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Precommercial Thinning in Mixed-Species Conifer Plantations Affected by Armillaria and Heterobasidion Root Diseases in West-Central Oregon and Washington: 30-Year Results

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Four 10- to 20-year-old plantations were precommercially thinned to determine the effects on tree growth and mortality caused by armillaria and heterobasidion root diseases. The plantations represented different species compositions with one each of (1) coastal Douglas-fir and noble fir, (2) Douglas-fir and western hemlock, (3) pure Douglas-fir, and (4) Shasta red fir and mountain hemlock. After 30 years, the probabilities of leave-tree survival and actual leave-tree survival (trees/ha [TPH]) were significantly ($P \leq 0.05$) higher in thinned versus unthinned plots in one of the four plantations with no significant differences in the other three plantations. Most tree mortality was caused by armillaria root disease. Despite the high frequency of *Heterobasidion occidentale* in overstory stumps, only two leave trees in one plantation were killed by this fungus after 30 years. Quadratic mean diameter (QMD) growth and basal area (BA) (per ha) growth of leave trees were significantly ($P \leq 0.05$) greater in thinned than in unthinned plots in one plantation for QMD and in three plantations for BA. Precommercial thinning does not appear to exacerbate the incidence of leave-tree mortality from armillaria or heterobasidion root diseases after 30 years, and leave-tree QMD and BA growth increased significantly in most but not all plantations for the tree species sampled. Armillaria and heterobasidion root diseases are not an impediment to precommercial thinning in plantations or stands similar to those we studied.

Keywords: Thinning, *Armillaria ostoyae*, *Heterobasidion occidentale*, tree mortality, tree growth

Armillaria root disease caused by the fungus, *Armillaria ostoyae* (Romagnesi) Herink, is one of the most common forest root diseases in Oregon and Washington and affects growth and survival of all conifer species to some degree (Hadfield et al. 1986, Goheen and Willhite 2006, Shaw et al. 2009, Filip et al. 2015). Interior Douglas-fir, *Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco, white fir, *Abies concolor* (Gord. & Glend.) Lindl. ex Hildebr., and grand fir, *Abies grandis* (Dougl. ex D. Don) Lindl., are severely damaged by armillaria root disease in Oregon and Washington (Goheen and Willhite 2006, Filip et al. 2015). Most other conifer species are moderately damaged. Western larch, *Larix occidentalis* Nutt., incense cedar, *Calocedrus decurrens* (Torr.) Florin, and Port-Orford-cedar, *Chamaecyparis lawsoniana* (A. Murr.) Parl., are seldom damaged by armillaria root disease.

An earlier described species, *Armillaria solidipes* Peck., has been proposed to replace *A. ostoyae* (Burdsall and Volk 2008), but this proposed name change is controversial and remains unresolved (Redhead et al. 2011). Seven species of *Armillaria*, to date, have

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This article uses metric units; the applicable conversion factors are: centimeters (cm): 1 cm = 0.39 in.; meters (m): 1 m = 3.3 ft; square meters (m^2): 1 m^2 = 10.8 ft^2; millimeters (mm): 1 mm = 0.039 in.; kilometers (km): 1 km = 0.6 mi; hectares (ha): 1 ha = 2.47 ac.

been reported in Oregon and Washington: *A. ostoyae, Armillaria mellea* (Vahl. ex Fr.) Kummer, *Armillaria gallica* Marxmüller & Romagnesi, *Armillaria sinapina* Bérubé & Dessureault, *Armillaria calvescens* Bérubé & Dessureault, *Armillaria cepistipes* Velenovsky, and *Armillaria altimontana* Brazee, B. Ortiz, Banik & D.L. Lindner (formerly NABS 10). Other *Armillaria* species may occur in Oregon and Washington, and a thorough study of species distribution needs to be conducted. Only *A. ostoyae* is considered highly virulent and a major concern for Pacific Northwest conifers. Single clones of *A. ostoyae*, the "humongous fungus," have been estimated to extend as much as 970 ha and live for 2,200 years in a mixed-conifer forest in northeastern Oregon (Ferguson et al. 2003). The fungus often causes openings in young plantations (Johnson 1975, Filip and Goheen 1995, Cruickshank et al. 1997). Besides tree mortality, *Armillaria* infections cause root lesions and growth reductions (Bloomberg and Morrison 1989, Cruickshank et al. 1997, Cruickshank 2002, 2010, Morrison 2011, Cleary et al. 2012, Cruickshank and Filipescu 2012, Filip et al. 2015).

For interior Douglas-fir in British Columbia, a higher proportion of armillaria root-diseased trees resulted in less stand basal area (Cruickshank et al. 2009), and individual tree-volume reductions of up to 27% were recorded (Cruickshank et al. 2011). In addition, per ha yield reductions of 7–15% of interior Douglas-fir were less affected by the proportion of infected primary roots per tree than by the cumulative time since initial infection (Cruickshank et al. 2011). Trees of reduced vigor resulting from poor planting, poor site quality, maladaptation, drought, overcrowding, climate change, and other stresses may be more susceptible to mortality associated with *A. ostoyae* than vigorous trees (Wargo and Shaw 1985, Shaw and Kile 1991). Treatments to prevent or alleviate poor vigor often are recommended to reduce mortality (Morrison 1981, Hadfield et al. 1986, Williams et al. 1986, Shaw et al. 2009).

Heterobasidion occidentale Garbelotto & Otrosina (formerly *Heterobasidion annosum* S-type) (Otrosina and Garbelotto 2010) causes a root disease and butt rot of many species of Pacific Northwest conifers (Hadfield et al. 1986, Schmitt et al. 2000, Goheen and Willhite 2006, Shaw et al. 2009). In western Oregon and Washington, the disease manifests itself mainly as a butt rot of wounded trees, especially older hemlocks, spruces, and true firs (*Abies* spp.), but direct mortality of young trees can occur. Wetwood often is associated with butt decay caused by *H. occidentale* (Shaw et al. 1995). White fir, grand fir, Pacific silver fir, *Abies amabilis* Dougl. ex Forbes, and mountain hemlock, *Tsuga mertensiana* (Bong.) Carr., are severely damaged by *H. occidentale* (Goheen and Willhite 2006). *H. occidentale* often colonizes thinning stumps of true firs (Woodward et al. 1998, Schmitt et al. 2000, Filip et al. 2006) and has been reported in western hemlock, *Tsuga heterophylla* (Raf.) Sarg., stands that have been precommercially (Chavez et al. 1980) or commercially (Goheen et al. 1980) thinned, and noble fir, *Abies procera* Rehd., stands that have been commercially thinned and wounded (Sullivan et al. 2001). Douglas-firs (both varieties) are seldom damaged by *H. occidentale* in Oregon and Washington, but susceptibility appears to increase with latitude. Douglas-fir is considered moderately susceptible in British Columbia and northern Idaho (Woodward et al. 1998). Western larch, cedar, spruce, and some pine species are seldom damaged by *H. occidentale* or the related fungus, *Heterobasidion irregulare* Garbelotto & Otrosina (formerly *Heterobasidion annosus* P-type) (Goheen and Willhite 2006, Otrosina and Garbelotto 2010). Ponderosa pine, *Pinus ponderosa* Dougl. ex Laws. and lodgepole pine, *Pinus contorta* Dougl. ex Loud., however, are moderately damaged by *H. irregulare*.

