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Laminated Root Rot of Western Conifers

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Laminated root rot is caused by the native fungus *Phellinus weirii* (Murr.) Gilb. It occurs throughout the Northwestern United States and in southern British Columbia, Canada. The disease has also been reported in Japan and Manchuria. In the United States, the pathogen is most destructive in pure Douglas-fir stands west of the crest of the Cascade Range in Washington and Oregon; in Douglas-fir/white fir stands in southern Oregon; and where Douglas-fir and grand fir are important components in mixed conifer types extending eastward from the Cascades into the western Rocky Mountains to the Continental Divide. Susceptibility of native conifers varies considerably among species. Annual losses are estimated at 4.4 million m^3 (157 million ft^3) of timber in the Northwestern United States and in British Columbia. Besides causing timber losses, infected trees pose serious threats to safety in high-use areas.

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Trees of all sizes and ages are attacked. But because early mortality is scattered and trees are small, the problem is often not conspicuous before stand age 40. Persistence from one rotation to the next and gradual spread of the fungus can maintain the disease on the site indefinitely. This role of *P. weirii* as a perennial inhabitant of the site, substantially reducing productivity, surpasses its importance as a killer of individual trees.

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Occurrence of Laminated Root Rot

The disease occurs in patches (infection centers) sporadically distributed in clusters throughout its range. The most susceptible hosts are Pacific silver fir, white fir, grand fir, Douglas-fir, and mountain hemlock. Other host species (western larch, spruce, some other true firs, and western hemlock) are also attacked and killed, especially when growing with the more susceptible infected species. Native pines and cedars, though relatively susceptible to butt rot, are resistant to killing. Hardwoods are not infected.

Diagnosing Laminated Root Rot

Symptoms of the disease (fig. 1) are not unlike those caused by other root pathogens which, especially in mixed stands in northern Idaho, frequently occur in the same infection center. Initially tree crowns become thin and yellow. As the disease progresses, terminal growth on branches progressively shortens. In the final stages of the disease, numerous small cones (distress cones) are often produced on the dying tree. Root systems of attacked trees, especially west of the Cascade Range, are so weakened structurally that the tree frequently topples before crown symptoms appear. Decay of wood (fig. 2) begins with a reddish-brown to brown stain, depending somewhat on host species. In butts and main roots, the stain appears as streaks or broad bands on longitudinal sections and as circular, crescent-shaped, or irregular areas on cross sections. Small oval pockets (about 1 mm

A

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B

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Figure 1.—Crown decline as seen on this young Douglas-fir (A) is a common indication of root rot. Larger trees can be toppled (B) before crown symptoms appear. Note stubbed roots on this "rot thrown" Douglas-fir.

A

B

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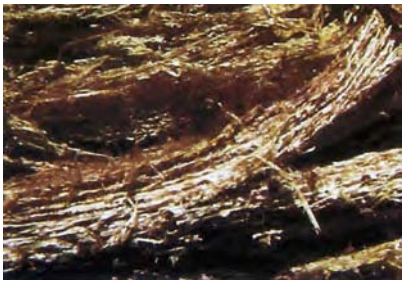
Figure 2.—Brown-stained wood (A) precedes advance decay (B).

long) begin to appear in stained wood, becoming larger and more numerous with time. As decay progresses, the wood tends to separate (laminate) along annual rings (fig. 3), thus, the common name, laminated root rot. As decay reaches this stage, trees are often windthrown, typically leaving all but the stubs of roots in the soil. Finally, the wood becomes a stringy mass and the lower bole becomes hollow.

A



B

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Figure 3.—Red-brown setal hyphae (A) are good diagnostic characteristics. Delamination in later decay stages (B) gives this disease its common name.

Decay can continue for several feet up into the bole while the tree is standing or after it is windthrown. Thin, velvety layers or sparse tufts of brown fungal threads (hyphae) may appear in crevices in the wood. These "setal hyphae" (fig. 3), along with other decay characteristics, are diagnostic of *P. weirii*. Sporophores (fig. 4) (fruiting bodies) are inconspicuous, brown, pore-covered crusts most often forming on the lower side of down trees, near or in contact with the forest floor. In some years and in many areas sporophores are rare. Because they occur infrequently and are inconspicuous, they are of limited value in identification of the disease in the field.

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Figure 4.—Sporophores most often occur on the undersurface of down trees, conforming to the shape of the supporting surface.

