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ScienceDirect
Forest Ecology and Management 235 (2006) 212–218
Forest Ecology and Management
www.elsevier.com/locate/foreco

Application of chloropicrin to Douglas-fir stumps to control laminated root rot does not affect infection or growth of regeneration 16 growing seasons after treatment☆

Walter G. Thies *, Douglas J. Westlind

USDA Forest Service, Pacific Northwest Research Station, Forestry Sciences Laboratory, 3200 Jefferson Way, Corvallis, OR 97331, USA

Received 8 July 2006; received in revised form 11 August 2006; accepted 11 August 2006

Abstract

Phellinus weirii (Murr.) Gilb. causes laminated root rot (LRR), a major disease affecting growth and survival of *Pseudotsuga menziesii* (Mirb.) Franco (Douglas-fir) and other commercially important conifer species throughout the Pacific Northwest. This disease is known to spread to a replacement stand by root contact between replacement trees and residual infected stumps and roots from the harvested stand. One strategy to manage LRR is to reduce the residual inoculum on a site through application of a fungicidal chemical to infested stumps. The first two studies in this series established that chloropicrin (trichloronitromethane), applied to infested stumps, largely eliminates *P. weirii* from most of the belowground biomass and determined the effective dosage to apply. This third study approximated an operational application and based treatment success on reduction of LRR in the replacement stand. The study area was an 8-ha, 65-year-old naturally regenerated second-growth stand that was predominantly Douglas-fir. The stand was surveyed preharvest and postharvest (clearcut). Each *P. weirii*-infected entity (standing dead or down trees and stumps) was marked, and the location of its center was mapped. Circular plots (0.04 ha) were located so as to include concentrations of infested stumps. Plots were stratified based on their estimated biomass of inoculum into eight replicate blocks of four plots each. Three chloropicrin treatments and an untreated control were assigned randomly to four plots in each of the eight blocks: (a) 100% labeled dosage to all stumps, (b) 20% labeled dosage to all stumps, (c) 100% labeled dosage to only visibly *P. weirii*-infected (stump-top stain or advanced decay) stumps, and (d) control (nothing done to the stumps). Holes were drilled into stump tops, a dose of chloropicrin poured in, and the holes plugged. The labeled dosage was 3.3 ml of chloropicrin per kilogram of estimated stump and root biomass. Douglas-fir seedlings were planted in the winter following treatment application. When the stand was considered established, each plot was thinned to an inter-tree spacing of 2.4 m and the trees tagged. Diameter at breast height, total height, and mortality of trees were recorded every 2–5 years. A total of 1041 tagged trees were observed for 16 growing seasons following treatment. Application of chloropicrin to stumps in the harvested stand did not influence the rate of LRR-caused mortality or growth of Douglas-fir seedlings in the replacement stand.
Published by Elsevier B.V.

Keywords: Laminated root rot; Disease control; Fumigation; Chloropicrin; Impacts; Epidemiology

1. Introduction

Phellinus weirii (Murr.) Gilb., causes laminated root rot (LRR), a major disease affecting growth and survival of *Pseudotsuga menziesii* (Mirb.) Franco (Douglas-fir) and other commercially important conifer species throughout the Pacific Northwest, and is responsible for large annual losses in stand productivity (Nelson et al., 1981; Thies, 1983; Bloomberg and Reynolds, 1985). The disease influences stand composition (Holah et al., 1993; Ingersoll et al., 1996) and dynamics (Cook, 1982; Holah et al., 1997) by directly killing susceptible trees or by predisposing them to windthrow, insect attack, and other secondary agents. *P. weirii* spreads throughout a stand when uninfected roots contact roots of previously infected living or dead trees. *P. weirii* has been reported to survive saprophytically in the large roots of dead trees or stumps for several decades (Hansen, 1976, 1979) and for as long as 50 years (Childs, 1963). Such

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* Corresponding author. Tel.: +1 541 750 7408; fax: +1 541 758 7760.
E-mail address: wthies@fs.fed.us (W.G. Thies).