Precommercial thinning of several conifer species can improve vigor and growth of leave trees (Tappeiner et al. 1982, Velazquez-Martinez et al. 1992, Rosso and Hansen 1998), but long-term thinning effects in root-infected plantations are largely unknown. Armillaria and heterobasidion root diseases often are associated with colonized stumps, and the incidence of infection and mortality of host trees increases with stump size (Filip 1979, Roth et al. 1980, Shaw and Kile 1991, Cruickshank et al. 1997, Woodward et al. 1998, Schmitt et al. 2000). Because stumps created by precommercial thinning are relatively small, they may not be adequate food-bases for infection and mortality. In coastal British Columbia, however, Cruickshank et al. (1997) found an increase in the amount of *A. ostoyae*-caused root lesions on residual trees after precommercial thinning of Douglas-fir, but rapid growth of residual trees resulted in 70% of the *Armillaria* root lesions being callused and therefore dormant. *A. ostoyae*-caused mortality of coastal Douglas-fir and other associated tolerant species thus may be reduced through precommercial thinning, because tree vigor is maintained or improved, whereas the size of the fungal food-base is kept to a minimum and most root lesions, should they occur, may become callused and inactive. Freshly cut, relatively small stumps (<20 cm diameter) of noble fir Christmas trees, however, can be infected by *H. occidentale* with subsequent fungal spread across roots and widespread mortality of adjacent trees (Dart et al. 2007), but this has not been observed in natural forests in the Pacific Northwest.

In central Oregon, precommercial thinning decreased leave-tree mortality caused by *A. ostoyae* after 40 years in ponderosa pine compared with mortality of leave trees in unthinned stands (Filip et al. 2009). In addition, leave-tree basal area/ha growth was significantly ($P = 0.03$) greater in thinned plots. Although ponderosa pine is considered moderately susceptible to armillaria root disease (Goheen and Willhite 2006), severe cases of pine root rot occurred in South-Central Washington (Shaw et al. 1976, 2012, Roth et al. 1980).

Ten years ago, we reported that leave-tree mortality between thinned and unthinned plots was not significantly different in four, mixed-species, infected plantations in West-Central Oregon and Washington (Filip and Ganio 2004). Leave-tree quadratic mean diameter growth and basal area/ha growth, however, were significantly greater in thinned plots. Thirty years after thinning, we report again on this comparison of leave-tree growth and mortality in precommercially thinned and unthinned plots.

Methods

Study Areas

In 1978–1981, we selected four plantations with root disease-caused mortality in 10- to 20-year-old trees that were planted (coastal Douglas-fir, *P. menziesii* [Mirb.] Franco var. *menziesii*) or naturally regenerated (all other species). The sites were clearcut of old-growth Douglas-fir and western hemlock on three sites and Shasta red fir, *Abies magnifica* var. *shastensis* A. Murr., and mountain hemlock on one site (Table 1). These plantations had young-tree mortality caused by *A. ostoyae*, as determined by pairings of single-spore isolates (Morrison et al. 1985). Other *Armillaria* species probably exist on our sites, especially in the old-growth stumps as saprophytes or weak pathogens, but we did not do a thorough survey to sample for them. Most of the young-tree mortality before thinning was caused by *A. ostoyae*, but *H. occidentale* also killed some trees.

Table 1. Stocking, tree mortality, and overstory-stump infection before treatment in thinned and unthinned, 0.1-ha plots in four *Armillaria* and *Heterobasidion*-infected plantations in West-Central Oregon and Washington.

	Treatment	Snoqualmie: Douglas-fir/noble fir	Evans Mountain: Douglas-fir/hemlock	McKenzie River: Douglas-fir	High Cascades: Red fir/hemlock
Plot pair		No. live TPH >15 cm in height (% tree mortality)			
1	Thinned	2,687 (4)	1,857 (9)	3,399 (7)	3,033 (6)
	Unthinned	1,778 (7)	1,522 (11)	3,656 (6)	1,689 (6)
2	Thinned	2,589 (7)	2,342 (10)	5,029 (3)	2,549 (7)
	Unthinned	3,428 (11)	3,122 (4)	4,130 (4)	3,656 (9)
3	Thinned	1,927 (9)	3,083 (5)	2,974 (8)	7,657 (1)
	Unthinned	3,152 (2)	1,452 (5)	2,608 (4)	9,030 (3)
4	Thinned	2,737 (3)		3,675 (9)	10,908 (7)
	Unthinned	2,460 (7)		2,470 (6)	6,155 (2)
Mean	Thinned	2,485 (5)	2,428 (8)	3,769 (6)	5,543 (5)
	Unthinned	2,705 (7)	2,033 (6)	3,216 (5)	5,133 (4)
Infected overstory stumps					
All plots		No. of infected stumps (% of total no. of stumps)			
Armillaria spp.	Thinned	3 (3)	2 (2)	5 (9)	26 (50)
	Unthinned	1 (1)	1 (3)	4 (9)	13 (22)
H. occidentale	Thinned	57 (43)	8 (12)	9 (16)	31 (57)
	Unthinned	55 (43)	7 (21)	8 (12)	44 (64)

Mortality caused by either *A. ostoyae* or *H. occidentale* probably was only infrequent in the former old-growth stands but became more evident after harvesting and subsequent regeneration. Both *Armillaria* spp. and *H. occidentale* were present in residual stumps of the former overstories (Table 1). All plantations were located on the west side of the Cascade Range.

The Snoqualmie site contains a Douglas-fir/noble fir plantation that is 32 km southeast of Enumclaw, Washington, on the Snoqualmie Ranger District, Mount Baker-Snoqualmie National Forest (47°N latitude, 122°W longitude). Elevation is 1,000–1,130 m with a 35–60% slope and east aspect. The plantation is composed mostly of equal amounts of Douglas-fir and noble fir with some western hemlock, western redcedar, *Thuja plicata* Donn ex D. Don, and western white pine, *Pinus monticola* Dougl. ex D. Don. Western hemlock-salal, *Gautheria shallon* Pursh-beargrass, *Xerophyllum tenax* (Pursh) Nutt., is the dominant plant association (Henderson et al. 1992). Plots were established and thinned in 1978.

The Evans Mountain site contains a Douglas-fir/western hemlock plantation that is 72 km east of Salem, Oregon, near Evans Mountain on the Salem District, Bureau of Land Management (45°N latitude, 122°W longitude). Elevation is 1,200 m with a 0–30% slope and a south to west aspect. More than half of the trees are Douglas-fir with some western hemlock. Western hemlock/dwarf Oregon grape, *Berberis nervosa* Pursh-salal, is the dominant plant association (Hemstrom et al. 1987). Plots were established and thinned in 1981, but one plot pair was eliminated when the designated unthinned plot was accidentally thinned.

The McKenzie River site contains a pure Douglas-fir plantation that is 80 km east of Eugene, Oregon, on the McKenzie River Ranger District, Willamette National Forest (44°N latitude, 122°W longitude). Elevation is 975–1,200 m with a 5–85% slope and a north to northwest aspect. The plantation is composed primarily of Douglas-fir with some hemlock. Western hemlock/rhododendron, *Rhododendron macrophyllum* D. Don-salal is the dominant plant association (Hemstrom et al. 1987). Plots were established and thinned in 1978.

The High Cascades site contains a Shasta red fir/mountain hemlock plantation that is 65 km northeast of Medford, Oregon, on the High Cascades Ranger District, Rogue River-Siskiyou National Forest (43°N latitude, 122°W longitude). Elevation is 1,700–1,800 m with a 30% slope and a north aspect. The plantation is composed mostly of equal amounts of red fir and mountain hemlock with some western white pine. Mountain hemlock-Shasta red fir/thinleaved huckleberry, *Vaccinium membranaceum* Dougl., is the dominant plant association (Atzet and McCrimmon 1990). Plots were established in 1980 and thinned in 1981.

Treatments, Data Collection, and Statistical Analysis

At least four paired, 0.1-ha square plots initially were established in each plantation. We attempted to locate paired plots so that equal amounts of root pathogen-caused tree mortality were present in each plot pair; the means of all four sites are approximately equal in percent mortality (Table 1). The plots were thinned or not after random selection of the plots. A 6-m-wide buffer of thinned trees was left around plots that were thinned; a similar-sized buffer of unthinned trees was left around plots that were not thinned.

