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Black Stain Root Disease

Leptographium wageneri (Kendr.) Wingf.

Hosts:
Douglas-fir
Ponderosa pine
Pinyon pine

Occasionally, other pine species

This disease is common and damaging in and west of the Cascade range of Oregon, Washington and northeast California, and in parts of eastern Oregon.

It rarely causes significant damage in inland or Rocky Mountain forests.

Topics

Introduction	1
Nomenclature	2
Host infection	2
Pathological anatomy	2
Pathogenesis	3
Symptoms	4
Pathogen survival	5
Insect vectors	5
Soil disturbance	6
Precommercial thinning	7
Disease management	8
References cited	9
Field Guide	
Management Guide Index	

Key Points

- A wilt disease
- Vectored by insects, and then spreads root-to-root
- Rarely damaging in the northern Rockies.
- May be serious in off-site plantations
- Avoid attracting the insect vectors

Introduction

Black stain root disease (BSRD) is a wilt-like disease of conifers caused by the native, insect-vectored, fungal pathogen *Leptographium wageneri* (Kendr.) Wingf. (Harrington and Cobb 1983). It is considered one of the five most-damaging root diseases in Western forests (Hadfield et al. 1986). Black stain root disease is widespread across much of the range of its hosts, but incidence and severity, and thus the importance to forest management, vary greatly.

Incidence and severity of BSRD in Douglas-fir and ponderosa pine is greatest west of the Cascade Range in Oregon, Washington, and northern California. In southwest Oregon BSRD was detected in 18.6% of 500, 10- to 30-year-old Douglas-fir plantations, compared to 1.2% of same with Armillaria root disease and 7.0% with laminated root rot (Hessburg et al. 2001). In a subset of eighty of the 500 plantations that underwent intensive BSRD surveys the percentage of crop trees affected was: <0.1% in 93% of stands; 0.1-2% in 3% of stands; 2.1-5% in 3% of stands; and >5% in 1% of the stands (Hessburg et al. 2001). In the most heavily impacted Douglas-fir plantations in southwest Oregon and northwest California, stocking levels were found to be reduced by as much as 50% (DeNitto 1982, Goheen et al. 1983, Goheen et al. 1984, Goheen et al. 1985).

Overview of Black Stain Root Disease Management

1. **Favor mixed species.** Plant mixtures that include resistant species and favor resistant species during precommercial thinning.
2. **Minimize soil disturbance.** Avoid tractor-logging which may compact soil and attract the insect vectors.
3. **Avoid tree injury or remove injured trees of host species.** Injured trees attract vectors. Minimize injuries during skidding, falling, road building, and brushing operations, especially near young trees.

PATHOGEN NOMENCLATURE, TAXONOMY, AND POPULATION GENETICS

There are three varieties of *L. wageneri*. They, and the hosts they primarily affect, are:

- ***L. wageneri* var. *pseudotsugae* -** Douglas-fir (Harrington and Cobb 1987);
- ***L. wageneri* var. *ponderosum* -** ponderosa, Jeffrey, and lodgepole pines (Harrington and Cobb 1986);
- ***L. wageneri* var. *wageneri* -** singleleaf pinyon and pinyon pines (Harrington and Cobb 1986).

Figure 1. Cross-section of infected Douglas-fir root showing typical staining pattern. (Photo courtesy of E. Hansen, Oregon State University)

Hansen and Goheen (1988) reported that 30-40 cm diameter Douglas-fir continued to die on long-term study plots across western Washington and Oregon. Losses in ponderosa pine can be locally severe as well (Byler et al. 1979, Owen 2000).

As an insect-vectored pathogen, and with the vectors generally attracted to weakened trees such as those infected by other root pathogens, *L. wageneri* often occurs in complexes with other root disease fungi (Filip and Goheen 1982, Goheen and Filip 1980, Morrison and Hunt 1988). It has been found colonizing the same roots of Douglas-fir as *Armillaria ostoyae* (Byler et al. 1983, Goheen and Hansen 1978, Morrison and Hunt 1988), *Phellinus weirii* (Filip and Goheen 1982, Goheen and Filip 1980, Morrison and Hunt 1988, Witcosky 1989), and *Heterobasidion annosum* (Kelsey et al. 1998b).

In Idaho and Montana it is rare for BSRD to be found acting as a primary pathogen of Douglas-fir or pines, and the disease is not perceived as a concern in forest management. Where it has been observed it is in conjunction with other root pathogens considered to be the primary agent of mortality (Byler et al. 1983), or in off-site ponderosa pine plantations. Other wood-staining, root-infecting fungi in the genus *Leptographium* can also be found in Idaho and Montana (Wingfield et al. 1994).