0378-1127/$ – see front matter. Published by Elsevier B.V.
doi:10.1016/j.foreco.2006.08.330

persistence and spread allow the fungus to remain on the site beyond the life of an individual host and to infect trees of the replacement stand (Tkacz and Hansen, 1982). The biology, distribution, and impact of the disease, relative susceptibility of host species, and options for management are summarized in Thies and Sturrock (1995) and Hansen and Goheen (2000).

Strategies to manage laminated root rot (Thies and Sturrock, 1995) have focused on changing species composition or reducing inoculum before a stand is regenerated with a susceptible species. Fumigation is one means of reducing inoculum of some root rotting fungi. Reports of fumigant application to soil as well as directly to wood to destroy particular fungi have been reviewed previously (Filip, 1976; Filip and Roth, 1977; Thies and Nelson, 1982). Examination of excavated stumps colonized by *P. weirii* has shown that extensive stain or decay of the stump top is contiguous with advanced decay and stained wood within the root collar and major roots (Thies and Sturrock, 1995). The presence of decay columns forming ''ducts'' from the stump top to infected portions of the root system suggested the possibility that placing a volatile liquid biocide, such as chloropicrin (trichloronitromethane), into an infected stump would allow the fumigant to volatilize and gasses to diffuse through the stump and roots and kill *P. weirii* (Thies and Nelson, 1982).

This paper describes the third in a series of stump fumigation studies. In the first study (Thies and Nelson, 1982) fumigants were applied for the first time (1978) to stumps colonized by *P. weirii*. Stumps were treated by drilling holes in their tops, pouring a dose of fumigant into the holes and plugging the holes. One year later, the stumps were evaluated and it was found that *P. weirii* had been eliminated from the stumps and most roots. A second study (Thies and Nelson, 1987) tested application techniques with doses based on estimated stump and root biomass. After 20 months, the fumigants had eliminated the fungus from the stumps and reduced the volume of roots supporting *P. weirii* to 22% of the prefumigation volume). Similar results have been reported when using the fumigant Telone II-B (Fraser et al., 1995). These early studies demonstrated that fumigants could move through wood (stumps and roots) to eliminate *P. weirii* inoculum, and that an effective dose was related to the quantity of estimated below ground biomass. During these stump fumigation studies, stumps were excavated and the pathogen viability was tested. The presumption was that, had the fumigated stumps remained undisturbed in the soil, the fumigants and the various microbial antagonists recolonizing the roots might have eliminated the pathogen.

This paper reports the results of the third stump fumigation study, which simulated an operational application in which the treated stumps remained undisturbed in the soil after fumigation. To evaluate the success of stump fumigation as an intervention strategy for LRR, the replacement stand was observed for 16 growing seasons following treatment for mortality caused by LRR.

2. Materials and methods

2.1. Study site

The study area is an 8-ha clearcut with very low relief (≤2% slope) on the Olympic Peninsula near Matlock, WA (47°13′N, 123°26′W). Mean elevation is 40 m, mean annual precipitation is 125 cm, and soil in the study area is a Hoodsport gravelly sandy loam (Haplorthod). The Hoodsport soil series formed in glacial deposits of 50–75 cm of loose ablation till overlaying very compact lodgment till. Surface soil consisted of a moss layer (probably in excess of 75% cover throughout), overlying poorly developed litter and fermentation layers, with a 1- to 5-cm deep humus soil horizon.

The site is class II (McArdle et al., 1961) and supported a 65-year-old naturally regenerated second-growth stand that was predominantly Douglas-fir (99% by harvest volume). Western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) constituted the remainder of the overstory with occasional western red-cedar (*Thuja plicata* Donn ex D. Don) regeneration and frequent patches of vine maple (*Acer circinatum* Pursh). Understory shrubs were primarily sclerophyllous salal (*Gaultheria shallon* Pursh) with frequent sword fern (*Polystichum munitum* (Kaulf.) Presl) and big huckleberry (*Vaccinium membranaceum* Douglas) and a partial ground cover of twin flower (*Linnaea borealis* L.). In-depth analysis of the herbaceous flora of the study area was presented in Ingersoll et al. (1996). Before harvest, the site was part of a contiguous forest stand bordered by an access road and a recent clearcut to the west (Fig. 1).