The following data were recorded for each plot before treatment: (1) number of live trees of ≥ 15 cm in height by species; (2) number of potential leave trees; (3) number of dead trees by species and cause of death; and (4) number of residual overstory stumps by species, diameter at stump top, and presence of root-disease fungi. Leave trees were ≥ 2.5 cm dbh and were selected and marked in all plots on a 3- × 3-m spacing (mean 1,075 trees/ha [TPH]) based on the best size and form regardless of species. All nonleave trees of ≥ 2.5 cm dbh were felled in thinned plots. All trees of <2.5 cm dbh were not treated.

Before and after thinning, dead trees and stumps were examined for root pathogens by partially excavating and dissecting major roots and observing signs of infection (stain, decay, mycelial fans, and rhizomorphs). Root systems were not totally excavated, except for small trees that could be tipped over, so the full extent and age of root infection are not known. We were particularly interested in recording the incidence of the root pathogens *A. ostoyae*, *H. occidentale*, *Leptographium wageneri* var. *pseudotsuga* Harrington & Cobb (the cause of black stain root disease of Douglas-fir), and *Phellinus sulfurascens* Pilát (formerly *Phellinus weirii*; the cause of laminated root rot). Infected wood samples were returned to the laboratory for culturing to supplement the field diagnosis. All 0.1-ha plots were inspected every other year for mortality of leave trees and incidence of causal agents. We did not record stem wounding or sample for

stem decay, a common result of *H. occidentale* infection, especially in true fir and hemlock.

Thirty-year tree survival (trees/ha [TPH]), quadratic mean diameter (QMD) (cm) growth, and basal area (BA) (m^2/ha) growth were calculated based on 0.02-ha circular plots for leave trees, nonleave trees, or ingrowth and used for statistical analysis. Because tree diameters were not recorded at the beginning of the study, a subsample was measured in 1991 as follows. One 0.02-ha circular plot was randomly established near the center of each 0.1-ha square plot. For all trees of ≥ 2.5 cm dbh in each circular plot, two cores were removed with an increment borer at 90° from each other, at 1.37 m aboveground. Radial increment (to the nearest mm) was measured in the field with a ruler for the last 10 years (back to 1981) for each core and averaged for each tree. This established dbh for 1981. The 1991 dbh was measured with a diameter tape. Each leave tree in the plot was marked with an aluminum nail and numbered tag at 1.37 m. Each nonleave tree of ≥2.5 cm dbh was marked only with an aluminum nail at 1.37 m. Ingrowth was considered to be trees that were not present or were <2.5 cm dbh in 1981. In 1991, 2001, and 2011 for all plot trees including ingrowth, dbh was measured with a diameter tape at the nail (if present), and any tree mortality was recorded by cause as above.

For this article, treatment differences in tree growth and survival were calculated at one time point: 30 years after treatment from 1981 to 2011. A mixed general linear model (Garrett et al. 2004) applied to each plantation and paired plots (blocks) within plantations were used to assess differences between thinned and unthinned plots for the responses of 30-year QMD growth, BA growth, and surviving TPH. Using tree dbh measured separately for leave trees, nonleave trees, and ingrowth trees, we calculated QMD, BA, and surviving TPH at the plot level for each tree type. These plot responses were assumed to be approximately normally distributed and modeled as the following regression for each plantation

$$\text{Plot response}_{ijk} = a_i + b_i \cdot \text{year}_j + B_k + \varepsilon_{ijk}$$

where plot response is QMD, BA, or surviving TPH in a plot within block B (set of paired plots), a_i and b_i are the regression line intercept and slope for treatment i (i = thinned or unthinned) on year j (j = 1981, 1991, 2001, and 2011), B_k (k = 1, 2, 3, or 4; 1 site had 3 blocks and the other 3 had 4 blocks) is the random effect due to block k, and ε_{ijk} is the residual error. Because the plot within block is the experimental unit, to account for repeated measurements in the same plot ik, after some testing, we modeled the residual error covariance for two periods, j and v, as follows: $\text{cov}(\varepsilon_{ijk}, \varepsilon_{ivk}) = \sigma^2\rho^{|j-v|}$ for the same plot ik and = 0 between plots in same block k; σ^2 and ρ are two constants to be estimated. The correlation between $2 \times j$ and v is given by $\rho^{|j-v|}$. The estimated parameters of this covariance matrix and estimated correlations for leave trees are shown in Tables 2A–C and 3A–C. The SAS MIXED procedure (SAS 9.3; SAS Institute, Inc. 2010) for repeated measurements was applied for the regression parameter estimation and statistical treatment comparisons. The SAS LIFETEST procedure was used to estimate the probability of a tree surviving longer than the given time, and the logrank test was used to compare the survival curves of thinned and unthinned plots (Lee and Wang 2003).

Table 2A. Leave-tree covariance parameter estimates of the block (σ^2_B) random effect and residual error (σ^2, ρ) from the fitted TPH regression model.

Site	Covariance parameter	Estimate	SE	*P* value
Evans Mountain	σ^2_B	582.84	716.60	0.21
	ρ	0.59	0.25	0.02
	σ^2	446.41	267.66	0.05
High Cascades	σ^2_B	382.26	1,576.51	0.40
	ρ	0.91	0.07	<0.0001
	σ^2	2,673.47	1,879.52	0.08
McKenzie River	σ^2_B	2,161.51	1,883.24	0.13
	ρ	0.29	0.26	0.26
	σ^2	756.703	271.88	0.003
Snoqualmie	σ^2_B	2,862.09	2,950.41	0.17
	ρ	0.69	0.17	<0.0001
	σ^2	2,052.24	1,116.25	0.03

Table 2B. Leave-tree covariance parameter estimates of the block (σ^2_B) random effect and residual error (σ^2, ρ) from the fitted QMD regression model.

Site	Covariance parameter	Estimate	SE	*P* value
Evans Mountain	σ^2_B	0		
	ρ	0.69	0.16	<0.0001
	σ^2	1.59	0.78	0.02
High Cascades	σ^2_B	1.35	1.62	0.20
	ρ	0.85	0.11	<0.0001
	σ^2	1.40	0.97	0.08
McKenzie River	σ^2_B	1.99	1.76	0.13
	ρ	0.66	0.18	0.0004
	σ^2	0.53	0.27	0.03
Snoqualmie	σ^2_B	0.04	0.30	0.45
	ρ	0.76	0.16	<0.0001
	σ^2	0.69	0.43	0.05

Table 2C. Leave-tree covariance parameter estimates of the block (σ^2_B) random effect and residual error (σ^2, ρ) from the fitted BA regression model.

Site	Covariance parameter	Estimate	SE	*P* value
Evans Mountain	σ^2_B	0		
	ρ	0.89	0.08	<0.0001
	σ^2	2,880.09	1,826.46	0.06
High Cascades	σ^2_B	0		
	ρ	0.80	0.11	<0.0001
	σ^2	808.42	378.91	0.02
McKenzie River	σ^2_B	654.90	777.56	0.20
	ρ	0.82	0.13	<0.0001
	σ^2	668.07	455.76	0.07
Snoqualmie	σ^2_B	18.59	448.33	0.48
	ρ	0.91	0.07	<0.0001
	σ^2	852.49	635.93	0.09

Results

Douglas-Fir/Noble Fir Plantation (Snoqualmie)

Most of the young-tree mortality before thinning was caused by *A. ostoyae*, but about 10% was attributed to *H. occidentale*. Overstory stumps (mean 314/ha, 45.2 cm diameter) were mostly western hemlock with some Douglas-fir. About 43% of the stumps, all hemlock, were infected by *H. occidentale*, and only 2% had *Armillaria* spp., although this estimate was probably very conservative, since most of the deeper roots were not examined (Table 1). Species of *Armillaria* in overstory stumps were not determined. No other root diseases were found.

Thirty years after treatment, the probability of leave-tree survival was not significantly (P = 0.15) different between thinned and unthinned plots (Figure 1A; Table 4A). The 30-year change in actual leave-tree survival (TPH) also was not significantly

Table 3A. Leave tree estimated 10-year correlation matrix from the fitted TPH regression model.