Host infection

Leptographium wageneri cannot decay wood or penetrate non-wounded host roots. Smith (1969), using an isolate collected ponderosa pine, found the fungus had no ability to break down cellulose. Hessburg (1984) concluded that *L. wageneri* cannot penetrate to the xylem on its own and requires an existing opening to initiate an infection. *Leptographium wageneri* spreads tree-to-tree at root contacts and grafts like other root disease fungi, but can also grow through the soil for a few centimeters (Goheen 1976, Hessburg and Hansen 1986a, Landis and Helburg 1976). Hessburg and Hansen (2000) demonstrated that minute wounds and natural openings in seedling root systems, where a direct path to the xylem was exposed, serve as infection points for *L. wageneri* hyphae growing in soil.

Pathological anatomy

The pathological anatomy of BSRD has been studied in pinyon pine (Wagener and Mielke 1961), ponderosa pine (Smith 1967), and Douglas-fir (Hessburg and Hansen 1987). Wagener and Mielke (1961) were the first to note that pathogen growth followed the annual rings (Figure 1), a unique pattern compared to the wedge-shaped staining commonly seen with blue stain fungi. Smith (1967) reported that mature host tracheids were the sole habitat for the fungus in infected ponderosa pine, and that hyphal movement between tracheids was via bordered-pit pairs.

Hessburg and Hansen (1987) found the pigmented hyphae of *L. wageneri* exclusively in the sapwood xylem tracheids of Douglas-fir, moving between tracheids via bordered-pit pairs (Figures 2-3).

The most noticeable host response to *L. wageneri* var. *pseudotsugae* infection is partial to complete plugging of tracheids by gums; this and plugging of tracheids by pathogen hyphae were proposed as the primary cause of water blockage and subsequent wilting (Hessburg and Hansen 1987). Non-pathogen-colonized tracheids adjacent to colonized tissue become plugged as well, and stain columns sometimes consist of 20% hyphae-clogged tracheids and 80% gum-clogged tracheids. Colonization of tissue by the amber-colored hyphae results in columns of black-stained tissue extending along infected roots and rootlets of all sizes, and for short distances up the stem from the root collar (Hessburg and Hansen, 1987) (Figure 4). Once a stain column extends up to the root collar it can descend down a non-infected root. Resinosus is often present on the exterior of infected roots or outside stain columns above the root collar. Earlywood is typically colonized prior to latewood within the same growth ring, so that in cross-sectional view the stain columns appear as concentric rings in the earlywood of successive growth rings (Figure 1).

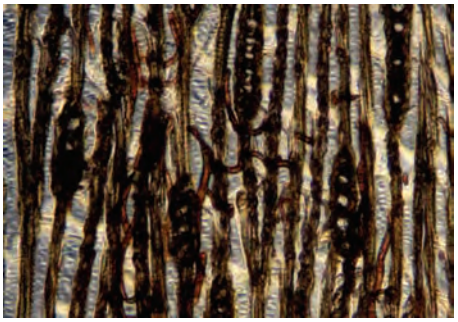

Figure 2. Tangential view of amber-colored *L. wageneri* hyphae within Douglas-fir tracheids.

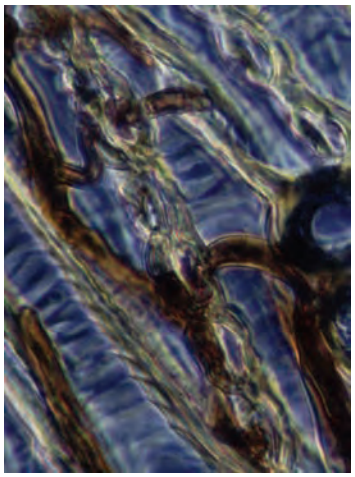

Figure 3. *L. wageneri* hyphae passing through pit pairs of Douglas-fir tracheids.

The most noticeable host response is partial to complete plugging of tracheids by gums.. the primary cause of water blockage and subsequent wilting (Hessburg and Hansen 1987)

Pathogenesis (disease development)

The wilt-inducing nature of BSRD is seen in host responses to advancing colonization. Hessburg (1984) reported a critical water shortage occurred in Douglas-fir seedlings 30 to 40 days after inoculation with *L. wageneri*. Lawson (1988) found colonization of Douglas-fir by *L. wageneri* corresponded to reduced water uptake and transpiration, and that an increase in colonization correlated with a 50% reduction in water transport to needles compared to non-infected controls. Douglas-fir forms pathological heartwood in reaction to colonization by *L. wageneri*, and like normal heartwood, pathological heartwood does not function in water transport. In 20-year-old Douglas-fir, the mean basal, cross-sectional area of normal heartwood in non-infected trees was 23%; the basal, normal-plus-pathological-heartwood in non-symptomatic, diseased trees was 44%; and the basal, normal-plus-pathological-heartwood in symptomatic, diseased trees was 77% (Lawson 1988) (Figure 5). At breast height the percentages of heartwood in these same trees were 18%, 32%, and 64%, respectively.

