United States
Department of
Agriculture

Forest Service

Pacific Northwest
Research Station

Research Paper
PNW-RP-569
November 2006

Laminated Root Rot in a Western Washington Plantation: 8-Year Mortality and Growth of Douglas-Fir as Related to Infected Stumps, Tree Density, and Fertilization

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Cover photo

Effects of tree mortality from laminated root rot and other causes are evident in this landscape view of the 12-year-old Douglas-fir plantation at Voight Creek.
All Photos by Tim Harrington

Abstract

Miller, Richard E.; Harrington, Timothy B.; Thies, Walter G.; Madsen, Jeff. 2006. Laminated root rot in a western Washington plantation: 8-year mortality and growth of Douglas-fir as related to infected stumps, tree density, and fertilization. Res. Pap. PNW-RP-569. Portland, OR: U.S. Department of Agriculture, Forest Service, Pacific Northwest Research Station. 37 p.

A 4-year-old Douglas-fir plantation in the western Washington Cascades was monitored for 8 years after fertilization with potassium (K), nitrogen (N), and K+N to determine fertilizer effects on rates of mortality from laminated root rot (LRR) and other causes relative to a nonfertilized control. Each element was applied at a rate of 300 lb/acre on and around 0.2-acre plots replicated seven times in a randomized complete block design. Cumulative mortality from LRR did not differ significantly among fertilizer treatments, and losses were strongly related to density of infected stumps from the previous stand ($r^2 = 0.74$). Mortality from disease and other sources accelerated during the 8 years of monitoring. Average tree growth and stand volume were greatest in treatment N and were reduced where N was combined with K. Continued monitoring is needed to identify potential longer term effects of the fertilizer treatments on susceptibility of Douglas-fir to LRR and *Armillaria* spp.

Summary

Laminated root rot (LRR) is the most damaging disease of young-growth Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) in the Pacific Northwest. Laminated root rot is caused by the fungus, *Phellinus weirii* (Murr.) Gilb., which survives for 50 years or more in roots after infected Douglas-fir are harvested. Successive stands can be infected as new roots contact infected wood from the previous stand. Without removing stumps, changing species, or increasing spacing among potential host trees, progressively more diseased area can develop in successive rotations. Some research suggests that application of nitrogen (N) fertilizer may increase incidence of some tree diseases and pest insects, but that application of potassium (K) fertilizer may reduce disease impacts.

Our study, started in a 4-year-old coast Douglas-fir plantation, is designed to compare the effects of fertilization with N and K (alone or in combination) on tree mortality and growth. The study site is located 7 mi east of Orting, Washington. Soils include one type derived from recessional outwash sand and two types from nonsorted basal moraine from the last continental glacier. The previous 70-year-old

Douglas-fir stand was harvested in 1993. Density and size of *P. weirii*-infected stumps from that stand were evaluated on each of 28, 0.2-acre plots and a surrounding 10-ft-wide buffer, and the plots were then grouped into seven blocks of four plots each according to their potential for *P. weirii* infection, estimated as inoculum index (I.I.). One of the following four treatments was randomly assigned to one plot per block: 300 lb elemental K/acre (treatment K, applied as potassium chloride), 300 lb elemental N/acre (treatment N, applied as urea), 300 lb K/acre plus 300 lb N/acre (treatment K+N), and a nonfertilized control. Fertilizer treatments were applied in March 1998. The experimental design is a randomized complete block with four fertilizer treatments replicated among the seven blocks. Tree mortality from LRR and other sources was monitored annually for 8 years, and tree size and growth were measured in the first and sixth years after fertilization.

Inoculum index did not differ significantly among soil types, despite their differences in slope percentage and gravel-cobble content. Among the 18 plots with infected stumps, cumulative 8-year mortality from LRR had a positive linear relationship with both I.I. ($r^2 = 0.53$) and density of LRR-infected stumps per acre ($r^2 = 0.74$). Cumulative tree mortality from LRR or *Armillaria* spp. did not differ among fertilizer treatments in the 8 years of monitoring. Cumulative mortality from other causes, however, averaged 15 trees per acre (TPA) greater in treatment K+N than in treatment N. We suspect excess of potassium or chloride ions in the soil solution may be responsible.

Relations among mean 100-needle weight and N and K concentration and content of foliage collected 6 months after fertilization were examined through graphical vector analysis. From this we infer that N fertilization offset growth-limiting N-status at this site and that K-fertilization resulted in excess K and toxic effects. This suspected toxicity may explain why 10-year-old trees in K-fertilized plots are shorter than those in nonfertilized plots. Conversely, trees in N-fertilized plots are taller and have greater stand basal areas and volumes than those in nonfertilized plots. Although an average 62 TPA died by the 12th year after planting, plantation stocking remained satisfactory at an average density of 390 TPA and did not differ among the fertilizer treatments. Longer term observations are needed to assess the future continued effects of fertilization on tree mortality and growth.

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Introduction

The fungus, *Phellinus weirii* (Murr.) Gilb., causes laminated root rot (LRR), which is the most damaging disease of young-growth Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) in the Pacific Northwest (Childs and Shea 1967). A survey of nonfederal forest land in northwestern Oregon found LRR to occur on 12 percent of the land (Gedney 1981). Goheen and Hansen (1993) later estimated that LRR occurs on 5 percent of the area occupied by Douglas-fir and losses approach 50 percent on the affected areas. Because the fungus survives for 50 or more years in infected roots and stumps of harvested trees (Childs 1963, Hansen 1976), successive stands of Douglas-fir can be infected as new roots contact infected wood from the previous stand.

Successive stands of Douglas-fir can be infected as new roots contact infected wood from the previous stand.

On sandy soils and gentle slopes, push-falling or stump removal is recommended to isolate infected stumps and trees (Thies and Sturrock 1995). Their other mitigative prescriptions include planting or retaining *P. weirii*-resistant conifers, e.g., western redcedar (*Thuja plicata* Donn. ex D. Don) or ponderosa pine (*Pinus ponderosa* Dougl. ex Laws. var. *ponderosa*) or immune species (hardwoods). To minimize root contact between infected and healthy Douglas-fir trees, Thies and Sturrock (1995) recommended spacing Douglas-fir at least 13 ft apart (about 260 trees/acre) when precommercial thinning on deep, well-drained soils. "This strategy is less likely to be successful where soil conditions encourage a shallow, spreading root habit" (p. 25). Without mitigative measures (such as removing stumps, changing species, or increasing spacing between highly susceptible Douglas-fir), *P. weirii* in residual stumps and roots will infect trees in the replacement stand and result in progressively more diseased area in successive rotations (Tkacz and Hansen 1982).

Potential Effects of Fertilization

Nitrogen-fertilization in interior forests—
Fertilizer trials east of the Cascade crest suggest that application of nitrogen (N) may increase incidence of some tree diseases and pest insects, but that application of potassium (K) may reduce disease.

- In the initial fertilizer trials, weak response of Rocky Mountain Douglas-fir (*Pseudotsuga menziesii* var. *glauca* (Beissn.) Franco) stands to N fertilization was related to low concentration of K in foliage. Specifically, in trials on soils derived from granitic and metamorphic parent materials, amount and duration of growth response to N was small, and the 400 lb N/acre dosage produced much less response than the 200 lb dosage because

***Armillaria* root disease, like *P. weirii*, spreads primarily by root contact.**

mortality was greater (Mika and Moore 1990). Of particular concern is mortality from *Armillaria* root disease (*Armillaria ostoyae* (Romagnesi) Herink*)* that, like *P. weirii*, spreads primarily by root contact.

- Rate of Douglas-fir root infection by *A. ostoyae* is correlated closely with the ratio of phenolic compounds to sugar compounds in roots. Ratios less than about 15 favor *Armillaria* spp. infection in Rocky Mountain Douglas-fir (Entry and others 1991). Phenolics are considered plant-defense compounds, but sugar is food for *Armillaria* (Wargo 1980). Trees fertilized with N (200 lb N/acre) exhibited a lower ratio as a result of decreasing concentration of phenolics (Entry and others 1991). This is consistent with Shigo (1973) who claimed that nitrogenous fertilizers reduce production of phenols in plant tissue, thereby lowering resistance to infection by pathogenic fungi.
- Large dosage of K (with either small or large dosage of N fertilizer) increases the phenolic/sugar ratio and also tannin concentrations in roots of potted seedlings of interior Douglas-fir (Shaw and others 1998). Form of K in the nutrient solutions was not specified.

Potassium fertilization and agricultural experience—
Research results in east-side forests are consistent with general relationships between K and agricultural plant health (Perrenoud 1990). Generally K tends to improve plant health, relative to fungal and bacterial diseases.

Nitrogen fertilization in coast Douglas-fir stands—
Knowing the consequences of N fertilizer for susceptibility of Douglas-fir to *P. weirii* is critical because N fertilizer has been conventionally used in recent decades to improve growth of coast Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*). The usual prescription of 200 lb N/acre as urea increases growth in established stands in about 70 percent of field trials in western Oregon, Washington, and British Columbia (Chappell and others 1992). Doubling this dosage to 400 lb N/acre provides additional increases in stand growth, in part, because duration of response is extended (Miller and others 1986). Although rates of Douglas-fir mortality in established stands often are accelerated by N fertilization, this has been attributed to accelerated growth that intensifies tree-to-tree competition and suppression.