Site	1981	1991	2001	2011
Evans Mountain				
1981	1	0.59	0.35	0.21
1991	0.59	1	0.59	0.35
2001	0.35	0.59	1	0.59
2011	0.21	0.35	0.59	1
High Cascades				
1981	1	0.91	0.82	0.74
1991	0.91	1	0.91	0.82
2001	0.82	0.91	1	0.91
2011	0.74	0.82	0.91	1
McKenzie River				
1981	1	0.29	0.08	0.024
1991	0.29	1	0.29	0.083
2001	0.08	0.29	1	0.29
2011	0.02	0.08	0.29	1
Snoqualmie				
1981	1	0.69	0.48	0.33
1991	0.69	1	0.69	0.48
2001	0.48	0.69	1	0.69
2011	0.33	0.48	0.69	1

Table 3B. Leave tree estimated 10-year correlation matrix from the fitted QMD regression model.

Site	1981	1991	2001	2011
Evans Mountain				
1981	1	0.69	0.48	0.33
1991	0.69	1	0.69	0.48
2001	0.48	0.69	1	0.69
2011	0.33	0.48	0.69	1
High Cascades				
1981	1	0.85	0.72	0.61
1991	0.85	1	0.85	0.72
2001	0.72	0.85	1	0.85
2011	0.61	0.72	0.85	1
McKenzie River				
1981	1	0.66	0.43	0.28
1991	0.66	1	0.66	0.43
2001	0.43	0.66	1	0.66
2011	0.28	0.43	0.66	1
Snoqualmie				
1981	1	0.76	0.58	0.44
1991	0.76	1	0.76	0.58
2001	0.58	0.76	1	0.76
2011	0.44	0.58	0.76	1

($P = 0.42$) different between thinned and unthinned plots (Figure 2A; Tables 4A and 5). Most mortality of leave trees in both thinned and unthinned plots was caused by *A. ostoyae*. Based on the 0.1-ha plots, 2.2 TPH/year (0.2%/ha/year) were killed by *A. ostoyae* in thinned plots, and 3.7 TPH/year (0.4%/ha/year) were killed by *A. ostoyae* in unthinned plots after 30 years. Most of the dead leave trees in thinned plots were Douglas-fir that were killed by *A. ostoyae*, but two Douglas-firs were killed by *H. occidentale*. The dead noble fir and western hemlock leave trees were killed primarily by *A. ostoyae*. Similarly, most leave trees killed in unthinned plots were Douglas-fir, but noble fir, western hemlock, and western redcedar also were killed. Over 30 years, 7 TPH were killed by windsnap in thinned plots and 2 TPH in unthinned plots.

Leave trees in thinned plots had significantly ($P = 0.08$ at α level $= 0.10$) greater QMD growth (Table 4B; Figure 3A). BA growth of leave trees also was significantly ($P = 0.003$) greater in thinned plots (Table 4C; Figure 4A).

Table 3C. Leave tree estimated 10-year correlation matrix from the fitted BA regression model.

Site	1981	1991	2001	2011
Evans Mountain				
1981	1	0.89	0.79	0.70
1991	0.89	1	0.89	0.79
2001	0.79	0.89	1	0.89
2011	0.70	0.79	0.89	1
High Cascades				
1981	1	0.80	0.63	0.50
1991	0.80	1	0.80	0.63
2001	0.63	0.80	1	0.80
2011	0.50	0.63	0.80	1
McKenzie River				
1981	1	0.82	0.67	0.55
1991	0.82	1	0.82	0.67
2001	0.67	0.82	1	0.82
2011	0.55	0.67	0.82	1
Snoqualmie				
1981	1	0.91	0.82	0.74
1991	0.91	1	0.91	0.82
2001	0.82	0.91	1	0.91
2011	0.74	0.82	0.91	1

Between 1981 and 2011, nonleave TPH in unthinned plots decreased with some mortality due to *A. ostoyae* but most due to suppression or other causes (Table 5). Differences in actual surviving nonleave TPH after 30 years were not significant ($P = 0.16$) (Table 6). QMD growth ($P < 0.0001$) and BA growth ($P < 0.0001$) of nonleave trees increased significantly after 30 years (Table 6).

Between 1991 and 2011, ingrowth TPH increased more for the unthinned plots than for the thinned plots, but differences were not significant ($P = 0.62$) (Table 7). Differences also were not significant for ingrowth QMD ($P = 0.98$) and BA ($P = 0.52$) between thinned and unthinned plots (Table 7).

Douglas-Fir/Hemlock Plantation (Evans Mountain)

All young-tree mortality before thinning was caused by *A. ostoyae* primarily in Douglas-fir. Overstory stumps (mean 165/ha, 57.4 cm diameter) were equally of Douglas-fir and hemlock. About 17% of the overstory stumps, primarily hemlock, had *H. occidentale*; 3% had *Armillaria* spp. (Table 1). No other root diseases were detected.

Thirty years after treatment, the probabilities of leave-tree survival ($P = 0.24$) (Tables 4A and 5; Figure 1B) and actual leave-tree survival (TPH) ($P = 0.14$) (Figure 2B) were not significantly different between thinned and unthinned plots (Tables 4A and 5). Over 30 years based on the 0.1-ha plots, 3.7 TPH/year (0.5%/ha/year) were killed by *A. ostoyae* in thinned plots, and 2.2 TPH/year (0.3%/ha/year) were killed by *A. ostoyae* in unthinned plots. Most of these leave trees were Douglas-fir. Over 30 years, about 22 leave TPH were killed by windsnap in thinned plots and 57 TPH in unthinned plots. Although there were slightly fewer trees killed by root disease in unthinned plots than in thinned plots (2.2 versus 3.7 TPH/year), the additional mortality from windsnap in the unthinned plots accounted for the lack of significant differences in leave-tree mortality between thinned and unthinned plots.

Leave trees in thinned plots had greater QMD growth than did trees in unthinned plots, but differences were not significant ($P = 0.40$) (Table 4B; Figure 3B). There was also no significant ($P = 0.22$) difference in BA growth of leave trees between thinned and unthinned plots (Table 4C; Figure 4B).