Figure 4. Staining caused by L. wageneri var. *pseudotsugae* in Douglas-fir. (Photo courtesy of E. Hansen, Oregon State University)

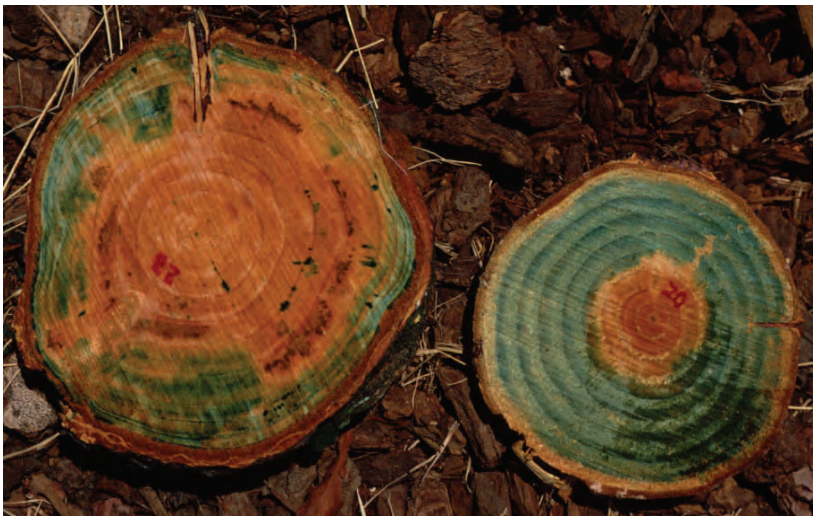

Figure 5. Dye-uptake (green) in BSRD-infected (left) and non-infected (right) basal cross-sections of Douglas-fir (See Lawson 1988). Pathological heartwood in infected cross-section has extended to encompass *L. wageneri* stain columns. (Photo by T.T. Lawson; courtesy of E. Hansen, Oregon State University)

Impacts on water transport are similar in ponderosa pine. Infection by *L. wageneri* had a profound effect on hydraulic conductivity in ponderosa pine roots examined by Joseph et al. (1998). Nearly 100% of the xylem in apparently non-infected, healthy roots was capable of conducting water, compared to just 12% in roots infected with *L. wageneri*. The specific conductivity of infected roots was 5% that of healthy roots, while the conductivity of functional xylem in diseased roots was only one third that of functional xylem in healthy roots.

Symptoms

Figure 6. Symptomatic crowns of Douglas-fir in a black stain root disease infection center.

Black stain root disease in Douglas-fir causes progressive crown symptoms similar to other root diseases (Figure 6). Leader, branch, and needle length is reduced, followed by loss of older needles and a resultant thinning crown, and finally development of off-color, or chlorotic, foliage (Cobb and Platt 1967, Goheen and Hansen 1978, Witcosky and Hansen 1985). Distress cone crops and basal resinosus on infected trees is common but not consistent. The typical black to dark-brown staining has usually reached the root collar by the time chlorosis is evident (Goheen and Hansen 1978).

The interval between appearance of above-ground symptoms until death lengthens with age in *L. wageneri*-infected Douglas-fir. Six- to eight-year-old Douglas-fir with BSRD in coastal British Columbia did not usually exhibit chlorosis or growth reduction prior to death (Morrison and Hunt 1988), while severe leader reduction in young Douglas-fir plantations in southwest Oregon preceded death in most cases by three and four years (Witcosky 1981). Terminal-growth reduction occurred one to four years prior to death in 15- to 25-year-old Douglas-fir in coastal British Columbia (Morrison and Hunt 1988), while measurable height reduction occurred for five or more years without mortality in 20-year-old Douglas-fir plantations in northern California (Lawson 1988).

Damage from BSRD in Douglas-fir stands appears as small infection centers consisting of combinations of dead, symptomatic, and non-infected trees, although infection centers up to 4 ha and involving hundreds of trees can occur (Goheen and Hansen 1978).

Infection centers in ponderosa pine in California can exceed 10 ha (Cobb et al. 1982).

The rate of expansion in BSRD mortality centers has been variously estimated. Hansen and Goheen (1988) monitored 27 infection centers in Douglas-fir across Oregon and Washington for 10 years and calculated an average rate of expansion of 0.7 m/year. Hessburg and Hansen (1986b) measured a growth rate of 2.2 m/year for *L. wageneri* in 20-year-old Douglas-fir, with a maximum rate of 3.6 m/year. In pine, the average rate of spread has been measured at 1.0 m/year, but varied widely from 0 to 7.0 m/year (Cobb et al. 1982); spread was significantly and positively related to host density. Expansion of BSRD centers in *P. monophylla* has been reported as 2 m/year (Wagener and Mielke, 1961). The average rate-of-expansion in 30 BSRD mortality centers in *P. edulis* was estimated at 1.1 m/year (Kearns and Jacobi 2005).