Increased tree mortality from LRR after N-fertilization of established coast Douglas-fir stands has not been reported, possibly because potential study areas with

symptoms of LRR are avoided and trials with high incidence of damage or disease are usually abandoned as atypical and of random occurrence. Nor has N fertilization reduced LRR-losses in established stands. The 2-year results after localized fertilization with 400, 1,000, or 2,000 lb N/acre of individual Douglas-fir in a 26-year-old plantation at Shawnigan Lake, British Columbia, were reported by Wallis and Reynolds (1974). These large dosages of N as urea failed to cause a visible degradation of *P. weirii* mycelium growing on the bark in vivo. All cultures from infected roots were viable. After fertilization, infected trees averaged about half as much basal area growth as noninfected trees, but a higher percentage of fertilized trees died. We surmise that the 1,000 and 2,000 lb N/acre-dosages may have created toxic effects from pH changes (Otchere-Boateng and Ballard 1978) associated with a high concentration of ions in the soil solution.

A series of investigations in recent decades tested N fertilization of newly planted trees to reduce mortality of coast Douglas-fir from LRR. Having documented lower incidence of Douglas-fir losses where this species was associated with red alder (*Alnus rubra* Bong.), and where total N and nitrate-N concentrations are characteristically greater, Nelson (1970) reported that survival of LRR was reduced when *P. weirii*-infected wood was buried in N-enhanced soil. Subsequently, large dosages of ammonium nitrate fertilizer were combined with stump removal at five test locations in Oregon and Washington. Although growth of planted Douglas-fir at some locations was enhanced by N fertilization (0, 300, 600, 1,200 lb N/acre), losses from LRR were not affected during the 23 to 27 years after fertilization (Thies and Westlind 2005). Losses from non-LRR agents were not reported, including possible losses after large doses of a highly soluble N fertilizer.

Additional research information is needed to identify the effects of N and K fertilization on susceptibility of Douglas-fir trees to *P. weirii* and other diseases. Such research should attempt to attribute mortality to specific agents and to adjust for effects of local inoculum potential and tree density. In designing our study, we speculated that above-average numbers of (1) *Pw*-infected stumps or (2) planted and volunteer Douglas-fir seedlings could increase seedling and sapling losses from LRR. We assumed that a high density of regenerating seedlings could increase the likelihood of root contact with infected legacy wood and infected seedling roots. We further assumed that seedling and sapling density (trees/acre, [TPA]) will also affect mean tree size and stand yield per acre (basal area or volume). Thus, to isolate the effects of fertilizer treatment, we needed to recognize, measure, and adjust for these potentially confounding or masking influences.

Objectives

Our study in a young coast Douglas-fir plantation is designed to compare the effects of fertilization with K and N (alone or in combination) on tree mortality and tree growth. Based on a literature review, we posed several working hypotheses:

1. Severity of LRR (i.e., concentration of inoculum) in a mature Douglas-fir stand is a good predictor of incidence of LRR-caused mortality in the succeeding stand.

2. Addition of 300 lb K/acre as KCl, either alone or combined with 300 lb N/acre, can reduce incidence of LRR-caused mortality.

3. Addition of 300 lb N/acre as urea can increase incidence of LRR-caused mortality.

4. Fertilization can increase tree and stand growth.

Methods

Plots were installed in a 4-year-old plantation of coast Douglas-fir. Before fertilizers were applied, each plot was evaluated for its potential to develop LRR, arrayed in order of that potential, and then separated into seven LRR inoculum concentration groups of four plots each. Different fertilizer treatments were randomly assigned to three plots in each group; the fourth plot remained nonfertilized. Tree mortality was monitored annually for 8 years; tree size was measured before fertilization and in the first and sixth years after fertilization.

The Experimental Area

Location–
Our study area is located 7 mi east of Orting, Washington. Plots were installed in a 4-year-old plantation (1994) managed by the Campbell Group, LLC. Nearly half of our study plots are located on the former Voight Creek experimental forest; the remaining plots were located in the same plantation that extends beyond the original experimental forest boundary. The USDA Forest Service's Pacific Northwest (PNW) Forest and Range Experiment Station (currently the PNW Research Station) conducted silvicultural research on the experimental forest between 1947 and 1981. Elevations range from 830 to 1,140 ft, and annual precipitation averages about 49 in with about 16 in falling during the growing season (April through September). Snowfall is infrequent.

Soils–

Soils supporting the test plantation were derived from either outwash deposits (stratified sand and gravel) or nonsorted basal moraine, both deposited by the last continental glacier, the Vashon Stade of the Fraser glaciation (fig. 1). Soils were originally mapped when the experimental forest was established and remapped in greater detail as a cooperative project with the Soil Conservation Service (currently the Natural Resource Conservation Service). Although site index (50-year base) averaged about 117 ft in the experimental forest, our study area is located on the lower - quality sands and gravels. Specific soil types are Barneston very gravelly sandy loam, either on (1) 6- to 30-percent slopes[1] or (2) 30- to 60-percent slopes and (3) Nargar sandy loam, 6- to 30-percent slopes. Most plots occupied only one of these soil types (table 1).

Although mean potassium concentration in tree foliage was estimated on some plots, the K-nutrient status of these soils was not determined before or after fertilization. Weathering of primary feldspar can provide K for cation-exchange sites, but these immature coarse-textured soils have relatively small content of clay for

Figure 1—The Barneston soil series at Voight Creek, Washington, is formed on unconsolidated glacial deposits (top several feet of this profile). Note the underlying stratified outwash sand that is the parent material of soil type 3 (Nargar series).

[1] Zulauf, A. S. 1982. Soil survey of Voight Creek Experimental Forest. Unpublished report On file with: Forestry Sciences Laboratory, 3625 93rd Avenue SW, Olympia, WA 98512.

Table 1—Plot information and treatment by block at Voight Creek, Washington, ordered within blocks by number of initially tagged trees

Block	Plot[a]	Soil[b]	Inoc. Index	Tagged		Cut	All	Fert.[d]	Block	Plot[a]	Soil[b]	Inoc. Index	Tagged		Cut	All	Fert.[d]
				Douglas-fir[c]	Other								Douglas-fir[c]	Other			
				– – – – – Trees per acre – – – – –									*– – – – – Trees per acre – – – – –*				
1	213	3	0	380	5	355	740	K	5	208	3	228	360	5	420	785	0
1	218	3	0	370	0	345	715	0	5	70*	3	475	380	10	320	710	K+N
1	15*	2	0	410	0	130	540	N	5	209	3	214	430	5	855	1,290	K
1	17*	2	0	470	0	290	760	K+N	5	19*	2/3	495	495	0	815	1,310	N
	Mean		0	408	1	280	689			Mean		353	416	5	603	1,024	
2	217	3	0	460	5	735	1,200	N	6	129*	3	1,168	415	0	425	840	N
2	111*	1	0	465	0	625	1,090	K	6	215	3	926	465	0	680	1,145	0
2	206	2	0	470	15	865	1,350	K+N	6	27*	2	956	380	0	35	415	K+N
2	207	2	0	505	5	1,450	1,960	0	6	204	1/3	310	425	0	1,500	1,925	K
	Mean		0	475	6	919	1,400			Mean		840	421	0	660	1,081	
3	4*	2	0	520	10	775	1,305	0	7	29*	2	1,951	445	5	630	1,080	0
3	201	2	0	540	0	695	1,235	N	7	205	1	894	415	0	540	955	K
3	202	1	0	570	15	95	680	K	7	221	2	1,389	435	0	810	1,245	N
3	8*	3/1	0	590	5	1,545	2,140	K+N	7	40*	2	2,649	450	0	195	645	K+N
	Mean		0	555	8	778	1,340			Mean		1,721	436	1	544	981	
4	10*	3/1	41	430	0	1,065	1,495	K									
4	203	3	92	440	5	900	1,345	0									
4	211	2	162	445	5	645	1,095	K+N									
4	216	3	282	480	0	545	1,025	N									
	Mean		144	449	3	789	1,240										

[a] Plots with asterisks correspond to those installed in 1947-48 within the former Voight Creek Experimental Forest. The 200-series of plots were newly installed.

[b] Soil numbers correspond to soil types identified in text; a few plots were located predominately on one soil type and a lesser portion on a second soil type: example 3/1.

[c] After the first growing season the initial number of tagged trees changed slightly to reduce within-block differences in trees per acre.

[d] 0 = no fertilizer.

cation-exchange. We assume that K is readily leached from these coarse-textured, clay-poor soils and that relatively small amounts of K are available to plants. Consequently, we applied a relatively heavy dosage of K and fertilized in spring to reduce leaching.

Plot Installation

Plot design consists of 0.20-acre (0.08-ha) circular tree-measurement plots centered within 0.50-acre (0.20-ha) circular treatment plots. Plot centers were spaced at least 180 ft apart to ensure that measurement trees were not affected by the treatment of adjacent plots. Of the 28 plots, 12 corresponded to original, mechanically spaced plots in a thinning study at the Voight Creek Experimental Forest. The remaining 16 plots were also systematically located, but with some adjustment to ensure that each plot sampled uniform topography and soil.

The Laminated Root Rot Survey to Determine Inoculum Index

The previous 70-year-old Douglas-fir stand on the study site was harvested in 1993. Stumps located within 62.7 ft of tree measurement plot centers (i.e., 52.7-ft plot radius plus an additional 10 ft) were examined in summer 1997 for the stain typical of laminated root rot (Thies and Sturrock 1995) (fig. 2). To facilitate examination for LRR, stumps were cut near ground level on the uphill side. To assure that inoculum remained undisturbed, we avoided excavation. Stain on fresh cut stumps appears in the outer heartwood and is above the point of attachment of major infected roots (Thies and Sturrock 1995). The stain appears as spots or broad bands. We assumed that the total root biomass that was infected was proportional to the total stump circumference showing typical stain.