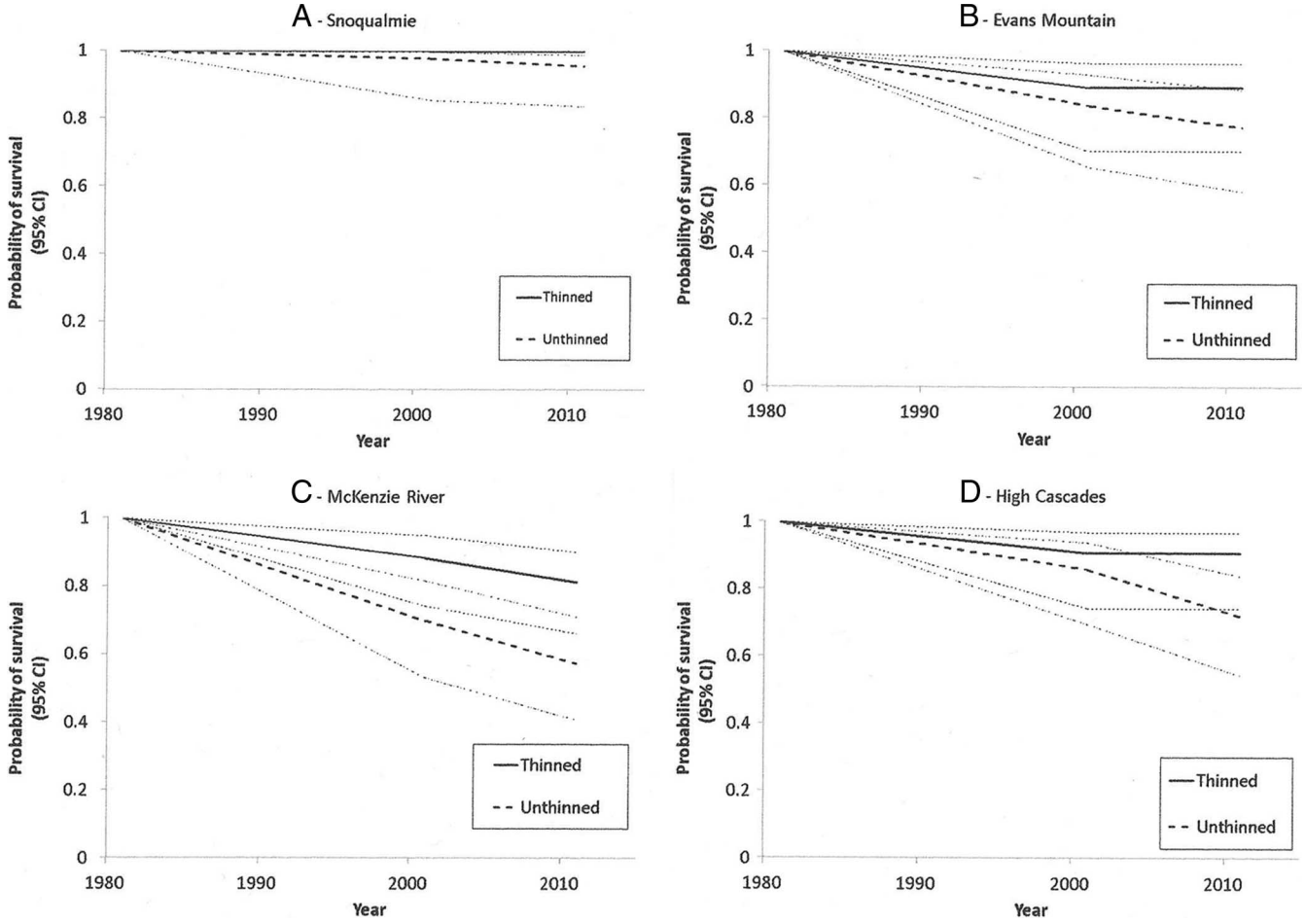

Figure 1. Probability of leave-tree survival for thinned and unthinned plots at four 10-year periods in mixed-conifer plantations with armillaria root disease at four sites: Snoqualmie (A), Evans Mountain (B), McKenzie River (C), and High Cascades (D). The dotted lines represent the 95% confidence limits.

Table 4A. Actual leave TPH and probability of leave-tree survival comparisons of thinned versus unthinned treatments for slope and 30-year TPH differences.

Site	TPH slope difference estimate (SE)	30-year TPH difference estimate (SE)	TPH difference *P* value	Survival sign	Survival *P* value
Evans Mountain	1.12 (0.72)	83.25 (53.5)	0.14	+	0.24
High Cascades	1.46 (0.87)	108.04 (64.64)	0.11	+	0.056**
McKenzie River	2.02 (0.90)	149.78 (66.39)	0.03*	+	0.017*
Snoqualmie	1.01 (1.23)	74.92 (91.28)	0.42	+	0.15

Positive estimates indicate that TPH in thinned plots is greater than in unthinned plots. Positive signs indicate that the probability of survival in thinned plots is greater than in unthinned plots.
* Statistically significant at α level = 0.05.
** Statistically significant at α level = 0.10.

Table 4B. Leave-tree QMD comparison of thinned versus unthinned treatments for slope and 30-year growth.

Site	QMD slope difference estimate (SE)	30-year QMD growth estimate (SE) (cm)	QMD *P* value
Evans Mountain	0.03 (0.04)	83.25 (53.5)	0.40
High Cascades	0.14 (0.02)	108.04 (64.64)	<0.0001*
McKenzie River	0.003 (0.02)	149.78 (66.39)	0.88
Snoqualmie	0.04 (0.02)	74.92 (91.28)	0.08**

Positive estimates indicate that the QMD in thinned plots is greater than in unthinned plots.
* Statistically significant at α level = 0.05.
** Statistically significant at α level = 0.10.

Table 4C. BA comparison of thinned versus unthinned treatments for slope and 30-year growth.

Site	BA slope difference estimate (SE)	30-year BA-growth estimate (SE) (m^2/ha)	BA *P* value
Evans Mountain	1.43 (1.13)	42.97 (33.96)	0.22
High Cascades	3.41 (0.67)	102.40 (20.03)	<0.0001*
McKenzie River	1.70 (0.58)	51.06 (17.28)	0.007*
Snoqualmie	1.63 (0.49)	48.83 (14.75)	0.003*

Positive estimates indicate that the QMD in thinned plots is greater than in unthinned plots.
* Statistically significant at α-level = 0.05.

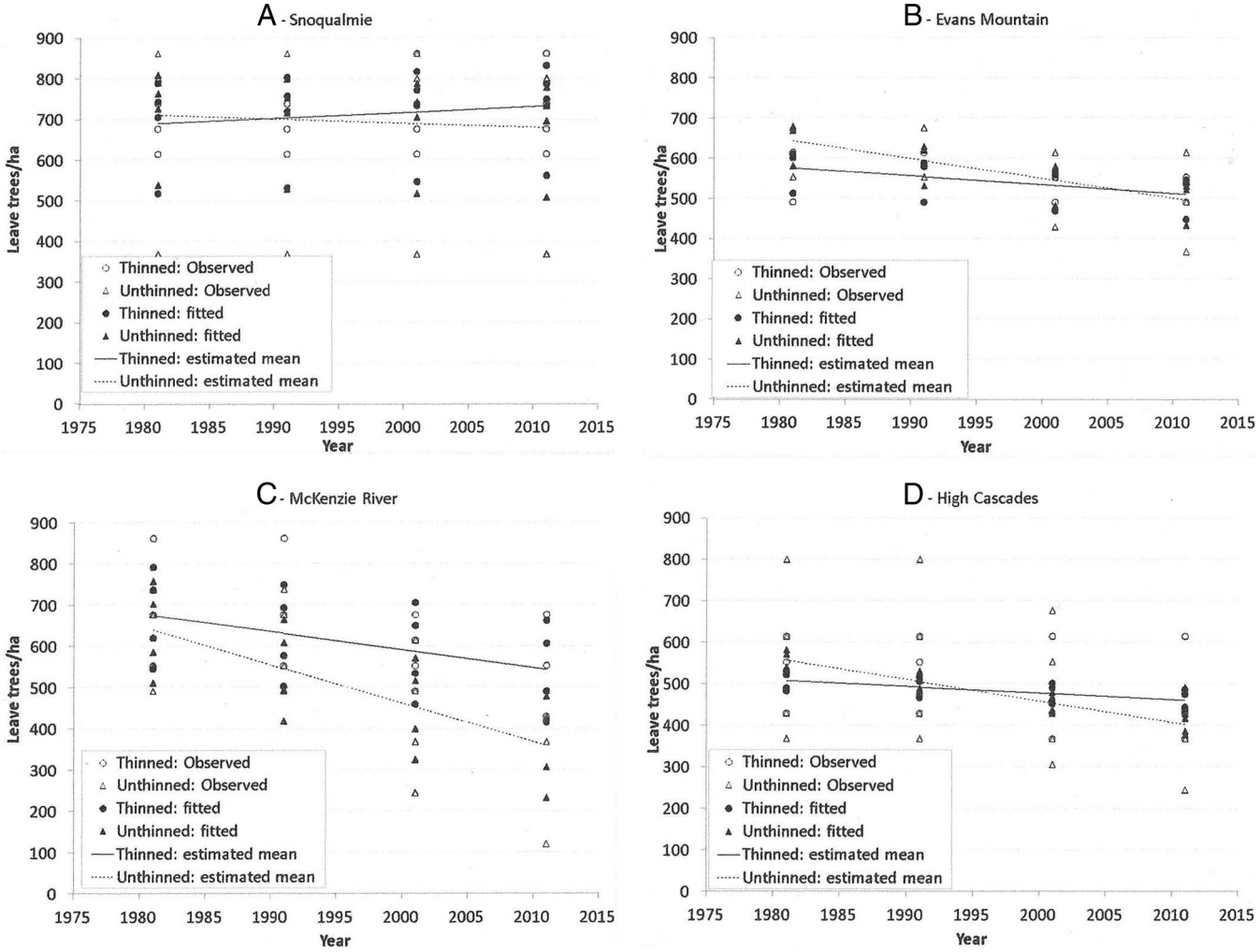

Figure 2. Actual leave-tree survival (TPH) for thinned and unthinned plots at four 10-year periods in mixed-conifer plantations with armillaria root disease at four sites: Snoqualmie (A), Evans Mountain (B), McKenzie River (C), and High Cascades (D).

