort was implemented on industrial forest land in Montana to control *Armillaria* root disease. Although it was not entirely successful, it did restrict expansion of the disease along most of the treated edge.



In most cases, infections are dispersed throughout the forest area, limiting the use of this control method.

Non-target effects of chloropicrin stump treatment

- No significant release of chloropicrin from treated stumps into surrounding soil at one year following treatment (Ingham and Thies 1995) and at 5 years following treatment (Massicotte and others 1998).
- Little or no effect on vegetation three years following fumigation (Luoma and Thies 1997)
- No effect on mycorrhizae on Douglas-fir seedlings 4 to 5 years after stump treatment (Massicotte and others 1998).
- Rare and minor effects on soil fungi, bacteria and amoeba populations near treated stumps in the first year following treatment ((Ingham and Thies 1995).
- No effect on soil arthropods and nematodes the first year after treatment (Ingham and Thies 1995).

Stump treatments to reduce inoculum

Emphasis for chemical control has been eradication or reduction of inoculum in dead tissues of stumps. We know that *P. sulphurascens* infections are often abundant in apparently healthy portions of a forest, not associated with recognizable disease centers (Thies and Nelson 1997). However, nearly all mortality occurs within active disease centers. Therefore it is tempting to try eliminating the fungus from disease centers thereby returning the site to Douglas-fir productivity. De-stumping is often not practical because of steep terrain, large stumps, soil disturbance and the difficulty of removing the decayed roots that break away from stumps.

Chemical fumigation of stumps to kill the pathogen has been somewhat more successful in eliminating most of the inoculum (Thies and Nelson 1987, Fraser and others 1995). Holes are drilled into the tops of stumps, the fumigant is poured in, and the holes are plugged. The authors reported reduction of infected root tissue to just 22% (by weight) of what they estimated to have been infected before treatment. Despite

this success in reducing inoculum, just 16 years after replanting with Douglas-fir, there was no reduction in the amount of laminated root rot near treated stumps compared to non-treated stumps.

Perhaps the most important discovery here is the realization that very little inoculum is needed to sustain a disease center from one generation of host trees to the next.

Curatives for live trees

More recently, Harrington and Thies (2007) have reported success of MITC, chloropicrin, and Vorlex as curatives for infected roots in live trees. Nearly all fumigant treatments reduced root infection and, in some cases eliminated infection. Live Douglas-fir tolerated most of the tested fumigant treatments, although there was some cambium damage. The authors suggested that altering the time of treatment to the dormant season may reduce tissue damage. These results are encouraging because they suggest a potential to treat high-value trees growing in residential or administrative locations. Further research is needed to refine the application procedures.

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