Stumps with LRR were identified, and their diameter and percentage of stump circumference infected (CI) were recorded. Locations of infected stumps were mapped to enable future spatial analysis of the pattern of mortality in the new plantation. We assumed that the amount of inoculum was proportional to the infected biomass of the stump and belowground roots. A regression equation was used to estimate belowground biomass of each stump (Thies and Cunningham 1996):

$$\ln(LRB) = -4.38 + 2.41 \ln(DSH)$$

where ln(*LRB*) is the natural logarithm of large-root biomass (kg of roots 1.0 cm [0.4 in] and larger in diameter), and *DSH* is the diameter at stump height (in cm

Figure 2—Stump of tree from previous 70-year-old Douglas-fir stand showing the stain and decomposed woody tissue associated with laminated root rot.

measured 15 cm [5.9 in] above mean ground level). Note that the preceding regression was based on trees found in Oregon; no other data are available. For each stump, an index of the amount of inoculum present was calculated as:

Inoculum Index (I.I.) = *LRB* x percent *CI*/1,000 .

Summing the I.I. of each infected stump on a plot provided the plot I.I. Additionally, assuming that some roots from trees near the measurement plot extended into the plot, we added 25 percent of the estimated infected biomass of stumps located within 10 ft of measurement plot boundaries.

Tree Tagging and Density Adjustments

Tree tagging–

Potential crop trees were identified with numbered tags; nearly all were planted Douglas-fir. Tags were wired to a major branch, then eventually moved and tethered at breast height (4.5 ft above ground). Tree numbering started north of the plot center and proceeded in clockwise direction within approximate 40° sectors. Number of Douglas-fir tagged at plot establishment ranged from 360 to 590 TPA (table 1). Additionally, 0 to 15 TPA of western redcedar or western hemlock *Tsuga*

heterophylla (Raf.) Sarg.) were tagged. Tagged crop trees were considered the starting stand for inventorying future losses from LRR or other causes.

Density adjustment–
Although originally planted at an average density of about 430 TPA, plots in the 4-year-old plantation differed greatly in numbers of volunteers (table 1); nearly all were Douglas-fir. Initially (1997), total TPA among the 28 plots ranged from 415 to 2,140 per acre. Of these TPA, 8 percent (plot 27) to 74 percent (plot 207) were cut to reduce variability in TPA among plots. We assumed that future tree mortality from LRR would be proportional to stand density, and that large differences in stand density also would influence tree growth. Consequently, to control Douglas-fir density within preset limits, we removed excess conifers and all N-fixing red alder from each plot and the surrounding 20-ft buffer in four stages:

1. At plot establishment (summer 1997), we tallied and then pulled or cut any tree that was within 5 ft of a larger, presumably planted tree likely to be retained as a crop tree.

2. After tagging potential crop trees and assigning plots to experimental blocks (four plots each), we further reduced large differences in TPA among plots in each block, either by cutting surplus trees on most plots or transplanting seedlings into gaps in a few plots to attain at least a 15-ft spacing between trees. Transplanted trees were tagged and tallied as crop trees.

3. After the first growing season (following fertilization in March 1998), we again adjusted stocking by cutting a few more surplus trees in some plots.

4. After the sixth growing season (Fall 2003) after fertilization, we made a final adjustment of tree numbers to limit within-block differences to <10 percent of block means. We also cut some tagged trees on some plots.

Fertilizer Treatments and Treatment Assignment

The four fertilizer treatments are: (1) K—300 lb elemental K/acre; (2) N-300 lb elemental N/acre; (3) K+N—300 lb K/acre plus 300 lb N/acre; (4) 0–nonfertilized control. These fertilizer treatments were assigned randomly within each block of four plots. This was accomplished by arraying the 28 plots in order of increasing I.I., then separating this array into seven groups (blocks) of four plots each. This initial sorting ensures that each fertilizer treatment sampled the full range of I.I. present in these 28 plots (table 1). Because of incorrect assignment of I.I. to two

plots and incorrect application of fertilizer to one plot, final treatment assignment differed from initial planned assignment. This accidental mix-up changed the order of I.I.

Fertilization

Agricultural-grade potassium chloride (50 percent K) and granular urea (46 percent N) were applied in March 1998 to designated plots. Each 0.5-acre treatment plot was divided into 10 equal sectors, and 10 percent of the fertilizer for that plot was spread evenly on each sector. Weather was overcast, cool, and often drizzling during and for several days after application; volatilization of urea was unlikely.

Tree Measurement and Laminated Root Rot Status

At plot establishment, trees recorded as dead from LRR probably died in the previous 2 years (in the third and fourth growing seasons after planting). Hence, this initial count of LRR-killed trees (after the fourth growing season) may have missed some mortality from the first two seasons after planting. After fertilization, tagged trees were observed annually (1998 through 2005) for mortality, and losses from *Armillaria* spp. were noted. We did not attempt to identify which of the several possible species of *Armillaria* were involved. We recorded azimuth and distance from plot center to each tree killed by LRR. Forest pathologists from PNW Research Station and Washington State Department of Natural Resources assisted in the identification of the mortality agents for dead trees (fig. 3).

Tree heights were measured after the 1998 and 2003 growing seasons (years 1 and 6 after fertilization). In year 1, height of all tagged trees was measured but in year 6, only 30 randomly selected trees per plot were measured. Prefertilization height (1997) was measured concurrently in 1998 by measuring to the whorl of branches initiated after the 1997 growing season. Tree diameter at breast height (d.b.h. or 4.5 ft above ground) was measured after the 2003 growing season (year 6 after fertilization). Height was measured to the nearest 0.1 ft with a telescopic pole or Haglof vertex hypsometer[2] to nearest 0.3 ft, and d.b.h. was measured to nearest 0.05 in with a diameter tape. Heights of nonmeasured trees were estimated by a height-diameter regression equation developed for each plot and based on measured d.b.h. Stand volume was calculated for each plot by entering tree height and d.b.h. data into a general volume equation (Bruce and DeMars 1974). Calculations of

[2] The use of trade or firm names in this publication is for reader information and does not imply endorsement by the U.S. Department of Agriculture of any product or service.

Figure 3—Each dead seedling was partially excavated to identify the cause of mortality.

mean stand characteristics were based on trees surviving through 2003. Trees identified as missing in 2003 were eliminated from all calculations because the cause of mortality could not be determined.

Analysis of Foliage Collected After Year 1

Foliage samples were collected in fall after the 1998 growing season (6 months after fertilization). Three of the seven plots representing each of the fertilization treatments were selected at random to ensure sampling the entire study area.

Two current-year twigs from the second whorl of branches were clipped from 14 systematically selected trees per plot. On the following day, the 28 twigs from each plot were composited and dried for about 12 hours in a forced-draft oven at 70 °C (158 °F). Buds were separated from the twigs before drying. The dried needles were easily stripped from the twigs and were thoroughly mixed before quartering to reduce sample volume; surplus needles were reserved. The reduced sample was again quartered and opposite quarters placed in separate plastic bags labeled subsample A or B for each plot. From each bag, 100 whole needles were selected, redried at 105 °C (221 °F), and weighed after cooling. This provided two samples of 100-needle weight for each plot. Remaining needles in the two subsamples for each plot were sent to Oregon State University (Central Analytical Laboratory, Department of Crop and Soil Science) for grinding and chemical analysis without replication. This facility uses standard analytical methods for agricultural laboratories (Gavlak and others 1994). Air-dried samples are ground

or pulverized to pass a 40-mesh screen and subsamples removed for determination of dry matter content. About 250 mg (0.088 oz) of sample is used to analyze K plus 11 other elements simultaneously by using nitric acid/hydrogen peroxide digestion (Kingston and Jassie 1986, Sah and Miller 1992) in conjunction with microwave heating in closed Teflon® vessels. Concentrations of the elements in the digest are determined by atomic absorption spectrometry or inductively coupled plasma atomic emissions spectroscopy. Nitrogen is determined with about 150 mg (0.053 oz) of tissue by using a resistance furnace and a thermal conductivity detector as described by Sweeny (1989). Concentrations of all elements are calculated on a dry matter basis.

Experimental Design and Analysis

All statistical analyses were conducted in SAS (SAS Institute Inc. 1999) by using a test criterion of $\alpha = 0.10$ to judge statistical significance. The experimental design is a randomized, complete block with four fertilizer treatments replicated seven times. Experimental units are 0.20-acre circular measurement plots. Four plots compose each of seven blocks; one of four fertilizer treatments was randomly assigned to each plot. Our blocking on the basis of I.I. should further ensure that all treatments had equal chance of sampling comparable intensities of I.I. The 28 plots were located among three soil types but not in equal numbers. Inoculum index (I.I.) was estimated for each plot after stumps were surveyed in summer 1997 for incidence and severity of LRR (table 1). Our intent was to evaluate I.I. in the previous, naturally regenerated stand as a predictor of LRR-caused mortality in the subsequent plantation.

Hypothesis 1 (Incidence of *Phellinus* in stumps of the original stand and characteristics of the 4-year-old plantation)–
Conventional correlation analysis was used to evaluate relations between Douglas-fir mortality from LRR, and TPA in the 4-year-old plantation before fertilization. The dependent variable was TPA killed by LRR as initially tallied in summer 1997.

In addition, we used analysis of variance (ANOVA) to test for differences in I.I. among the three soil types in the study area. We assumed that differences in I.I. among the three soil types would support earlier speculation that risk of LRR was related to soil characteristics.

Regression analysis was used to test the relation of cumulative 8-year mortality from LRR after fertilization versus either I.I. or density of *P. weiri*-infected stumps

(stumps/acre). Indicator variables were specified in each model to enable testing of separate intercepts and slopes for each fertilizer treatment with the extra-sums-of-squares method (Neter and others 1989). To avoid inflating the coefficient of determination (r^2), only those plots with LRR-caused mortality, *P. weirii*-infected stumps, or both were used in the analysis (18 of 28 plots).