The study area was clearcut during the summer of 1988. Because success of the fumigation required retention of a volatile chemical in each stump, care was taken to not crack or penetrate residual stumps or disrupt their roots. After conventional felling, whole trees were moved to the landings by shovel logging where they were limbed and bucked. Shovel logging was accomplished by using an excavator with a modified grapple so that trees were lifted over stumps and placed on piles. Next, the excavator was carefully relocated to avoid damage to stumps or roots, and trees were lifted and restacked in another pile. In this way the trees were moved from pile to pile advancing across the clearcut with a minimum of machine travel. This method of moving the trees resulted in little damage to the residual stumps and surface roots of the sort that might be experienced by dragging logs and making multiple trips with skidders. Further, this method of moving the trees significantly reduced the amount of slash on the study area to predominantly small pieces directly in contact with the soil surface. On this very rocky site, the logging activity disturbed much of the thin soil layer and exposed mineral soil.

2.2. Estimation of biomass

An estimate of belowground stump and root biomass for individual stumps was required twice during establishment of this study: (a) for blocking treatment plots according to the relative amount of inoculum present belowground, and (b) to

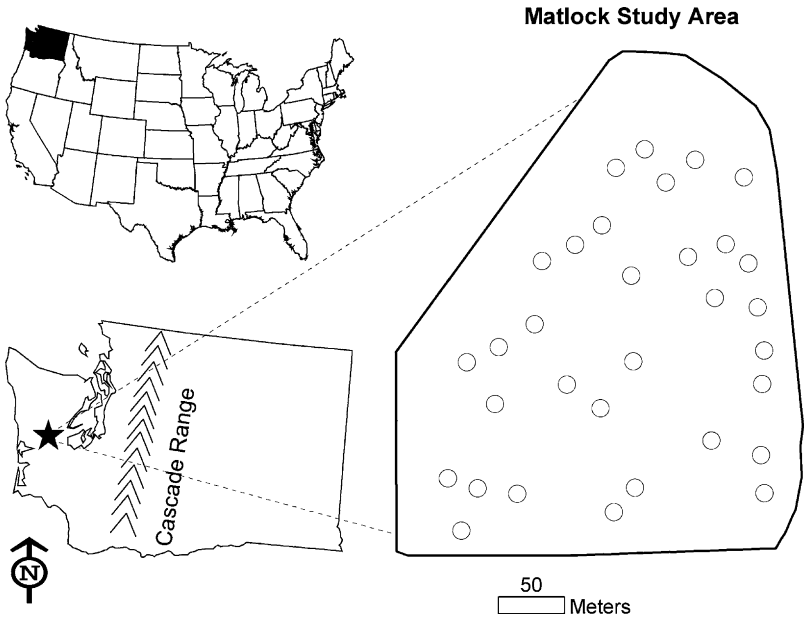

Fig. 1. Location of study area.

establish the dose of chloropicrin to be applied to stumps on treatment plots. Because tree diameter at breast high (dbh) was not available after the harvest, below-ground biomass was estimated from the diameter stump high (dsh) as measured 15 cm above the ground (outside bark) by 2.5-cm diameter classes. Although the relationship of stump diameter to belowground biomass is known now, based on trees from western Oregon (Thies and Cunningham, 1996), at the time this study was begun we were not aware of any reports providing a direct relationship between stump diameter and belowground biomass of Douglas-fir. We therefore used data from trees in this study area to estimate the dbh based on the diameter stump high (dsh) and then applied a published (Gholz et al., 1979) relationship that uses dbh to estimate the below-ground biomass—(a) relationship of dbh to dsh: $Y = -0.2014 + 0.8266\ X$, where Y = dbh in cm, X = dsh in cm ($n = 57$, $r^2 = 0.991$, $S_{y.x} = 0.0109$); trees were from this stand and ranged from 18.3 to 87.4 cm dbh with a mean of 44.2 cm dbh. (b) Relationship of dbh to belowground biomass: $\ln Y = -4.6961 + 2.6929 \ln X$, where Y is the belowground biomass in kg, X the dbh in cm and ln is the logarithm to the base e ($n = 26$, $r^2 = 0.96$, $S_{y.x} = 0.127$; trees ranged from 2.3 to 135 cm dbh. Biomass was based on all roots having a diameter equal to or greater than 10 mm). Data were from trees on the west slope of the Cascade Range (Gholz et al., 1979). (c) Stump biomass: based on field observations, all stumps were assumed to be cut to an average 30 cm above the ground; the aboveground biomass of the stump was estimated as the mass of a cylinder equal in diameter to the stump and 30 cm high. Based on values given for Douglas-fir stem wood (USDA Forest Service, 1974), we assumed an average density of 0.44 g cm^{-3} for this study. (d) Treated biomass: the total biomass to be treated with chloropicrin is the combined estimated *belowground* and *stump biomasses* in kg.