RATE OF ROOT TO ROOT SPREAD

In Douglas-fir: 0.7 to 3.6 meters/year

In ponderosa pine: 0 to 7.0 meters/year

In pinyon pine: average 1.1 meters/year

Pathogen survival

As a non-wood-decay fungus that likely depends on cell contents for nutrition (Hessburg 1984), *L. wageneri*'s ability to survive after the death of a host is relatively limited compared to root diseases caused by the wood decaying fungi, *Armillaria ostoyae*, *Phellinus weirii*, and *Heterobasidion annosum*, all fungi that can survive for decades after host death by utilizing the woody biomass of roots and stumps.

Wagener and Mielke (1961) reported that "the fungus does not appear to maintain its viability for long after the affected host or host part dies", but noted it had been isolated one time from a pinyon pine dead for ten years. Kearns and Jacobi (2005) found the pathogen could be isolated regularly from roots of pinyon dead five years, occasionally from trees dead eight years, and once from a tree estimated dead for 16 years. Goheen (1976) attempted to isolate *L. wageneri* from infected ponderosa pine roots which had been excavated and reburied for six or nine months, and from roots of ponderosa pine which had been dead from 1 to 10 years; only one of 400 isolation attempts, from a tree dead three years, yielded the fungus. Adams and Cobb (1986) tested survival of *L. wageneri* var. *pseudotsugae* by planting Douglas-fir seedlings around *L. wageneri*-infected and non-infected stumps of Douglas-fir in both clearcuts and selection cuts. Subsequent infections of seedlings indicated the fungus remained viable for at least two years following harvest of the infected overstory.

Insect vectors

The pathogen-vector relationship of BSRD is well-defined for *L. wageneri* var. *pseudotsugae* and Douglas-fir. It is less clear for *L. wageneri* var. *ponderosum* and *L. wageneri* var. *wageneri* and their hosts, but very likely involves root- and root collar-feeding bark beetles and weevils in the genera *Hylastes*, *Hylurgops*, and *Pissodes*.

INSECT VECTORS

Root and root collar -feeding bark beetles and weevils are the most commonly reported vectors of *L. wageneri.*

Bark beetle genera
Hylastes and *Hylurgops*

Weevil genera
Pissodes and *Steremnius*

INSECT VECTORS OF BLACKSTAIN ROOT

Figure 7. Adult *Hylastes nigrinus*; average length 4-5 mm.

Figure 8. Adult *Pissodes fasciatus*; average length 5-8 mm.

Figure 9. Adult *Steremnius carinatus*; average length 7-10 mm.

Three beetles vector *L. wageneri* var. *pseudotsugae* (Harrington et al. 1985, Witcosky et al. 1986a). One is a root-feeding bark beetle, *Hylastes nigrinus*, and two are weevils, *Pissodes fasciatus* and *Steremnius carinatus* (Figures 7-9). All are consistently associated with *L. wageneri*-infected Douglas-fir in all stages of decline (Witcosky and Hansen 1985). Prior to being recognized as a vector of *L. wageneri* var. *pseudotsugae* the weevil *S. carinatus* was best known as a regeneration pest (Condrashoff 1968). An important distinction between *S. carinatus* and the other two vectors is that *S. carinatus* is flightless; it is therefore considered a likely factor in within-stand spread.

Leptographium wageneri var. *pseudotsugae* can sporulate adjacent to or within feeding galleries and pupal chambers of insects on infected roots (Witcosky 1981). The spores (Figure 10) stick to the exterior of adult vectors and are carried to new feeding sites. Infections can be initiated after the vectors transmit the spores to seedlings (Harrington et al. 1985), or to the roots of a living tree or recently cut stump that is grafted to a live tree (Witcosky et al. 1986a). The proportion of emerging beetles carrying spores is estimated at under 5% for all three vectors (Witcosky et al. 1986a).

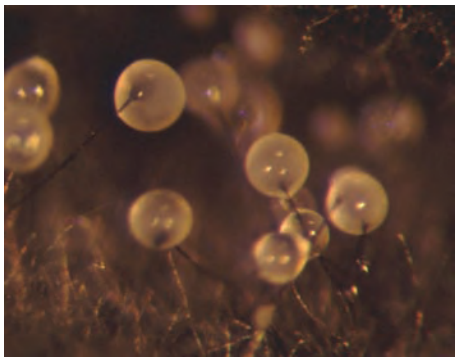

Figure 10. Conidiophores of *L. wageneri* (in vitro) bearing spores in a sticky matrix.