Hypotheses 2 and 3 (tree mortality related to K and N fertilizers)– First-year (1998) and cumulative 8-year (1998 through 2005) mortality (TPA) were analyzed by analysis of covariance (ANCOVA) by using 1997 TPA (tagged trees) as the covariate. With seven blocks of four plots each, sources of variation in the ANCOVA were:

Source	d.f.
Covariate (1997 TPA)	1
Block effects (I.I.)	6
Orthogonal comparisons:	(3)
0 and K vs. N and K + N	1
0 vs. K	1
N vs. K + N	1
Error	17
Total (7 X 4) -1	27

To test for treatment effects over time on cumulative yearly mortality from LRR, *Armillaria* spp., and other causes, and on current TPA, yearly values for each variable were subjected to a repeated-measures ANCOVA in SAS procedure MIXED. The covariate, TPA 1997, was included in each analysis to adjust for initial postthinning differences in TPA. The analyses were conducted with maximum likelihood estimation assuming an autoregressive covariance structure to account for time series trends.

Hypothesis 4 (tree growth related to K and N fertilizers)– Average height of surviving Douglas-fir in years 1 and 6 after fertilization and also 6-year periodic annual height increment (PAI; 1998-2003) of survivors were analyzed by ANCOVA using average height of tagged trees at fertilization (1997 HT) as the covariate. Average d.b.h. (calculated as quadratic mean diameter = diameter of the tree of mean basal area), stand basal area, and volume 6 years after fertilization were analyzed by ANCOVA also using 1997 height as the covariate. Except for differences in the covariate used, the ANCOVA tables for tree growth were identical to that for cumulative mortality.

Table 2—Correlation matrix and mean values of selected variables before fertilization at Voight Creek, Washington, per-acre basis

Variable	Inoculum index	Douglas-fir density: Initial density (total)	Douglas-fir density: Postthinning density (1997 trees per acre)	Prefertilization mortality (trees per acre) from laminated root rot
		– – – – – Correlation coefficient (r)[a] – – – – –		
Inoculum index				
Initial density	-0.264			
Postthinning density	-.203	0.509		
Prefertilization mortality from laminated root rot	.328	.218	-0.046	
	Index	*– – – – – – – –Trees per acre – – – – – – – –*		
Mean	437	1,108	451	2.1
CV[b]	153	38	13	301

[a] With sample size of 28 (26 d.f.), the sample correlation coefficient, r, must be ≥ 0.317 for statistical significance at $\alpha = 0.10$, and ≥ 0.375 at $\alpha = 0.05$.

[b] CV = coefficient of variation = (standard deviation/mean) x 100.

Results

Prethinning Stand Density and Tree Losses From Laminated Root Rot, by Inoculum Index

Douglas-fir density before thinning averaged 1,108 TPA and it was not significantly correlated with I.I. (r = -0.26, table 2). Likewise, early mortality from LRR was not significantly correlated with initial TPA (r = 0.22).

Only 4 of the 28 plots contained LRR-killed seedlings at plot establishment in 1997 (4 years after planting). Number of LRR-killed trees tallied at plot establishment ranged from 0 to 30 TPA, and averaged 2.1 TPA among the 28 plots (table 2). This early mortality was related positively to I.I.; the correlation coefficient, r, was 0.33 and statistically significant (table 2).

Inoculum Index by Soil Type

In the preharvest stand, I.I. on soil type 2 (I.I. = 634) averaged about 2.2-fold that on soil type 3 (I.I. = 286); these means were not significantly different (p = 0.42, table 3).

Postthinning Stand Density by Soil Type and Subsequent Fertilizer Treatments

Although one plot on each soil before thinning was clearly overstocked (about 2,000 TPA; table 1), stand density of Douglas-fir after initial thinning and before fertilization did not differ significantly among the three soils ($p = 0.13$; statistical test not displayed). Initial stand density of Douglas-fir after thinning averaged 451 TPA (table 2) and also did not differ significantly among the subsequently assigned fertilizer treatments ($p = 0.14$; statistical test not displayed). We infer that future effects of fertilizer treatments were not likely to be confounded or masked by initially greater numbers of *P. weirii*-susceptible Douglas-fir among the fertilizer treatments.

Initial Mean Height by Soil Type

As anticipated, mean height of Douglas-fir before fertilization averaged greater (about 30 percent) on soil 3 (Nargar sandy loam) than on soils 1 and 2 (Barneston very gravelly sandy loam).

Table 3—Unbalanced analysis of variance of inoculum index and mean 1997 Douglas-fir height among soil types before fertilization at Voight Creek, Washington

		Inoculum index		1997 height	
Source of variation	**Degrees of freedom**	**Mean squares**	**p**	**Mean squares**	**p**
Soil type	2	406,379	0.420	3.557	<0.001
Error	25	452,474		0.307	
Total	27				

Means (and standard errors)

	N[a]	**Inoculum index**	**1997 height**
			Feet
Soil type:			
1	4	301 (336)	3.2 (0.3)
2	12	634 (194)	3.3 (0.2)
3	12	286 (194)	4.3 (0.2)

[a] N count equals the number of plots on specified soil type.

Foliar Analysis

Needle weight–
Although the mean weight of 100 needles averaged about 12 percent less for treatment K than for the nonfertilized treatment, the difference was nonsignificant ($p = 0.17$, table 4). An increase of about 11 percent in mean needle weight after N fertilization also was nonsignificant ($p = 0.13$).

Nitrogen concentration averaged about 40 percent greater in the presence versus absence of N fertilizer.

N and K concentration–
The four fertilizer treatments differed in mean N and K concentrations in Douglas-fir foliage collected in October after the first growing season after fertilization (table 4). Nitrogen concentration averaged about 40 percent greater in the presence versus absence of N fertilizer (treatments N and K+N vs. treatments 0 [non-fertilized] and K). Although fertilization with 300 lb K/acre increased foliar K-concentrations by 12 percent over that of nonfertilized trees, this difference was not significant ($p = 0.30$). In contrast, N fertilization reduced foliar K concentration in first-year needles by about 14 percent ($p = 0.10$).

N and K content–
Trees fertilized with N had needles that averaged 53 percent greater N content ($p = 0.002$). Mean content of K in current foliage ranged from 4.8 to 5.1 mg/100 needles, and did not differ among treatments ($p = 0.58$).

Tree Losses From Laminated Root Rot After Fertilization

Among the four treatments, mean cumulative 8-year mortality from LRR ranged from 20 to 41 TPA.

Relation to fertilization—
Douglas-fir losses from LRR continued on the four plots with initial losses from the disease and commenced on half of the remaining 24 plots, including some where I.I. = 0. Among the four treatments, mean cumulative 8-year mortality from LRR ranged from 20 to 41 TPA (table 5). Initial tree density (tagged TPA in 1997) was significant as a covariate in the ANCOVA for prefertilization mortality from LRR ($p = 0.028$). Adjusted treatment means of TPA killed by LRR did not differ among fertilizer treatments in the year before fertilization (as expected) nor in the first year or the cumulative 8-year total after fertilization (table 5).

Plots with the highest density of LRR-infected stumps in the original stand tended to be those with the greatest numbers of trees killed by LRR.

Relation to inoculum index—
Plots with the highest density of LRR-infected stumps in the original stand tended to be those with the greatest numbers of trees killed by LRR (fig. 4). Cumulative 8-year mortality of Douglas-fir from LRR had positive, statistically significant

Table 4—Analysis of variance of needle attributes of Douglas-fir one growing season after fertilization at Voight Creek, Washington

Source of variation	df	Weight of 100 needles		N percent		K percent		N content		K content	
		Mean squares	p	Mean squares	p	Mean squares	p	Mean squares	p	Mean squares	p
Treatment[a]	(3)										
(1) 0 and K vs. N and K+N	1	0.010	0.133	0.816	<0.001	0.046	0.096	46.26	0.002	0.173	0.581
(2) 0 vs. K	1	.008	.173	.001	.802	.016	.296	1.94	.363	.009	.897
(3) N vs. K+N	1	<.001	.842	.052	.146	<.001	.917	1.52	.419	<.001	.986
Error	8	.003	—	.020	—	.013	—	2.09	—	.524	—
Total	11										

Means (and standard errors)

Fertilizer treatment	Weight of 100 needles		N percent		K percent		N content		K content	
	Weight	Relative[b]	Percent	Relative	Percent	Relative	Weight	Relative	Weight	Relative
	Grams						*Milligrams*		*Milligrams*	
0	0.60	100	1.32	100	0.85	100	7.95	100	5.08	100
K	.53	88	1.29	98	.95	112	6.81	86	5.00	98
N	.63	105	1.73	131	.77	91	10.81	136	4.80	94
K+N	.62	103	1.92	145	.78	92	11.81	149	4.79	94
Standard error (pooled from ANOVA)	.03	—	0.08	—	.07	—	.83	—	.42	—

[a] 0 = no fertilizer.

[b] Relative, where 0 fertilization = 100

Table 5—Analysis of covariance of initial (1997), first-year (1998), and cumulative (1998-2005) Douglas-fir mortality (trees per acre) from laminated root rot at Voight Creek, Washington

		Douglas-fir mortality					
		Prefertilization		First year		Cumulative	
Source of variation	df	Mean squares	p	Mean squares	p	Mean squares	p
Blocks	6	58.7	0.151	73.3	0.051	6,146.6	0.002
Treatment[a]	(3)						
(1) 0 and K vs. N and K+N	1	68.2	.162	3.4	.727	940.0	.367
(2) 0 vs. K	1	13.6	.522	42.8	.227	288.7	.614
(3) N vs. K+N	1	83.4	.124	4.0	.706	230.9	.652
TPA[b]	1	184.6	.028	64.3	.143	504.2	.506
Error	17	31.9		27.3		1,094.9	
Total	27						

Adjusted means and standard errors	Douglas-fir mortality		
Fertilizer treatment[b]	Prefertilization	First year	Cumulative
		Trees per acre	
0	0.0	2.5	20.4
K	1.5	6.0	29.5
N	6.2	3.0	32.8
K+N	1.3	4.1	40.9
Standard error (pooled from ANOVA)	2.1	2.0	12.6

[a] 0 = no fertilizer.