2.3. Plots

The study area was subdivided into 30 m × 30 m units and each was systematically surveyed and mapped on two occasions: (a) a preharvest survey to record infected standing dead or down trees or stumps (entities that could be moved during harvest), topographic features such as the stand edge and skid trails from earlier entries, and living trees at the edge of each identifiable LRR-opening; (b) a postharvest survey to identify infected newly created stumps. Each *P. weirii*-infected entity (standing or down tree, or stump) was marked and measured for diameter, and the location of its center was mapped (Thies and Hoopes, 1979). Infected stumps or down trees were identified based on the presence of stain (incipient decay) or advanced decay typical of that caused by *P. weirii* or by presence of ectotrophic mycelia typical of *P. weirii* on roots near the root collar (Thies and Sturrock, 1995). The survey underestimated the number of infected entities. This is because trees with infected root tissue but lacking visible stain on the stump top or ectotrophic mycelia at the root collar were not diagnosed and marked as infected. Thus, although all visibly infected entities were identified as infected, an unknown number were missed. Identification of all infected stumps would have required excavation or chopping into roots; however, destructive sampling was not compatible with maintaining the integrity of the roots so that they would contain the fumigant.

An index of relative inoculum potential (II) was calculated for each infected entity and aggregated to provide an index for each plot in the study. We assumed that the probability of infection of a tree in the replacement stand is directly proportional to the biomass of the inoculum per unit of area in the cut stand. The index is based on the estimated infected belowground biomass, tree orientation and time since death. The inoculum index for a stump was calculated as follows:

II = BM × O × Y, where II is the relative biomass of potential inoculum, BM the estimated belowground biomass determined as in Section 2.2 above, O the orientation (factors: standing = 1.00; leaning, roots partially out of the ground = 0.66; down, tree on the ground = 0.20), and Y is the years dead (factors: live = 0.75, dead 0–5 years = 1.0, dead 6–10 years = 0.90, dead more than 10 years = 0.50, stump left from an earlier thinning = 0.10). Factor values were based on the senior author's experience with excavating and examining Douglas-fir stumps and roots.

On a map depicting the locations of infected trees or stumps within the study area, points were marked as centers for 0.04-ha circular, nonoverlapping treatment plots positioned to include concentrations of known *P. weirii* inoculum, but avoiding the LRR-caused openings. Centered within each 0.04-ha circular treatment plot was a 0.02-ha circular seedling-measurement (data) plot. This resulted in a 3-m-wide treated buffer around each 0.02-ha measurement plot. Each measurement plot was rated for inoculum potential by totaling the II for stumps within the plot and adding 25% of the II for stumps within the buffer. We assumed that an average of 25% of the roots of infected trees in the buffer would extend into the measurement plot, thus increasing available inoculum. Plots were stratified based on total II into eight blocks of four plots each. The range of II ratings within a block was reduced to a minimum by shifting some plots on the map, thus changing their II rating. Based on the map, we temporarily established plot centers on the ground, and after visually verifying the presence of the mapped infected stumps (and thus the II) on each plot, we permanently marked the center of the plot with a steel rod. Each stump within a plot was labeled with a uniquely numbered aluminum tag nailed to the stump top, its location on the plot was mapped, and its dsh and *P. weirii*-infection status were recorded.