Relation of soil disturbance to disease incidence

Soil compaction can result in increased soil bulk density, reduced porosity, reduced aeration, and changes in drainage (Adams and Froehlich 1981). These soil conditions are likely to result in microsites with year-round or seasonal hypoxic growth conditions, and Douglas-fir roots are known to synthesize ethanol, a common byproduct of stressed trees, under hypoxic or anoxic conditions (Joseph and Kelsey 1997, Kelsey 1996, Kelsey et al. 1998a). A large proportion of this ethanol then diffuses into the soil water (Joseph and Kelsey 1997). Witcosky et al. (1987) demonstrated that *H. nigrinus* and *S. carinatus* are attracted to *L. wageneri*-infected roots of Douglas-fir, as well as ethanol and alpha-pinene, and speculated that periodic anaerobic conditions produced host metabolites, such as ethanol, which attracted the insects.

Incidence of BSRD infection centers in Douglas-fir plantations is closely correlated with soil disturbance (Goheen and Hansen 1978, Hansen 1978, Hessburg et al. 2001). Eighty percent of 202 BSRD infection centers across western Washington and Oregon occurred on disturbed areas such as stream drainages, road edges, clearcut margins, and thinned plantations (Goheen and Hansen 1978).

Hansen (1978) found significantly more BSRD infection centers along roads compared to 25 m or more within the stand. Incidence and damage from BSRD increased in association with a stand history of tractor versus cable yarding (Goheen et al. 1983, Goheen et al. 1984). Hessburg et al. (2001) also found BSRD more frequently adjacent to roads and major skid trails than within plantations and away from skid trails.

Effects of soil compaction may cause tree roots to release ethanol, an attractant to root-feeding beetles.

The beetles may transmit blackstain root disease while feeding.

Relation of precommercial thinning to disease incidence

Black stain root disease has been repeatedly correlated with prior precommercial thinning in Douglas-fir plantations (DeNitto 1985, Goheen and Hansen 1978, Harrington 1983). Precommercial thinning results in an often heavy, homogenous distribution of slash and small stumps across a site. The thinning slash and stumps then release volatiles, including alpha-pinene (a conifer monoterpene attractive to the vectors of black stain root disease) and ethanol, as they dry.

Harrington et al. (1985) examined a precommercial-thinning study in 27-year-old Douglas-fir in northwest California 12 years after treatment. Eighteen of 23 thinned plots had mortality from BSRD, while no BSRD was found on the six non-thinned control plots. It was speculated that the high incidence of disease on thinned plots resulted from increased vector response following thinning, but a baseline disease survey was not available for comparison. DeNitto (1985) surveyed BSRD on thirty, 12- to 27-year-old Douglas-fir plantations, half thinned and half non-thinned, on the Six Rivers National Forest in northern California. Mortality from BSRD was found on nine of 15 thinned plantations three to nine years after precommercial thinning, compared to two of 15 non-thinned plantations.

Witcosky et al. (1986b) used non-baited flight and pitfall traps to monitor the two-year response of *H. nigrinus*, *P. fasciatus*, and *S. carinatus* to precommercial thinnings of Douglas-fir in southwest Oregon. Treatments were applied in September, January, and May of the year prior to trapping. All three vectors responded in higher numbers to thinned plots, regardless of the time of thinning, compared to control plots. Fewer *H. nigrinus* and *P. fasciatus* were caught in the May-thinned plots relative to the September and January thinnings, but no significant difference was seen in the response of *S. carinatus* based on treatment. The conclusion was reached that precommercial thinning done at any time of the year prior to insect flight increased the stand's attractiveness to all BSRD vectors, and that timing of thinning could influence the response of *H. nigrinus* and *P. fasciatus*.

Following the insect-trapping portion of the study (above), Witcosky et al. (1986b) then excavated roots of both stumps and crop trees in the precommercially-thinned plots.

Precommercial thinning

Can increase the incidence of blackstain root disease in areas where the disease is common.

Risk can be reduced by timing thinning so that slash is less attractive to the beetles the following year.

Stumps may be colonized by *L. wageneri* up to seven months after precommercial thinning.

A significantly higher number of *H. nigrinus* individuals and egg galleries were found on roots from the September and January thinnings than from the May thinning. Also, more residual crop trees on thinned plots had insect wounding compared to the non-thinned plots. The mean attack intensity of *H. nigrinus* on stumps from the June-July precommercial thinning was 2.9, compared to 14.6 for stumps from the September and January thinnings, while the attack intensity on residual crop trees was 0.02 in June-July versus 0.14 for September and January. Stumps from the May thinning best supported growth of *L. wageneri* in inoculation trials, and appeared to be suitable for colonization up to seven months following thinning (Witcosky 1985).

Hessburg et al. (2001) surveyed 500 Douglas-fir stands in southwest Oregon and found that precommercially thinned stands were significantly more likely to have BSRD whether prior harvest had been done by either tractor logging or cable yarding. Stands that were at least partially tractor logged, however, were somewhat more likely to have BSRD than stands that had undergone only cable yarding.