[b] Means for prefertilization mortality have been adjusted for prethinning density, and those for first-year and cumulative mortality have been adjusted for postthinning density.

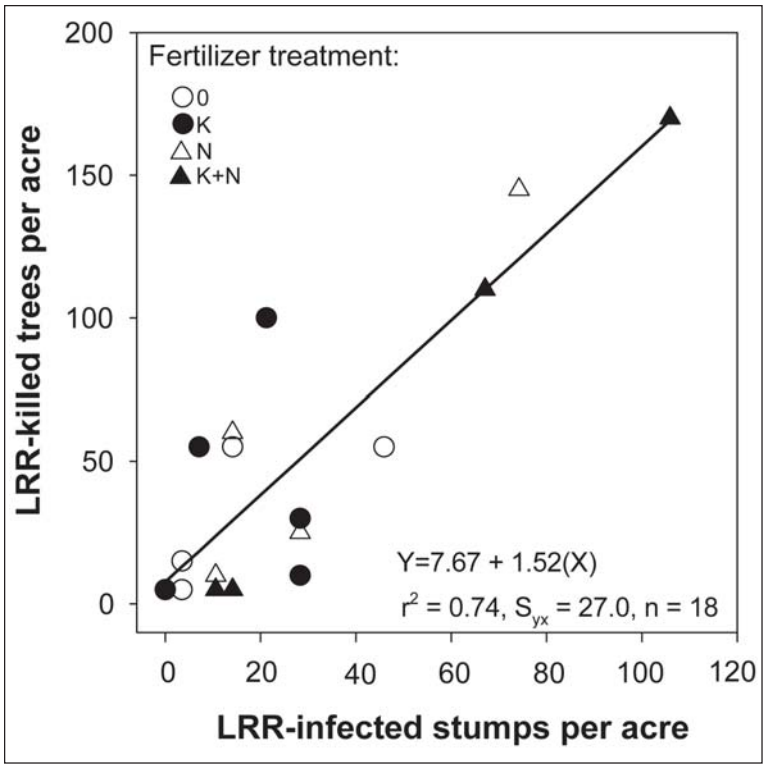

Figure 4—Cumulative (1998-2003) mortality of Douglas-fir from laminated root rot (LRR) as related to density of LRR-infected stumps of Douglas-fir at Voight Creek, Washington. Only those plots with LRR-caused mortality, LRR-infected stumps, or both are shown. 0 = no fertilizer.

relationships with both I.I. ($r^2 = 0.53$) and density of LRR-infected stumps ($r^2 = 0.74$) (fig. 4). We infer from the linear equation ($y = 7.67 + 1.52\ x$) that in the absence of visually detectable infected stumps at this location, about eight Douglas-fir per acre died from LRR in years 4 to 12 after planting. About 1.5 additional seedlings died per LRR-infected stump.

Regression intercepts and slopes for both relations did not differ significantly among fertilizer treatments, indicating there were no significant differences in cumulative mortality per unit of potential LRR infection. Proximity of a tree to infected stumps may have increased the likelihood of its death from LRR, but spatial relationships were not obvious on all plots (fig. 5).

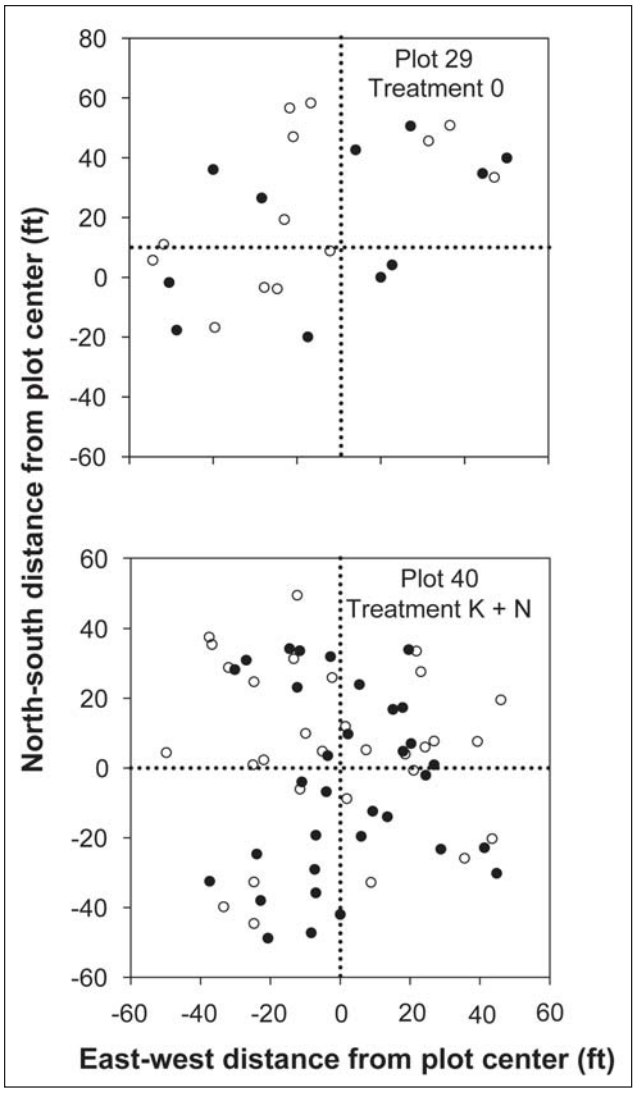

Figure 5—Relative spatial distribution of Douglas-fir killed by laminated root rot (LRR) and of Douglas-fir stumps infected with LRR at Voight Creek, Washington. Two plots (29 and 40) illustrate the relative locations of mortality in plots with low and high densities of infected stumps, respectively. Treatment 0 = no fertilizer.

On nonfertilized control plots, seedlings lost to *Armillaria* spp. in the 8 years after fertilization averaged 34 TPA.

Tree Losses From *Armillaria*

On nonfertilized control plots, seedlings lost to *Armillaria* spp. in the 8 years after fertilization averaged 34 TPA (table 6), 70 percent more than trees killed by LRR (20 TPA, table 5). First-year mortality from *Armillaria* was significantly greater on K-fertilized plots (2 TPA) than on control plots (0 TPA) ($p = 0.09$).

Table 6—Analysis of covariance of first-year (1998) and cumulative (1998-2005) Douglas-fir mortality from *Armillaria* spp. at Voight Creek, Washington

		Douglas-fir mortality			
		First year		Cumulative	
Source of variation	df	Mean squares	p	Mean squares	p
Blocks	6	17.0	0.023	903.7	0.450
Treatments[a]	(3)				
(1) 0 and K vs. N and K+N	1	2.5	.493	1,830.0	.170
(2) 0 vs. K	1	16.5	.090	1,140.6	.274
(3) N vs. K+N	1	5.9	.298	96.7	.746
Trees per acre[b]	1	8.17	.222	484.0	.472
Error	17	5.1		893.6	
Total	27				

Adjusted means and standard errors	Douglas-fir mortality	
Fertilizer treatment[b]	First year	Cumulative
	Trees per acre	
0	0	33.9
K	2.0	15.9
N	.9	5.7
K+N	2.2	10.9
Standard error (pooled from ANOVA)	.9	11.4

[a] 0 = no fertilizer

[b] Means for first-year and cumulative mortality have been adjusted for postthinning density (1997 TPA).

Cumulative losses from *Armillaria* spp. in the 8 years after fertilization were fewer on N-fertilized plots but not significantly.

Cumulative losses from *Armillaria* spp. in the 8 years after fertilization were fewer on N-fertilized plots but not significantly. Treatment means adjusted to a common initial TPA, averaged 8 TPA on N-fertilized (N, K+N) plots vs. 25 TPA on 0- and K-fertilized plots (p = 0.17, table 6).

Tree Losses From Other Agents

In year 1 and in the 8-year period after fertilization, cumulative mortality from agents other than LRR and *Armillaria* was greater after K+N fertilization than after N fertilization (table 7). "Other" agents included some losses from black-stain root

Table 7—Analysis of covariance of first-year (1998) and cumulative (1998-2005) Douglas-fir mortality from other causes at Voight Creek, Washington

		Douglas-fir mortality			
		First year		Cumulative	
Source of variation	df	Mean squares	p	Mean squares	p
Blocks	6	193.6	0.190	426.2	0.074
Treatments[a]	(3)				
(1) 0 and K vs. N and K+N	1	159.8	.257	35.1	.663
(2) 0 vs. K	1	217.5	.189	517.3	.107
(3) N vs. K+N	1	562.6	.042	773.7	.053
Trees per acre[b]	1	9.0	.785	3.1	.896
Error	17	116.3		178.6	
Total	27				

Adjusted means and standard errors	Douglas-fir mortality	
Fertilizer treatment	First year	Cumulative
	Trees per acre	
0	2.7	7.1
K	10.6	19.2
N	5.2	8.0
K+N	17.9	22.9
Standard error (pooled from ANOVA)	4.1	5.1

[a] 0 = no fertilizer.

[b] Means for first-year and cumulative mortality have been adjusted for postthinning density (1997).

The fertilizer treatments did not have a detectable influence on cumulative Douglas-fir mortality from LRR or *Armillaria* spp. over time.

disease, caused by *Leptographium wageneri,* but most causes were not identifiable. By year 8, plots treated with K+N averaged 23 TPA killed by "other" agents, compared to 8 TPA for N-only plots; the difference was significant (p = 0.05).