Table 5. Survival summary from 1981 to 2011 for leave, nonleave, and ingrowth trees in four 0.02-ha circular plots per treatment on four sites.

Tree type	Site	Treatment	Total no. of trees	No. dead	No. alive	% alive
Leave trees	Snoqualmie	Thinned	47	0	47	100
		Unthinned	46	2	44	95.7
	Evans Mountain	Thinned	28	3	25	89.3
		Unthinned	31	7	24	77.4
	McKenzie River	Thinned	43	8	35	81.4
		Unthinned	40	17	23	57.5
	High Cascades	Thinned	33	3	30	90.9
		Unthinned	36	10	26	72.2
Nonleave trees	Snoqualmie	Unthinned	35	5	30	85.7
	Evans Mountain	Unthinned	26	12	14	53.8
	McKenzie River	Unthinned	48	20	28	58.3
	High Cascades	Unthinned	21	3	18	85.7
Ingrowth trees	Snoqualmie	Thinned	38	1	37	97.4
		Unthinned	49	5	44	89.8
	Evans Mountain	Thinned	18	0	8	100
		Unthinned	40	22	18	45
	McKenzie River	Thinned	24	4	20	83.3
		Unthinned	25	4	21	84
	High Cascades	Thinned	18	0	18	100
		Unthinned	157	65	92	58.6

Between 1981 and 2011, nonleave TPH in unthinned plots decreased (Table 5) with some mortality due to *Armillaria* but most due to suppression or other causes. Actual TPH after 30 years were significantly fewer ($P = 0.002$) (Table 6). QMD ($P < 0.0001$) and BA ($P = 0.014$) of nonleave trees, however, increased significantly after 30 years (Table 6).

Between 1991 and 2011, ingrowth TPH increased more for the unthinned plots than for the thinned plots, but differences were not significant ($P = 0.56$) (Table 7). Differences also were not significant for ingrowth QMD ($P = 0.77$) and BA ($P = 0.85$) between thinned and unthinned plots (Table 7).

Douglas-Fir Plantation (McKenzie River)

Most of the young-tree mortality before thinning was caused by *A. ostoyae*; however, in one plot pair, most mortality was caused by black stain root disease. Data from this plot pair were omitted from the analysis. Overstory stumps (mean 151/ha, 62.2 cm diameter) were mostly Douglas-fir with some hemlock. About 14% of the stumps had *H. occidentale*; 9% had *Armillaria* spp. (Table 1).

Thirty years after treatment, the probabilities of leave-tree survival ($P = 0.017$) (Figure 1C) and actual leave-tree survival (TPH) ($P = 0.03$) (Figure 2C) were significantly greater in thinned than in unthinned plots (Tables 4A and 5). Over 30 years, based on the 0.1-ha plots, 6.7 TPH/year (0.8%/ha/year) were killed by *A. ostoyae*

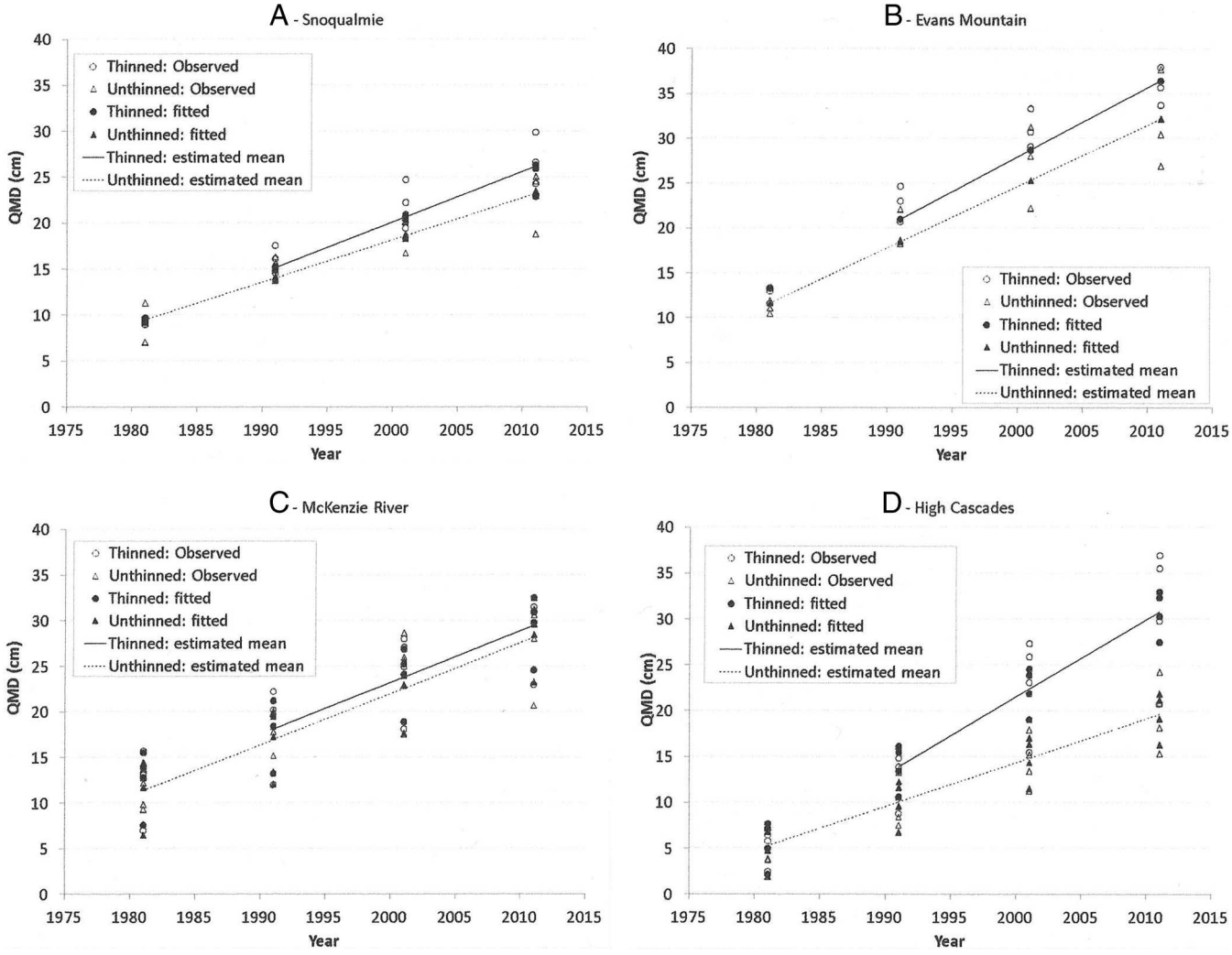

Figure 3. Leave-tree QMD (cm) growth for thinned and unthinned plots at four 10-year periods in mixed-conifer plantations with armillaria root disease at four sites: Snoqualmie (A), Evans Mountain (B), McKenzie River (C), and High Cascades (D).

in thinned plots, and 5.TPH/year (0.8%/ha/year) were killed by *A. ostoyae* in unthinned plots. Almost all of the leave trees killed were Douglas-fir. Over 30 years, about 10 leave TPH were killed by black stain root disease in thinned plots and none in unthinned plots. About 2 leave TPH were killed by windsnap in thinned plots and 44 leave TPH in unthinned plots. Although there were fewer trees killed by root disease (armillaria and black stain) in unthinned plots than in thinned plots (5.9 versus 6.7 TPH/year), mortality from windsnap accounted for the significantly greater leave-tree mortality in unthinned plots.