MANAGEMENT IN BRIEF

- **Use mixed-species planting on high-risk sites,**
- **Favor species resistant to BSRD when thinning in infection centers,**
- **Perform precommercial thinning between late June and early August,**
- **Minimize soil disturbance and tree injury,**
- **Avoid road-side tree damage during road building, maintenance, and brushing,**
- **Offsite plantations are especially susceptible to BSRD. Ensure planting stock is appropriate for the site.**

Disease management

Management recommendations for BSRD in Douglas-fir arose from Witcosky et al.'s (1986b) work on the effect of seasonal timing of precommercial thinning on vector response. Hansen et al. (1986) expanded upon these findings by recommending: 1) favoring trees other than Douglas-fir when thinning in existing infection centers; 2) reducing the potential for new centers by minimizing disturbance and tree injury; and 3) limiting chances for successful vectoring. Specific actions on high-risk sites, or those within approximately 1.6 km of active infection centers, include: 1) no tractor logging, 2) using mixed-species plantings on high-risk microsites, 3) avoiding road building or other actions that result in tree injuries in plantations, and 4) performing precommercial thinning between late June and early August.

Hessburg et al. (1995) and Hessburg et al. (2001) present preventive and corrective management measures for BSRD. Preventive measures include: 1) use of high-lead and skyline logging in place of tractor logging, 2) use of skid trail layout and dry-season yarding to reduce soil compaction if tractor logging is necessary, 3) removal of high-risk host species along each side of newly constructed roads, 4) avoiding the creation of high-moisture microsites, 5) avoiding use of rotary-blade brushing and branching along roads, and 6) performing precommercial thinning between the end of June and the end of August. Corrective measures included planting a mix of species in new plantations proximal to existing infection centers, favoring non-hosts during thinning in infected stands, and avoiding thinning in active infection centers.

Management of BSRD is critical on ponderosa pine sites with a known history of the disease (Owen 2000).

Recommendations include avoiding development of a pure pine type by promoting mixed-species stands, keeping overly dense stands from developing, and limiting management activities such as precommercial thinning to seasons that are less likely to promote disease development.

PROMOTE TREE SPECIES DIVERSITY AND CONTROL TREE DENSITY

References Cited

Adams, D.H., and Cobb, F.W., Jr. 1986. Infection of outplanted Douglas-fir seedlings by *Verticicladiella wageneri* (black stain root disease) when planted around infected Douglas-fir stumps. Forestry Note No. 98. California Department of Forestry, Sacramento, California. 12 p.

Adams, P.W., and Froehlich, H.A. 1981. Compaction of forest soils. Publication PNW-217. Pacific Northwest Extension Cooperative, Corvallis, Oregon. 13 p.

Byler, J.W., Cobb, F.W., Jr., and Rowney, D.L. 1979. An evaluation of black stain root disease on the Georgetown Divide, El Dorado County, California. Report No. 79-2. USDA Forest Service, Pacific Southwest Region, Forest Insect and Disease Management, San Francisco, California. 14 p.

Byler, J.W., Harrington, T.C., James, R.L., and Haglund, S. 1983. Black stain root disease in Douglas-fir in western Montana. Plant Disease 67: 1037-1038.

Cobb, F.W., Jr., and Platt, W.D. 1967. Pathogenicity of *Verticicladiella wageneri* to Douglas-fir. Phytopathology 57: 998-999.

Cobb, F.W., Jr., Slaughter, G.W., Rowney, D.L., and DeMars, C.J. 1982. Rate of spread of *Ceratocystis wageneri* in ponderosa pine stands in the central Sierra Nevada. Phytopathology 72: 1359-1362.

Condrashoff, S.F. 1968. Biology of *Steremnius carinatus* (Coleoptera: Curculionidae), a reforestation pest in coastal British Columbia. The Canadian Entomologist 100: 386-394.

DeNitto, G. 1982. Distribution of black stain root disease in California. Report No. 82-1. USDA Forest Service, Pacific Southwest Region, Forest Insect and Disease Management, San Francisco, California. 9 p.

DeNitto, G. 1985. Biological evaluation of mortality in Douglas-fir plantations on the Gasquet Ranger District, Six Rivers National Forest. Report No. 85-08. USDA Forest Service, Pacific Southwest Region, Forest Insect and Disease Management, San Francisco, California. 4 p.

Filip, G.M., and Goheen, D.J. 1982. Tree mortality caused by root pathogen complex in Deschutes National Forest, Oregon. Plant Disease 66: 240-243.

Goheen, D.J. 1976. *Verticicladiella wageneri* on *Pinus ponderosa*: epidemiology and interrelationships with insects. Ph.D. Dissertation. University of California, Berkeley. 118 p.

Goheen, D.J., and Filip, G.M. 1980. Root pathogen complexes in Pacific Northwest forests. Plant Disease 64: 793-794.

Goheen, D.J., and Hansen, E.M. 1978. Black stain root disease in Oregon and Washington. Plant Disease Reporter 62: 1098-1102.