Aerial Detection

Infection centers (fig. 5) frequently can be recognized from the air or on good-quality, large-scale aerial photographs by characteristics of openings in the forest canopy. Trees on the edges of these openings are in a declining state or have recently been killed. Toward the interior of the openings, scattered standing dead trees (some without tops or near collapse) and windthrown trees occur. Numbers of surviving trees within the centers depend largely on susceptibility of the species. Centers become filled with brush, or with both brush and susceptible conifer seedlings which themselves become liable to infection. Though the ap-

A

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B

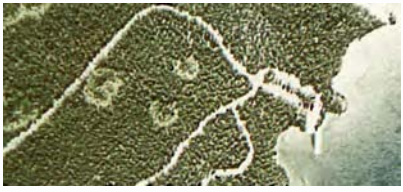

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Figure 5.—The disease as it occurs in mixed conifer forests high in the Oregon Cascade Range (A) is easily spotted on small-scale aerial photos (B). Aerial detection is more difficult in the moist, heavily vegetated coastal forests.

pearance of mortality over time tends to separate root disease from other causes, ground checking is necessary to determine which root pathogens are acting in the centers.

How Laminated Root Rot Acts

Though some evidence exists for spread of the disease by spores, most infection centers in young stands begin when roots grow out and contact the fungus surviving in decaying wood from infected trees of past generations. The fungus readily penetrates bark of roots of recently cut (within 12 months) or living susceptible trees and colonizes the underlying wood. Growth of the fungus continues over the root surface as well, and infection is spread when roots of other trees contact these infected roots. Rate of spread through a stand in this manner is estimated at about 34 cm (1 ft) per year.

As a rule of thumb, losses can be expected to double about every 15 years. Where infection centers are common and well distributed in young stands, serious losses must be expected before stands reach commercial maturity. New stands of susceptible species established on infested sites can be expected to suffer continuing and increasing mortality. Because the fungus can survive in roots of killed trees and in stumps and because it spreads from one tree to another where their roots contact, the more prompt and better stocked the regeneration, the more severe this continuing mortality is likely to be.

Aside from history of infection, damage does not seem to be associated with any specific stand history. Probability of disease occurrence in northern Idaho, however, has been linked to habitat type. A high probability of *P. weirii* root disease centers occurs in the highly productive *Abies grandis/Pachistima myrsinites* habitat type[4], which is characterized by a warm forest environment between wet and dry extremes. There is no obvious relationship of the disease to habitat types in the Douglas-fir forests west of the Cascade Range of Washington and Oregon.

[4] Ecological classification of sites by vegetation that would occur in the absence of major disturbance. In this case, the forest cover would primarily be grand fir.

Management Guidelines for Diseased Stands

Direct, effective control measures are largely untested or unknown. Management of disease areas involves changes in normal silvicultural procedures both in the Douglas-fir region west of the Cascade Range and mixed conifer forests to the east. This makes detection of the disease and permanent documentation of its existence in a stand critical in long-range planning and management.

In Young Stands

If disease centers are numerous and evenly distributed over the stand, do not plan stand improvement work. These stands can probably be grown to pole size before severe damage occurs. Where centers are not numerous, thinning can be done, but more resistant conifers or hardwoods should be favored in the vicinity of infection centers. In some cases, clearcutting well-defined infection centers and replanting to hardwoods or resistant conifers may be advisable.

In Poles or Small Sawtimber

If the disease is not severe, thinning can be done with only minor variation in procedure. Within and beyond about 15 m (50 ft) of the boundary of infection centers, only dead or visibly infected trees should be removed. Thinning infection centers to a greater extent will contribute to windthrow losses. Severely infested stands should be scheduled for early harvest.

In Large Sawtimber

Infested stands should be clearcut when net volume increment drops below acceptable levels. At this time, there are more options available to the forest manager to minimize disease in future rotations.

In all situations, centers should be mapped and areas critically evaluated before a management direction is developed.

Though removal of infested stumps will likely reduce incidence of disease in future stands, the degree of control is yet to be determined. Factors such as topography, site productivity, adverse soil changes, and intended land use must be taken into consideration if stump removal is to be prescribed.

Without susceptible conifer hosts, *P. weirii* dies out over time. Severely infested sites, then, should not be replanted to conifers unless resistant or disease-tolerant species are used. Even so, the fungus may successfully attack some of the trees and survive into the next rotation, though at a much reduced level. Economic considerations may make this alternative more attractive than accepting heavy losses in highly susceptible species, or replanting to immune but less valuable hardwoods for one or more rotations. In either case, only those species suited to the site should be used, and more susceptible conifers must be weeded from the site if the control is to be effective. In some situations severely infested sites can be seeded to grass and browse species for use as big game habitat.

When only a few, small infection centers occur in a stand, they can be

treated individually in the same ways as heavily infested stands. Or, if costs of treatment exceed expected benefits, a manager may elect to treat the infested areas the same way as the rest of the stand and accept inevitable future losses.

Since boundaries of infection centers cannot be determined accurately, that area within 15 m (50 ft) or more of the apparent limits of the center should also be treated as infected.

Assistance in recognition of the disease and managing diseased stands may be available through such agencies as: State University Extention Service; State Department of Forestry; or Forest Pest Management, U.S. Department of Agriculture, Forest Service.

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