There was no significant ($P = 0.88$) (Figure 3C) difference in QMD growth of leave trees between thinned and unthinned plots (Table 4B). BA growth of leave trees, however, was significantly ($P = 0.007$) (Figure 4C) higher in thinned plots (Table 4C).

Between 1981 and 2011, nonleave TPH in unthinned plots decreased (Table 5), and 30-year differences were significant ($P = 0.02$) (Table 6). Some tree mortality was due to *Armillaria*, but most was due to suppression or other causes. QMD growth ($P < 0.0001$) and BA growth ($P = 0.02$) increased significantly after 30 years (Table 6).

Between 2001 and 2011, ingrowth TPH decreased, but differences were not significant ($P = 0.93$) between thinned and unthinned plots (Table 7). Differences also were not significant for ingrowth QMD ($P = 0.32$) and BA ($P = 0.88$) between thinned and unthinned plots (Table 7).

Shasta Red Fir/Mountain Hemlock Plantation (High Cascades)

Most of the young-tree mortality before treatment was caused by *A. ostoyae*. Overstory stumps (mean 153/ha, 52.8 cm diameter) were mostly red fir with some mountain hemlock stumps. About 61% of the stumps had *H. occidentale*, 41% had *Armillaria* spp., and many had both pathogens present (Table 1). More overstory stumps in unthinned plots had *H. occidentale*, but more stumps in thinned plots had *Armillaria* spp. No other root diseases were found.

Thirty years after treatment, the probability of leave-tree survival ($P = 0.056$) (Figure 1D) was significant at α level = 0.10 (Table 4A). Actual leave-tree survival, however, was not significantly ($P = 0.11$) different between thinned and unthinned plots (Table 4A; Figure 2D). Over 30 years based on the 0.1-ha plots, 4.2 TPH/year (0.6%/ha/year) were killed by *A. ostoyae* in thinned plots, and 5.4 TPH/year (0.7%/ha/yr) were killed by *A. ostoyae* in unthinned plots. Nearly all of the mortality occurred in red fir, although some white pines also were killed. Western white pine is moderately damaged by armillaria root disease and seldom damaged by heterobasidion root

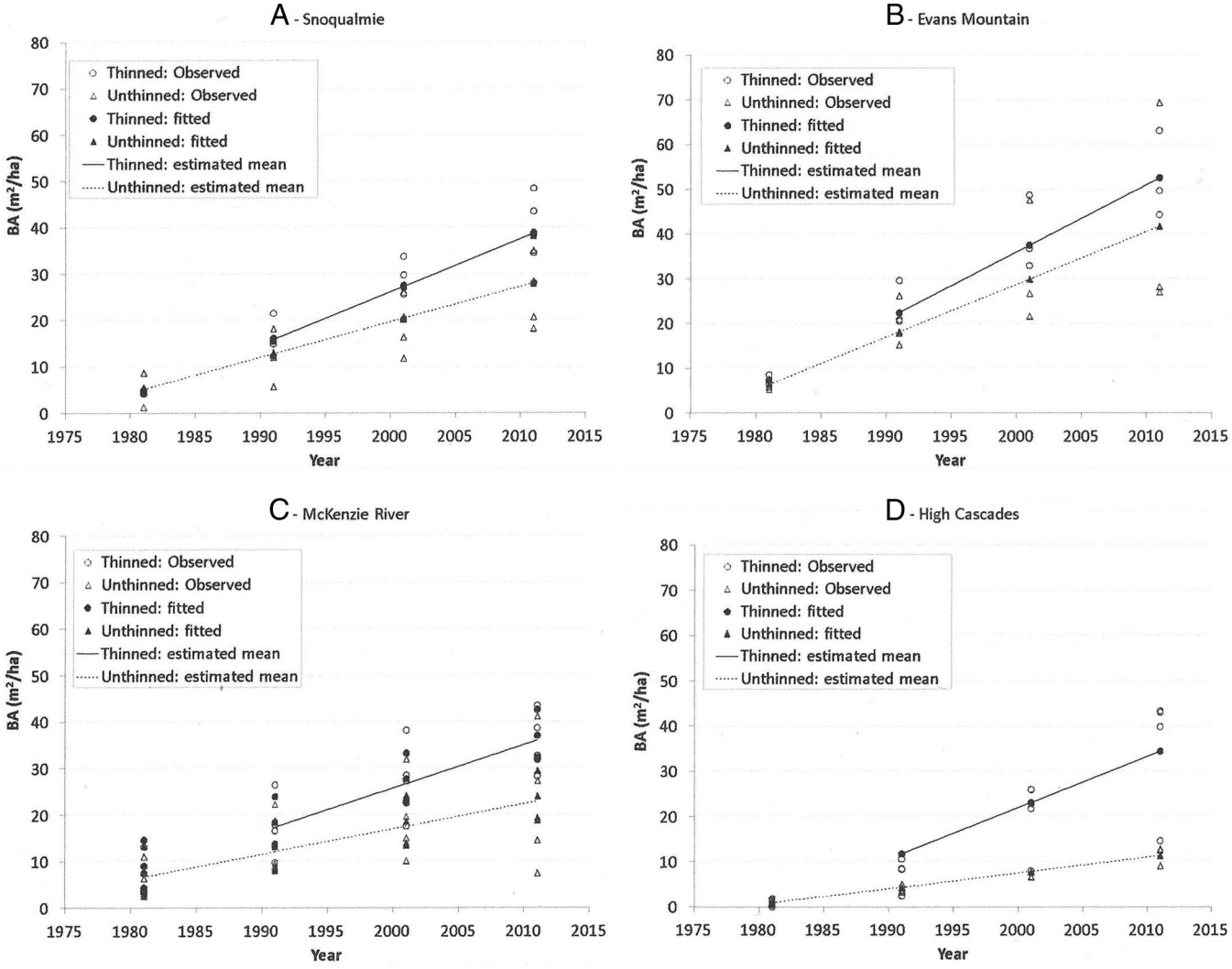

Figure 4. Leave-tree BA (m^2/ha) growth for thinned and unthinned plots at four 10-year periods in mixed-conifer plantations with armillaria root disease at four sites: Snoqualmie (A), Evans Mountain (B), McKenzie River (C), and High Cascades (D).

Table 6. Nonleave-tree comparison for 30-year growth for TPH, QMD, and BA responses.

Site	30-year TPH difference estimate (SE)	TPH difference *P* value	30-year QMD growth estimate (SE) (cm)	QMD difference *P* value	30-year BA growth estimate (SE) (m^2/ha)	BA difference *P* value
Evans Mountain	−266.83 (58.15)	0.002*	13.89 (1.68)	<0.0001*	88.04 (27.95)	0.014*
High Cascades	−46.30 (23.30)	0.07	12.35 (1.32)	<0.0001*	68.18 (10.89)	<0.0001*
McKenzie River	−309.08 (110.72)	0.02*	10.98 (1.23)	<0.0001*	99.35 (36.58)	0.02*
Snoqualmie	−77.21 (51.54)	0.16	11.51 (0.60)	<0.0001*	100.19 (8.74)	<0.0001*

Negative estimates indicate that the value of the response in 1981 was greater than in 2011.
* Statistically significant at α level = 0.05.

Table 7. Ingrowth tree comparison of thinned versus unthinned treatments for the 2011 TPH, QMD, and BA responses.