Goheen, D.J., Kanaskie, A.M., and Frankel, S.J. 1983. Black stain root disease survey in 15- to 25- year old Douglas-fir plantations, Siskiyou National Forest. Non-numbered report. USDA Forest Service, Pacific Northwest Region, Forest Pest Management, Portland, Oregon. 10 p.

Goheen, D.J., Frankel, S.J., and Michaels, E. 1984. Black stain root disease surveys in 15- to 25- year old Douglas-fir plantations on the Tioga Resource Area, Coos Bay District, Bureau of Land Management. Non-numbered report. USDA Forest Service, Pacific Northwest Region, Forest Pest Management, Portland, Oregon. 14 p.

Goheen, D.J., Goheen, E.M., and Dunham, D.A. 1985. Black stain root disease surveys of the Kelley and McCo-

mas Creek drainages, North Umpqua Resource Area, Roseburg District, Bureau of Land Management. Non-numbered report. USDA Forest Service, Pacific Northwest Region, Forest Pest Management, Portland, Oregon. 5 p.

Hadfield, J.S., Goheen, D.J., Filip, G.M., Schmitt, C.L., and Harvey, R.D. 1986. Root diseases in Oregon and Washington conifers. Report No. R6-FPM-250-86. USDA Forest Service, Pacific Northwest Region, Portland, Oregon. 27 p.

Hansen, E.M. 1978. Incidence of *Verticicladiella wageneri* and *Phellinus weirii* in Douglas-fir adjacent to and away from roads in western Oregon. Plant Disease Reporter 62: 179-181.

Hansen, E.M., and Goheen, D.J. 1988. Rate of increase of black-stain root disease in Douglas-fir plantations in Oregon and Washington. Canadian Journal of Forest Research 18: 942-946.

Hansen, E.M., Goheen, D.J., Hessburg, P.F., Witcosky, J.J., and Schowalter, T.D. 1986. Biology and management of black-stain root disease in Douglas-fir. Pages 13-19 *in*: Forest pest management in southwest Oregon; proceedings of a workshop; August 19-20, 1985; Grants Pass, Oregon. Helgerson, O.T., ed. Forest Research Laboratory, Oregon State University, Corvallis, Oregon. 88 p.

Harrington, T.C. 1983. *Verticicladiella wageneri*: taxonomy and vector relations. Ph.D. Dissertation. University of California, Berkeley. 113 p.

Harrington, T.C., and Cobb, F.W., Jr. 1983. Pathogenicity of *Leptographium* and *Verticicladiella* spp. isolated from roots of western North American conifers. Phytopathology 73: 596-599.

Harrington, T.C., and Cobb, F.W., Jr. 1986. Varieties of *Verticicladiella wageneri*. Mycologia 78: 562-567.

Harrington, T.C., and Cobb, F.W., Jr. 1987. *Leptographium wageneri* var. *pseudotsugae*, var. nov., cause of black stain root disease on Douglas-fir. Mycotaxon 30: 501-507.

Harrington, T.C., Cobb, F.W., Jr., and Lownsbery, J.W. 1985. Activity of *Hylastes nigrinus*, a vector of *Verticicladiella wageneri*, in thinned stands of Douglas-fir. Canadian Journal of Forest Research 15: 519-523.

Hessburg, P.F. 1984. Pathogenesis and intertree transmission of *Verticicladiella wageneri* in Douglas-fir *Pseudotsuga menziesii*. Ph.D. Dissertation. Oregon State University, Corvallis. 164 p.

Hessburg, P.F., and Hansen, E.M. 1986a. Mechanisms of intertree transmission of *Ceratocystis wageneri* in young Douglas-fir. Canadian Journal of Forest Research 16: 1250-1254.

Hessburg, P.F., and Hansen, E.M. 1986b. Soil temperature and rate of colonization of *Ceratocystis wageneri* in Douglas-fir. Phytopathology 76: 627-631.

Hessburg, P.F., and Hansen, E.M. 1987. Pathological anatomy of black stain root disease of Douglas-fir. Canadian Journal of Botany 65: 962-971.

Hessburg, P.F., and Hansen, E.M. 2000. Infection of Douglas-fir by *Leptographium wageneri*. Canadian Journal of Botany 78: 1254-1261.

Hessburg, P.F., Goheen, D.J., and Bega, R.V. 1995. Black stain root disease of conifers. Forest Insect & Disease Leaflet 145 (revised). USDA Forest Service, Washington, D.C. 9 p.

Hessburg, P.F., Goheen, D.J., and Koester, H. 2001. Association of black stain root disease with roads, skid trails, and precommercial thinning in southwest Oregon. Western Journal of Applied Forestry 16: 127-135.

Joseph, G., and Kelsey, R.G. 1997. Ethanol synthesis and water relations of flooded *Pseudotsuga menziesii* (Mirb.) Franco (Douglas-fir) seedlings under controlled conditions. International Journal of Plant Sciences 158: 844-850.