2.4. Treatments

Treatment involved application of chloropicrin at either 100% or 20% of the labeled dosage. Either all stumps were treated or only those with stain (or advanced decay) visible on the stump top that was typical of *P. weirii* (Thies and Sturrock, 1995) were treated. Scattered large stumps from the previous old-growth stand, cut about 70 years earlier, remain on the site. Because most of the LRR inoculum remains viable for only a few decades (Hansen, 1976, 1979) it was assumed that these stumps were no longer serving as effective inoculum and they were not treated. Stumps from the stand harvested for this study but from trees cut earlier as salvage or thinning were treated if they appeared sound enough to retain the fumigant. Three chloropicrin treatments and an untreated control were assigned randomly to the four plots in each of the eight blocks: (a) 100% labeled dosage to all stumps, (b) 20% labeled dosage to all stumps, (c) 100% labeled dosage to only visibly *P. weirii*-infected stumps, and (d) control (nothing done to the stumps). Holes drilled into stump tops to apply the chloropicrin were considered part of the treatment; thus holes were not drilled into stumps in treatment (d) (control) or untreated stumps in treatment (c). The labeled dosage was 3.3 ml of chloropicrin per kilogram of treated stump and root biomass calculated as treated biomass in Section 2.2 above. Measured stump diameter was rounded up to the nearest 2.5-cm increment. Because the available equipment for metering chloropicrin was calibrated in ounces (US, fl.), the calculated dose for each stump was rounded up to the nearest 1.0 oz (29.57 ml).

2.5. Application of fumigant

Holes were drilled in stump tops and fumigant applied in November 1988. Treatment holes, 3.2 cm diameter, were drilled vertically into each stump top at areas of *P. weirii*-caused stain, when present, or in unstained wood. The number of holes in each stump was appropriate to the volume of chloropicrin to be applied. In general the holes were 30 cm deep, 5 cm from the bark, and every 15 cm (for the 100% labeled dosage treatment) around the circumference of the stump. At least two holes were drilled in each treated stump. To avoid drilling through the stump, holes extended only slightly below the soil line. The dose of chloropicrin was distributed approximately equally to all holes in a stump. After fumigation each hole was plugged tightly with a hemlock dowel 3.3 cm in diameter and 8 cm long. One end of each plug was beveled to facilitate driving the plugs into the holes. The beveled end was coated with resorcinol glue (to resist passage of the fumigant through the dowel), and the glue was allowed to harden before the plug was used.

2.6. Seedling establishment

Douglas-fir seedlings (plug-1) from a local seed source were planted on the study area in March 1989. To compensate for losses to animal browsing or damage during planting, plots were examined each fall and interplanted the following winter for three growing seasons to achieve a minimum density of 2200 seedlings ha^{-1}.

In September 1995, the study area was declared established when a majority of the seedlings were considered vigorous and large enough to be unlikely to suffer significant browse damage. Excess seedlings were pulled by hand to leave a final density of 34 Douglas-fir seedlings on each 0.02-ha measurement plot (1700 seedlings ha^{-1}). Retained seedlings were selected to achieve even spacing (approximately 2.4 m intertree spacing) over the entire plot (first priority) and to leave the most vigorous seedlings (second priority). The roots of removed seedlings were examined for evidence of LRR. Each retained seedling was labeled with a uniquely numbered aluminum tag, and its location on the plot was mapped.

Because of animal damage not all plots had 34 seedlings at tagging. The site is rocky and difficult to plant and there was continuous wide spread animal damage from both rodents and elk. After three attempts to bring the seedling count up to the desired density by planting additional seedlings, we accepted 1041 well-established seedlings instead of the intended 1088. Eleven plots had less than 34 seedlings; those plots were

Table 1
Seedling means with standard errors (in parentheses) by treatments and ANOVA p-value of treatment differences

	Ht 1995 (m)	Ht 2005 (m)	dbh 2005 (cm)	Mortality proportion	
				LRR	All causes
p-Value	0.450	0.254	0.494	0.676	0.359
Treatments[a]					
A100	1.21 (0.11)	5.13 (0.43)	7.14 (0.55)	0.12 (0.05)	0.22 (0.06)
A20	1.11 (0.07)	4.52 (0.32)	6.54 (0.41)	0.14 (0.04)	0.26 (0.02)
S100	1.23 (0.05)	5.08 (0.14)	7.12 (0.38)	0.09 (0.04)	0.17 (0.04)
Control	1.05 (0.08)	4.29 (0.36)	6.30 (0.60)	0.11 (0.04)	0.28 (0.06)