Site	TPH 2011 treatment difference estimate (SE)	TPH difference *P* value	QMD 2011 treatment difference estimate (SE) (cm)	QMD difference *P* value	BA 2011 treatment difference estimate (SE) (m^2/ha)	BA difference *P* value
Evans Mountain	−60.42 (93.94)	0.56	0.29 (0.95)	0.77	0.72 (27.52)	0.85
High Cascades	−500 (173.59)	0.03*	−1.50 (0.24)	<0.0001*	−42.25 (13.62)	0.01*
McKenzie River	−6.25 (69.78)	0.93	−0.36 (0.34)	0.32	−0.39 (2.68)	0.88
Snoqualmie	−56.25 (105.99)	0.62	−0.02 (0.66)	0.98	5.02 (7.59)	0.52

Positive estimates indicate that the value of the response in thinned plots is greater than in unthinned plots.
* Statistically significant at α level = 0.05.

disease (Goheen and Willhite 2006). Most of the mortality in red fir and white pine was caused by *A. ostoyae*. Three white pines were killed by white pine blister rust (*Cronartium ribicola* J.C. Fisch.). Surprisingly, no mortality of mountain hemlock caused by *H. occidentale* was observed for 30 years after thinning despite the large number of infected stumps from the former overstory (Table 1).

Mountain hemlock is severely damaged by *H. occidentale* (Goheen and Willhite 2006).

Leave trees in thinned plots had significantly ($P < 0.0001$) greater QMD growth than did leave trees in unthinned plots (Figure 3D; Table 4B). In addition, BA growth of leave trees was significantly ($P < 0.0001$) higher in thinned plots (Figure 4D; Table 4C).

Between 1981 and 2011, nonleave TPH in unthinned plots decreased (Table 5), and differences were significant ($P = 0.07$) at α level = 0.10 (Table 6). Some mortality was due to armillaria root disease, but most was due to suppression or other causes. Nonleave tree QMD and BA growth ($P < 0.0001$) increased significantly after 30 years (Table 6).

Between 1991 and 2011 ingrowth TPH increased with significantly ($P = 0.03$) more trees in unthinned plots (Table 7). Ingrowth QMD ($P < 0.0001$) and BA ($P = 0.01$) were significantly higher in unthinned plots than in thinned plots (Table 7).

Discussion and Conclusions

Thirty years after treatment, there was a significant increase in leave-tree survival in thinned plots versus unthinned plots in one of four plantations with no significant difference in survival in the other three plantations. Mortality over the 30-year period remained constant or decreased in all plantations; most trees grew large enough so that crown closure has reduced gaps where single trees died. Gaps caused by multiple-tree mortality are still evident.

More overstory stumps had detectable decay caused by *H. occidentale* than by *Armillaria* spp. in all plantations before thinning (Table 1). Despite this, only one plantation (Snoqualmie) had leave-tree mortality (two Douglas-firs) caused by *H. occidentale* after 30 years, including the highly susceptible mountain hemlock/Shasta red fir plantation (High Cascades) where no *Heterobasidion*-caused tree mortality occurred. We do not know, however, the extent of possible *Heterobasidion*-caused stem decay in live but wounded trees, which often is a more common expression of disease than tree mortality (Filip et al. 1992, 2006).

Stumps decayed by *H. occidentale,* however, usually are also infected by *Armillaria* spp. in the outer portions of the stump (Morrison and Johnson 1978, Filip et al. 1992), which may inhibit the spread of *H. occidentale* across root systems. Conversely, colonization of stump centers by *H. occidentale* could reduce the spread of *Armillaria* spp. to the stump center and thus reduce the total amount of *Armillaria* spp. inoculum, although some of this inoculum may be of saprophytic or nonpathogenic species. In nearly all cases, leave-tree mortality after 30 years was greater in plots where initial mortality was greater, regardless of whether plots were thinned. Blenis (2000) made the same observation in lodgepole pine infected with *A. ostoyae* in central Alberta.

Ten years after thinning, trees in thinned plots showed a tendency toward faster QMD growth than trees in unthinned plots (Filip and Goheen 1995). Twenty years after thinning, we found that differences in QMD growth were statistically significant between thinned and unthinned plots across all plots and sites (Filip and Ganio 2004). Rosso and Hansen (1998) also found significant differences in radial growth of Douglas-firs between thinned and unthinned plots infected by *A. ostoyae* in West-Central Oregon. Although diameter growth was significantly improved by thinning after 30 years, root infections on residual trees probably are causing growth loss as was found by Cruickshank et al. (1997) in coastal British Columbia.

In western Cascades mixed-conifer plantations, it appears that, after 30 years, precommercial thinning of Douglas-fir, western hemlock, mountain hemlock, noble fir, and Shasta red fir does not significantly increase mortality caused by armillaria or heterobasidion root diseases. Therefore, the decision to precommercially thin may be based on the costs of thinning compared with the benefits of increased leave-tree growth from reduced stand density. Similar recommendations may not be appropriate in stands east of the Cascade crest of interior Douglas-fir, hemlock, and true fir, because differences within tree and fungal genetics and in site characteristics usually are expressed as much more severe tree mortality in interior forests. For example, in interior British Columbia, Cruickshank et al. (1997) found that the limited ability of most interior Douglas-firs to produce callus at *Armillaria* infections was probably related to their much slower juvenile growth than that of coastal Douglas-fir.

The results of our study, which investigated thinning effects in *Armillaria*- and *Heterobasidion*-infected plantations of small diameter trees (5–13 cm dbh), may not be applicable to thinning in stands with larger trees. Felling larger trees leaves larger stumps that provide more potent and persistent inoculum sources for root disease fungi, a caution also expressed by Blenis (2000) in central Alberta. Although no results have been reported concerning the effects of thinning of *Armillaria*-infected stands of trees of >13 cm dbh in western Oregon or Washington, thinning of large (30–60 cm dbh) white and Shasta red firs showed no significant increases in leave-tree survival or diameter growth after 10 years in South-Central Oregon (Filip et al. 2010). In southern interior British Columbia, Morrison et al. (2001) found that the percentage of merchantable volume of mixed-conifers killed by *A. ostoyae* was usually higher in selectively harvested plots of mature trees than in undisturbed plots. Similar results have been reported in large-diameter ponderosa pines in South-Central Washington (Shaw et al. 1976, 2012, Roth et al. 1980) and in mixed-conifer stands in northern Idaho (McDonald et al. 1987).

Although precommercial thinning does not appear to increase mortality caused by armillaria or heterobasidion root diseases in western Cascade plantations, other root diseases often are associated with precommercial thinning and should be considered when plantations are treated. Black stain root disease kills Douglas-fir or frequently predisposes trees to infection by *Armillaria* spp. (Goheen and Hansen 1978, Hessburg et al. 2001) and often is associated with precommercially thinned stands of Douglas-fir (Harrington et al. 1983). Laminated root rot caused by *Phellinus sulfurascens* (formerly *Phellinus weirii*) is the most damaging root disease in western Oregon and Washington (Hadfield et al. 1986, Shaw et al. 2009). Young stands of Douglas-fir, hemlock, or true fir with numerous and widely distributed pockets of laminated root rot should not be precommercially thinned unless sufficient numbers of low-susceptibility tree species (pine, cedar, or hardwoods) can be favored as leave trees (Thies and Sturrock 1995, Shaw et al. 2009).

Recommendations

We recommend that young stands or plantations of mixed-species conifers, similar to those we studied in the western Cascades of Oregon and Washington, be thinned to meet management objectives, even if such stands are affected by armillaria or heterobasidion root diseases. Thinning does not appear to significantly increase leave-tree mortality caused by *A. ostoyae* or *H. occidentale*, and most residual trees show significant increases in QMD and BA growth 30

years after thinning. In the western Cascades of Oregon and Washington in young, *Armillaria*- or *Heterobasidion*-infected stands of white fir, grand fir, and/or Pacific silver fir, we do not recommend thinning but instead consider planting or favoring root disease-tolerant species such as coastal Douglas-fir, hemlock, cedar, and/or pine after thoroughly evaluating root-disease type and occurrence, and local tree species suitability and susceptibility (Hadfield et al. 1986, Goheen and Willhite 2006, Shaw et al. 2009, Filip et al. 2010, 2015).

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