Joseph, G., Kelsey, R.G., and Thies, W.G. 1998. Hydraulic conductivity in roots of ponderosa pine infected with black-stain (*Leptographium wageneri*) or annosus (*Heterobasidion annosum*) root disease. Tree Physiology 18: 333-339.

Kearns, H.S.J., and Jacobi, W.R. 2005. Impacts of black stain root disease in recently formed mortality centers in the pinon-juniper woodlands of southwestern Colorado. Canadian Journal of Forest Research 35: 461-471.

Kelsey, R.G. 1996. Anaerobic induced ethanol synthesis in the stems of greenhouse-grown conifer seedlings. Trees 10: 183-188.

Kelsey, R.G., Joseph, G., and Gerson, E.A. 1998a. Ethanol synthesis, nitrogen, carbohydrates, and growth in tissues from nitrogen fertilized *Pseudotsuga menziesii* (Mirb.) Franco and *Pinus ponderosa* Dougl. ex Laws. seedlings. Trees 13: 103-111.

Kelsey, R.G., Joseph, G., and Thies, W.G. 1998b. Sapwood and crown symptoms in ponderosa pine infected with black-stain and annosum root disease. Forest Ecology and Management 111: 181-191.

Landis, T.D., and Helburg, L.B. 1976. Black stain root disease of pinyon pine in Colorado. Plant Disease Reporter 60: 713-717.

Lawson, T.T. 1988. Stand and site conditions associated with *Leptographium wageneri* var. *pseudotsugae* in Douglas-fir trees and effects of infection on host physiology. Ph.D. Dissertation. University of California, Berkeley. 168 p.

Morrison, D.J., and Hunt, R.S. 1988. *Leptographium* species associated with root disease of conifers in British Columbia. Pages 81-95 *in*: Leptographium Root Diseases on Conifers. Harrington, T.C., and Cobb, F.W., Jr., eds. APS Press, St. Paul, Minnesota. 149 p.

Owen, D.R. 2000. Black stain root disease of ponderosa pine in California. Tree Notes No. 25. California Department of Forestry and Fire Protection, Sacramento, California. 4 p.

Smith, R.S., Jr. 1967. Verticicladiella root disease of pines. Phytopathology 57: 935-938.

Smith, R.S., Jr. 1969. The inability of *Verticicladiella wagenerii* to break down cellulose. Phytopathology 59: 1050 (Abstr.).

Wagener, W.W., and Mielke, J.L. 1961. A staining-fungus root disease of ponderosa, Jeffrey, and pinyon pines. Plant Disease Reporter 45: 831-835.

Wingfield, M.J., Harrington, T.C., and Crous, P.W. 1994. Three new *Leptographium* species associated with conifer roots in the United States. Canadian Journal of Botany 72: 227-238.

Witcosky, J.J. 1981. Insects associated with black stain root disease of Douglas-fir in Oregon. M.Sc. Thesis. Oregon State University, Corvallis. 51 p.

Witcosky, J.J. 1985. The root insect - black stain root disease association in Douglas-fir: vector relationships and implications for forest management. Ph.D. Dissertation. Oregon State University, Corvallis. 134 p.

Witcosky, J.J. 1989. Root beetles, stand disturbance, and management of black-stain root disease in plantations of Douglas-fir. Pages 58-70 *in*: Insects affecting reforestation: biology and damage; proceedings of a meeting of the IUFRO Working Group on Insects Affecting Reforestation (S2.07-03) held under the auspices of the XVIII International Congress of Entomology; Vancouver, British Columbia, Canada; July 3-6, 1988. Alfaro, R.I., and Glover, S.G., eds. Forestry Canada, Pacific and Yukon Region, Pacific Forestry Centre, Victoria, British Columbia. 256 p.

Witcosky, J.J., and Hansen, E.M. 1985. Root-colonizing insects recovered from Douglas-fir in various stages of decline due to black-stain root disease. Phytopathology 75: 399-402.

Witcosky, J.J., Schowalter, T.D., and Hansen, E.M. 1986a. *Hylastes nigrinus* (Coleoptera: Scolytidae), *Pissodes fasciatus* and *Steremnius carinatus* (Coleoptera: Curculionidae) as vectors of black-stain root disease of Douglas-fir. Environmental Entomology 15: 1090-1095.

Witcosky, J.J., Schowalter, T.D., and Hansen, E.M. 1986b. The influence of time of precommercial thinning on the colonization of Douglas-fir by three species of root-colonizing insects. Canadian Journal of Forest Research 16: 745-749.

Witcosky, J.J., Schowalter, T.D., and Hansen, E.M. 1987. Host-derived attractants for the beetles *Hylastes nigrinus* (Coleoptera: Scolytidae) and *Steremnius carinatus* (Coleoptera: Scolytidae). Environmental Entomology 16: 1310-1313.

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