Cumulative Losses and Residual Trees per Acre

The fertilizer treatments did not have a detectable influence on cumulative Douglas-fir mortality from LRR or *Armillaria* spp. over time (table 8). However, cumulative mortality increased over time, especially that resulting from *Armillaria* on nonfertilized plots (fig. 6). During each of the 8 years of monitoring, mortality from other causes was greater after K+N fertilization than after N fertilization (p = 0.04).

Table 8—Repeated-measures analysis of covariance of cumulative yearly Douglas-fir mortality (trees per acre) in 1998-2005 from laminated root rot, *Armillaria*, and other causes, and surviving TPA at Voight Creek, Washington

		Laminated root rot		*Armillaria*		Other[a]		Surviving TPA	
Source of variation	df	F[b]	p	F	p	F	p	F	p
Treatments[c]	(3)								
(1) 0 and K vs. N and K+N	1	0.37	0.547	0.90	0.352	0.25	0.625	0.06	0.815
(2) 0 vs. K	1	.18	.678	.33	.569	2.47	.130	.35	.558
(3) N vs. K+N	1	.05	.826	.14	.711	4.65	.042	1.06	.313
TPA (1997)	1	.92	.348	.04	.852	2.18	.154	66.43	<.001
Error (treatment effects)	23								
Year	7	8.25	<.001	5.93	<.001	6.31	<.001	15.84	<.001
Treatment x year	21	.95	.527	1.11	.346	.74	.790	.73	.803
Error (year effects)	168								
Total	223								

Note: Adjusted means for each variable are plotted in figures 6. TPA = trees per acre

[a] Includes some losses from black-stain fungus.

[b] ANOVA mean squares are not reported by SAS Procedure MIXED; F-statistics are computed via a formula that does not use the conventional ratios of mean squares.

[c] 0 = no fertilizer.

After thinning and before fertilization, average TPA of Douglas-fir was similar among the four fertilizer treatments (table 9). Despite subsequent losses from disease and other agents in the subsequent 8 years, treatment, average tree density in the 12-year-old plantation in 2005 did not differ significantly as a result of the fertilizer treatments, ranging from 377 to 405 TPA (table 9). Total mortality of tagged crop trees from all causes averaged only 62 TPA (-14 percent). By the 12th year after planting, stocking remained satisfactory at an average density of 390 TPA.

Average tree density in the 12-year-old plantation in 2005 did not differ significantly as a result of the fertilizer treatments, ranging from 377 to 405 TPA.

Tree and Stand Growth

Tree size and growth–

As anticipated, tree height in 1997 before fertilization did not differ among the pending fertilizer treatments (table 9). After covariance adjustment to a common height before fertilization, both average height growth and total height in 1998 were significantly greater in the presence versus absence of N fertilization (p = 0.01). Growth after N fertilizer was significantly greater for height in 2003, height PAI from 1998 to 2003, and d.b.h. in 2003. Among these tree-size variables, the

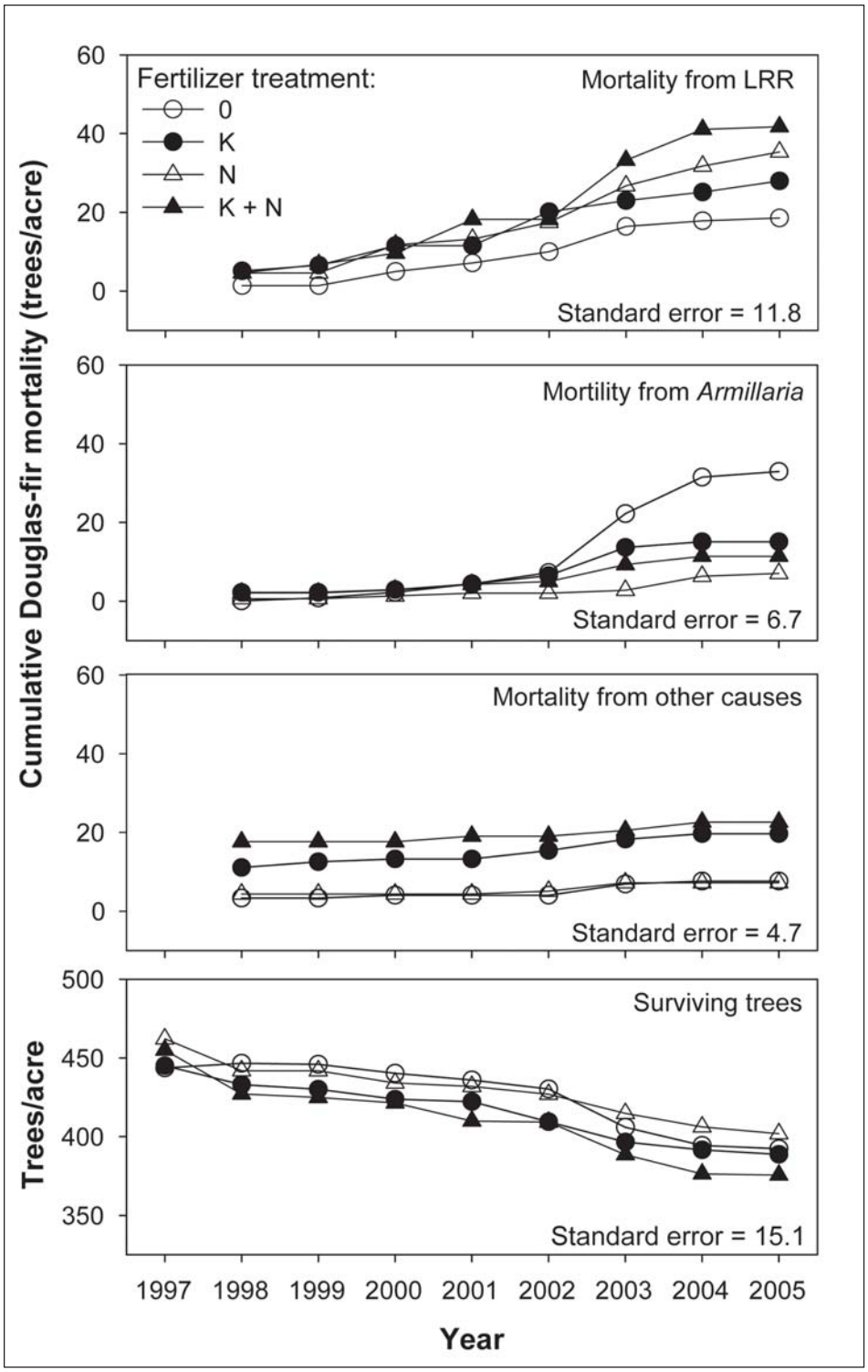

Figure 6—Cumulative yearly mortality of Douglas-fir from laminated root rot (LRR), *Armillaria*, and other causes and resulting density of surviving trees in the 8 years (1998-2005) after fertilization at Voight Creek, Washington. Means have been adjusted for postthinning density (1997 TPA). 0 = no fertilizer.

Table 9—Tree and stand statistics of Douglas-fir averaged by fertilizer treatment at Voight Creek, Washington

Variable	Fertilizer treatment[a]				Standard error	Significant contrasts ($p \leq 0.10$)[b]
	0	K	N	K+N		
1997 height (ft)	4.0	3.5	3.8	3.6	0.3	NS
1998 height (ft)	5.5	5.5	5.7	5.6	.1	(1)
1998 height growth (ft)	1.7	1.8	2.0	1.9	.1	(1)
2003 height (ft)	18.5	17.4	19.8	18.6	.4	(1), (2), (3)
1998-2003 height PAI (ft)	2.4	2.3	2.7	2.5	.1	(1), (3)
2003 diameter breast height (in.)	3.0	2.8	3.2	3.0	.1	(1)
1997 trees/acre	443.6	445.0	462.1	455.0	14.2	NS
2005 trees/acre	390.0	386.8	405.0	376.7	18.6	NS
2003 basal area (ft^2/acre)	18.9	17.4	25.0	21.3	1.5	(1), (3)
2003 volume (ft^3/acre)	169.9	171.6	259.7	215.8	16.7	(1), (3)

[a] Means for trees/acre have been adjusted for postthinning density; all other means have been adjusted for initial height.

[b] Orthogonal comparisons tested were (1) 0 and K vs. N and K+N, (2) 0 vs. K, and (3) N vs. K+N; 0 = no fertilizer.

Note: NS = statistically nonsignificant, PAI = periodic annual increment.

effects of N and K fertilizer were often significant but in opposing direction: growth increased 7 to 12 percent in response to the general effects of N fertilizer, but decreased 4 to 7 percent in response to K fertilizer.

Effects of N and K fertilizer were often significant but in opposing direction.

Stand basal area and volume—

Both live-stand basal area and volume per acre in the 10-year-old plantation (after the 2003 growing season) differed among treatments. Plots fertilized with N (either N or K+N) averaged about 28 percent more basal area and about 39 percent more volume than those not fertilized with N (either 0 or K) (table 9). Moreover, both basal area and volume per acre in plots fertilized with K+N were 15 to 17 percent less than in plots fertilized with N only. The results correspond to the effects of these treatments on tree diameter and height growth. Note that surviving live trees that had not attained breast height (4.5 ft) were not included in the computation of stand basal area and volume. They were included in TPA estimates.

Plots fertilized with N (either N or K+N) averaged about 28 percent more basal area and about 39 percent more volume than those not fertilized with N.

Discussion

Based on number and size of infected stumps in the preceding stand, the estimated amount of *P. weirrii* innoculum (I.I.) was unrelated to soil type.

Laminated Root Rot in the Preceding Stand

Based on number and size of infected stumps in the preceding stand, the estimated amount of *P. weirrii* innoculum (I.I.) was unrelated to soil type, despite large differences in slope percentage and gravel-cobble content among the three soils. These results do not support earlier speculations: Reynolds and Bloomberg (1982) suggested that gravel content and shallow soils decrease soil volume available to roots and thus increase frequency of root contacts and spread of LRR. Based on literature reviews, Littke[3] stated that factors that increase frequency of root contacts or shorten the interval to root occupancy will increase mortality from LRR.

Laminated Root Rot in Current Plantation

When predicting plantation susceptibility to LRR, there may be no benefit from taking the additional measurements required to calculate I.I.

Cumulative 8-year tree mortality from LRR was clearly related to I.I. of the previous stand. This supports our working hypothesis 1 that I.I. is a useful predictor of future LRR. However, the relation with density of LRR-infected stumps (r^2 = 0.74) was considerably stronger than that with I.I. (r^2 = 0.53). This result suggests that, when predicting plantation susceptibility to LRR, there may be no benefit from taking the additional measurements required to calculate I.I.

Thies and others (2005) also compared the utility of I.I. and a simple count of infected stumps in the parent stand to predict losses from LRR in the replacement stand of Douglas-fir. Their sample consisted of a total of 119 plots at five locations. Note that each 0.10-acre plot was centered on a *P. weirii*-infected stump. Therefore in contrast to our fewer but larger plots at a single location, their range of I.I. or number of infected stumps did not include zero. This smaller range of values for the predictor variables could help explain their low r^2 value (0.31 and 0.39 for I.I. and number of infected stumps per acre, respectively) and their conclusion that there was too much unexplained variation in their models for either variable to be useful in predicting future losses from LRR.

The trend of accelerating rates of mortality from LRR for all treatments is probably a result of accelerating rates of the tree growth and their ability to occupy progressively larger soil volumes, which would increase their probability of contact

[3] Littke, W. [N.d.]. Detection and evaluation of laminated root rot disease in Douglas-fir stands. [Centralia, WA]: [Weyerhaeuser Western Forestry Forest Pest Management Team] 38 p. Unpublished study plan. On file with: Forestry Sciences Laboratory, 3625 93rd Avenue SW, Olympia, WA 98512.

with LRR inoculum. Based on our recent surveys, LRR-caused mortality is increasing faster in treatment K+N than in the other treatments; however, we cannot predict if this trend will continue.

Foliage Indicators

Current-year foliage collected in year 5 after planting on nonfertilized plots averaged 1.32 percent N (moderately deficient) and 0.85 percent K (more than adequate for normal growth of coast Douglas-fir saplings) (Carter 1992). Mean weight of 100 needles the first growing season after fertilization did not differ significantly among fertilized and nonfertilized trees. Needles of K-only-fertilized trees averaged about 12 percent lighter than nonfertilized, but N-only fertilized averaged about 5 percent heavier. When the foliage was collected, current twigs were visibly longest on N-fertilized trees and shortest on K-only fertilized trees. Fertilization with 300 lb N/acre, both as a single treatment or combined with K, increased both N concentration and content in needles by at least 30 percent, and decreased K percentage by 8 to 9 percent. Reduction of K concentration in Douglas-fir foliage after N fertilization is frequently reported (Heilman and Gessel 1963, Peterson and others 1986, Thies and others 2006).

Fertilization with K-only may have slightly increased K concentration but not content over that in control needles, in part because needles were lighter (table 4). Thies and others (2006) applied both smaller (200 lb K/acre) and larger (400 lb K/acre) amounts of potassium chloride (KCI) in spring to newly planted Douglas-fir. Needles collected that fall averaged slightly greater K-concentration than needles from nonfertilized seedlings, although differences were statistically nonsignificant ($p > 0.05$). Although average concentration of K in needles from nonfertilized seedlings at that site (0.85 percent) was similar to that at our Voight Creek study area, further comparisons are not possible because 100-needle weights were not determined.

Moore and others (2004) provided useful information about foliar nutrient concentrations and variation in four species, including Rocky Mountain Douglas-fir (var. *glauca*). Their recommended K-concentration for optimal growth of that variety is 0.60 percent, which is considerably lower than the 0.85 percent that both we and Thies and others (2006) measured in foliage on nonfertilized coast Douglas-fir.

These relations among foliar weight, nutrient concentration, and content can be presented graphically (fig. 7) and used to assess effects of fertilization on nutrient status (Timmer and Ray 1988). Their table for interpreting relations after fertilization follows.

Direction of shift	Response in: Needle weight	Response in: Nutrient Conc.	Response in: Nutrient Content	Change in: Nutrient status	Change in: Possible diagnosis
A	+	-	+	Dilution	Nonlimiting
B	+	O	+	Unchanged	Nonlimiting
C	+	-	-	Deficiency	Limiting
D	O	-	-	Luxury consumption	Nontoxic
E	-	++	±	Excess	Toxic
F	-	-	-	Excess	Antagonistic

Addition of 300 lb K/acre at Voight Creek resulted in a type F shift in needle weight and N relations, which suggests excess K status and antagonistic interactions between K and N status. This treatment also resulted in a type E shift in needle weight and K relations, which suggests excess K in the needles and possible toxic effects. Our mortality and growth data (table 9) generally support these indications of unfavorable nutrient status after K-fertilization.

Our mortality and growth data generally support these indications of unfavorable nutrient status after K-fertilization.

The combined K+N treatment did not result in major shifts in foliar relations that would have indicated unfavorable nutrient status or a growth-limiting diagnosis (fig. 7). We infer from mortality and growth data (table 9), however, that this combination resulted in increased mortality from "other" causes (table 7) and reduced 6-year height growth and stand basal area and volume in 2003 (table 9).

Our K source, KCl, is a highly soluble fertilizer. We used KCl instead of potassium sulfate (K_2SO_4) because it is a more concentrated source of K and to avoid adding another nutrient, sulphur. Our experimental dosage was deliberately large to ensure adequate treatment strength to induce an effect. We infer from the visually poorer vigor of K-only fertilized trees relative to the nonfertilized control, however, that excessive K^+ or Cl^- ions may have had a temporary toxic effect on these trees. Because K-fertilization did not reduce foliar concentration of magnesium (Mg), we infer that a Mg deficiency was not induced by our K fertilization (as a potential explanation for reduced growth of K-fertilized trees).

Excessive K^+ or Cl^- ions may have had a temporary toxic effect on these trees.

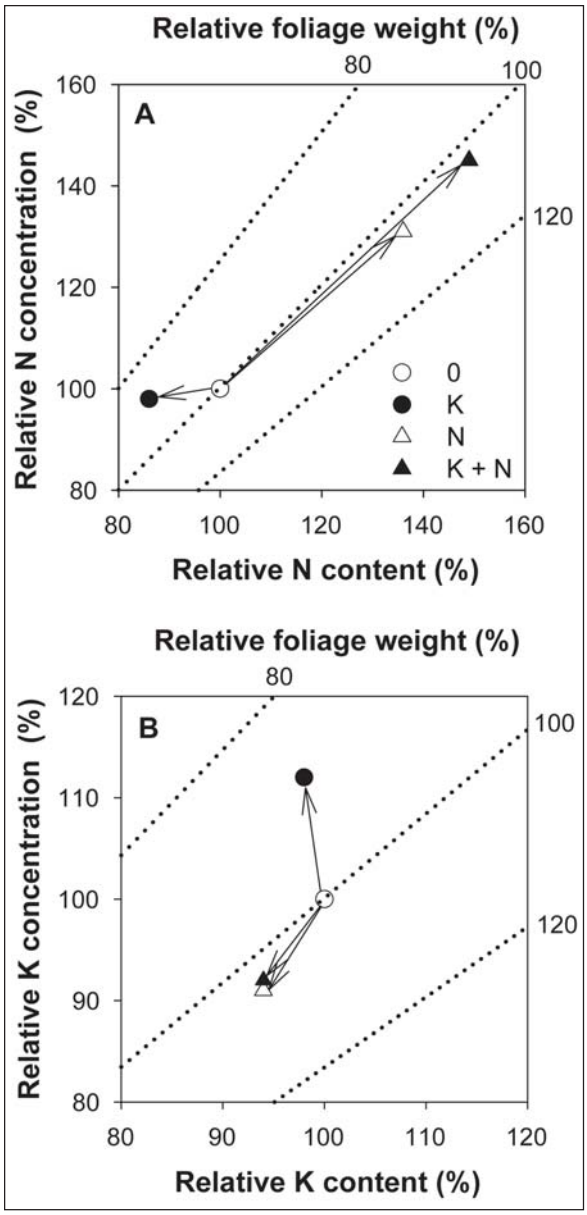

Figure 7—Graphical analysis of nutrient concentration, nutrient content, and dry weight of Douglas-fir needles for (a) nitrogen (N) and (b) potassium (K) 1 year following fertilizer treatments at Voight Creek, Washington. Each variable is expressed as a percentage of the nonfertilized control.

Tree Mortality and Growth Responses to Fertilization

Seedlings died from LRR, *Armillaria,* and other agents (table 10). Despite seedling losses from various agents in the 8-year period after fertilization, the 12-year-old plantation averaged 390 TPA and was adequately stocked. Because initial number of tagged trees (crop trees) varied among the 28 plots, we used initial TPA after thinning as a covariate to adjust observed treatment means of mortality to the common or average TPA for all plots (451 TPA). Observed means for the nonfertilized and K-only treatments were increased slightly through this adjustment because their

Table 10—Summary of initial trees per acre after thinning (TPA97), cumulative (1998-2005) mortality after fertilization by source, and final trees per acre (TPA05) for Douglas-fir at Voight Creek, Washington

Fertilizer treatment[b]	Cumulative mortality by source					TPA05
	TPA97	LRR	*Armillaria*	Other	Total	
	Trees per acre					
Adjusted means from tables 5, 6, 7, and 9[a]						
0	443.6	20.4	33.9	7.1	61.4	390.0
K	445.0	29.5	15.9	19.2	64.6	386.8
N	462.1	32.8	5.7	8.0	46.4	405.0
K+N	455.0	40.9	10.9	22.9	74.8	376.7
Unadjusted means[c]						
0	443.6	19.3	32.9	7.1	59.3	384.3
K	445.0	28.6	15.0	19.3	62.9	382.1
N	462.1	34.3	7.1	7.9	49.3	412.9
K+N	455.0	41.4	11.4	22.9	75.7	379.3

Note: LRR = laminated root rot.

[a] Adjusted means from analyses of covariance adjusted for initial trees per acre.

[b] 0 = no fertilizer.

[c] Unadjusted means from analysis of variance.

Fertilizer at 300 lb K/acre did not reduce tree mortality from LRR.

mean TPA in 1997 was less than the overall average TPA. Conversely, raw means for the N and K+N treatments were adjusted slightly downward. This adjustment changed treatment means, but seldom changed treatment rankings.

Contrary to our hypothesis 2, fertilization with K fertilizer at 300 lb K/acre did not reduce tree mortality from LRR. Instead, we observed increased cumulative mortality from "other" causes in K-fertilized plots (12 more TPA died after K-only fertilization than in the nonfertilized control and 15 more TPA died after K+N than after N-only fertilization (table 7). We hypothesize that additional losses after KCI fertilizer may be caused by a toxic effect of excess potassium or chloride ions in the soil solution. Moreover, this suspected toxic effect apparently decreased tree growth response to N.

Thies and others (2006) tested the efficacy of the same KCI fertilizer at smaller and larger dosages (200 and 400 lb K/acre) and also in combination with urea (200 lb K and 200 lb N/acre). The fertilizers were broadcast shortly before 2-1 Douglas-fir seedlings were planted in and around a total of 44, 0.05-acre plots. The soil at their study area was mapped as Cispus cindery sandy loam, a very deep, somewhat

excessively drained soil on slopes of zero to 8 percent (USDA SCS 1987). Based on modal characteristics of this soil (coarse texture, low clay content), we infer that reactions and fate of K (KCI) and N fertilizers at this location would be similar to those at our Voight Creek study area. Losses from LRR in the 7 years after fertilization were similar for all fertilizer treatments and the no-fertilizer treatment. They concluded that a single application of K or K+N to Douglas-fir seedlings at the rates tested was not an effective management strategy for LRR. Losses from other causes averaged 51 percent of total losses in the 4 years before the plots were thinned to 500 TPA. Of the total losses in the next 3 years, 17 percent were not from LRR. For the entire 7-year period after planting and fertilization, non-LRR losses were not related significantly to fertilizer treatment (Thies, unpublished data).

Results of this research do not support our hypothesis 3 that application of N fertilizer could increase tree mortality from LRR. Nitrogen-fertilized plots did not have a greater number of LRR-killed trees (table 5) or mortality per unit of I.I. or density of LRR-infected stumps (fig. 4). Nitrogen-fertilized plots, however, did have reduced losses from *Armillaria,* although these reductions were not statistically significant (table 6).

Six years after fertilization, N-only fertilization resulted in the largest trees and the greatest stand basal area and volume, presumably in response to improved fertility. Thies and Westlind (2005) also reported no effect of N fertilization (300, 600, 1,200 lb N/acre as ammonium nitrate) on losses of Douglas-fir from LRR at five locations, and reported a decade-long growth response to N at one of these locations.

To confirm observations from our study at Voight Creek, we plan to assess the continued effects of fertilization on tree mortality. We acknowledge that detecting treatment effects on rates of tree mortality is often difficult. Statistical tests of short-term changes in LRR-caused mortality are often statistically nonsignificant despite large differences in mortality among treatment means, because statistical comparisons are based on a large experimental error (Thies, unpublished data).

We surmise that detecting effects of N fertilizer on Douglas-fir growth at N-deficient sites (like our Voight Creek site) is inherently easier than detecting changes in rates of mortality from LRR. One simple explanation is limited sample size: all trees contribute to growth (for some period of time), but relatively few trees die, especially from LRR.

Design Considerations

Our study design differs from the other study involving N- and K-fertilization to reduce losses of Douglas-fir seedling to LRR (Thies and others 2006). Our circular (0.2 acre) plots were systematically located in a 4-year-old plantation, whereas those near Morton, Washington, were smaller (0.05 acre) and each was centered on a *P. weirii*-infected stump. Our Voight Creek plots are four times larger and should, therefore, provide a larger sampling of *P. weirii* -infected area. At plot establishment and after an initial thinning to reduce stem density by cutting mostly volunteers, our plots averaged 451 TPA at fertilization. In contrast, the deliberately close planting spacing near Morton provided a greater TPA sample when fertilized (4,720 TPA). Fertilizers in our study were applied before the fifth growing season after planting rather than before planting. Consequently, in our study, older seedlings and greater vegetative cover probably captured more of the fertilizer. Our dosage (300 lb N/acre) exceeded the dosage near Morton (200 lb N/acre).

We suggest the following techniques could improve effectiveness of future field trials testing fertilization and root disease:

1. Conduct trials at locations where treatment is likely to eliminate a growth-limiting factor; e.g., where there is a large amount of disease inoculum or where N or K is deficient.

2. Confine trials to uniform conditions or increase plot size. Create experimental blocks on recognizable strata, e.g., similar inoculum index, stand density, soil type, or slope class. This can reduce the error term by partitioning out variation associated with those strata.

3. Install an adequate number of replications; more replications are desirable where plots are small or site or stand conditions are variable.

4. Use a covariate that expresses differences in pretreatment or starting conditions among plots, e.g., number of Phellinus-infected stumps, I.I., or stem density.

As documented in the “Methods” section, we used several of these techniques when designing this field trial. Fortuitously, our blocking on I.I. resulted in the four treatments also being nearly balanced among the two contrasting soil series in experimental area (table 1). Because the gravelly Barneston series (soils 1 and 2) is much less productive than the Nargar sandy loam (our soil 3) This balance avoided a potential confounding with the fertilizer treatments and simplified our interpretations of growth response related to fertilizer (table 11).

Table 11—Growth characteristics for the seven nonfertilization plots

Item	Barneston (N=3)		Nargar (N=4)		Difference	
					Absolute	*Percent*
Mean height (SE) (ft):						
1997	3.5	(0.3)	4.4	(0.5)	0.9	26
2003	17.8	(0.8)	20.7	(1.7)	2.9	16
Mean d.b.h. (SE) (in)						
2003	2.7	(0.1)	3.5	(0.3)	.8	30
Mean stand (SE) in 2003:						
Basal area (ft^2/acre)	17.1	(0.7)	25.9	(5.3)	8.8	51
Volume (ft^3/acre)	128	(35)	276	(66)	148	116

Conclusions

1. Numbers of and disease severity in *P. weirii*-infected stumps from the preceding 70-year-old stand differed greatly among our 28, 0.20-acre plots and their immediate buffer areas. Twelve plots had no infected stumps. Inoculum index estimated from infected stumps of the preceding stand was not related to soil type, which included both gravelly and nongravelly soils.

2. The I.I. was a useful blocking factor to ensure uniform sampling of the potential for LRR-infection among the fertilizer treatments.

3. Mortality of Douglas-fir trees caused by LRR averaged only about 2 TPA in years 3 and 4 after planting, but the subsequent annual rate of mortality increased through year 12.

4. Subsequent cumulative 8-year mortality from LRR was significantly related to both I.I. and density of infected stumps per acre. Seedlings also died from LRR in plots in which no infected stumps were detected.

5. Cumulative mortality per unit of potential LRR inoculum did not differ significantly among treatments, indicating that neither K nor N fertilization changed Douglas-fir susceptibility to LRR.

6. Cumulative tree mortality from other causes in the first year and 8 years after fertilization was greater on K-fertilized plots. We infer that trees died soon after fertilization from possible toxic effect of excess potassium salts, especially when combined with urea fertilizer.

7. The N fertilization increased tree growth and size by year 10 after planting; K fertilization may have decreased tree growth.

Acknowledgments

We thank the former forest owner, Champion Pacific Timberlands, Inc. and the PNW Research Station for initially funding this study, and the Campbell Group, LLC for permitting our continued use of the study area. Michael Anne French, Dan Lovato, Brian Burke, Harry Anderson, and Douglas Westlind assisted us greatly. Thanks to Pam Phillips, Robert Nichelson, Dan Omdal, Amy Ramsey, and Melanie Kallas for their assistance with the field measurements. Thanks also to technical reviewers of our draft manuscript: Tim Max, Terry Shaw, Pat Cunningham, Gregory Filip, and Darlene Zabowski.

Metric Equivalents

When you know:	Multiply by:	To find:
Inches (in)	2.54	Centimeters
Feet (ft)	.305	Meters
Miles (mi)	1.609	Kilometers
Acres	0.405	Hectares
Ounces (oz)	2840	Milligrams (mg)
Pounds (lb)	0.454	Kilogram
Pounds per acre (lbs/acre)	1.1208	Kilograms per hectare
Degrees Fahrenheit (^{o}F)	0.556 and subtract 17.8	Degrees Celsius
Square feet per acre (ft^2/per acre)	0.230	Square meters per hectare
Cubic feet per acre (ft^3/acre)	0.070	Cubic meters per hectare

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