[a] A100: all stumps treated at 100% of label dosage; A20: all stumps treated at 20% of label dosage; S100: visibly *P. weirii*-infected stumps treated at 100% of label dosage.

distributed evenly among treatments: (a) two plots, (b) three plots, (c) three plots, and (d) three plots.

2.7. Data collection and analysis

At tagging, height and dbh (1.4 m above the ground) of each plot tree were recorded. After tagging, plots were examined at intervals of 2–5 years for the next 10 years, tree mortality from LRR and height and diameter of live trees were recorded, and volunteer trees and competing brush were removed. Time interval was dependent on available resources. Final data collection was done in June 2005.

Five variables as plot means were analyzed to evaluate the effect of the treatments: (a) tree height at tagging, (b) final height of seedlings, (c) final diameter of seedlings, (d) mortality—the proportion of seedlings that died from LRR, and (e) mortality—the proportion of seedlings that died from all causes.

Analysis of variance (ANOVA) was used to test plot means for treatment effects, and multiple linear regression was used to test the utility of either II or a count of infected entities from the parent stand as a predictor of seedling mortality. Normality and homogeneity of variance were checked during analysis for all data sets by examining normal probability plots, and plots of the residuals (observed vs. predicted), respectively. To correct for nonnormality and unequal variance the mortality proportions were transformed by using an arcsine square root transformation, and the height and diameter data were transformed by using the natural log function before ANOVA evaluation. All statistical analyses were conducted with S-PLUS 6.1 (Insightful, 2002). Differences were considered to be significant when $p \leq 0.05$.

3. Results

A total of 1041 seedlings on 32 treatment plots were tagged, measured and observed for an additional 10 growing seasons. Although not analyzable owing to initial variable seedling density, data were recorded for about five seedlings from each plot that died from LRR before tagging. There was no obvious treatment bias. Of the 239 tagged trees that died, 118 died from LRR. Both mortality and seedling growth are summarized as treatment means (Table 1). There was no evidence of a treatment effect for any variable tested (Table 1).

We examined the possibility that multiple plantings had resulted in a bias in the measured growth of the seedlings. Seedlings from the original planting were not specifically marked; however, no plot received more than 17 seedlings in the three subsequent plantings. Therefore, we assumed that the tallest 15 trees on each plot were from the first planting and analyzed their mean height and dbh at final measurement by ANOVA. There was no evidence of a significant difference owing to treatment for either height ($p = 0.140$) or dbh ($p = 0.187$).

There was evidence of a significant difference in the LRR-caused mortality by block ($p = 0.0064$). Because there was no evidence that the treatments influenced mortality from LRR ($p = 0.676$), all 32 plots were used to test the ability of II or a simple count of infected stumps in the parent stand to predict the proportion of *P. weirii*-killed seedlings in the replacement stand. Testing through linear regression found that II is a significant predictor ($p = 0.0117$) of the proportion of *P. weirii*-killed seedlings (Pw prop): Pw prop $= -0.0294 + 0.0004$ II; however, the fit is poor: $r^2 = 0.1936$. There is no evidence that a simple count of infected stumps ($p = 0.1849$) predicts the proportion of *P. weirii*-killed seedlings in the replacement stand.

4. Discussion

This paper reports the results of the third and final study in a series that was undertaken to develop a postharvest chemical stump treatment to reduce the occurrence of LRR-caused mortality in the replacement stand. In the first study (Thies and Nelson, 1982), four fumigants used in agriculture (chloropicrin, allyl alcohol, Vapam and Vorlex) were applied at a common dose to holes in *P. weirii*-infested Douglas-fir stumps and eliminated the fungus from the stump and most roots. The second study (Thies and Nelson, 1987) built on the first by testing chloropicrin (effective at reducing the volume of wood occupied by *P. weirii* and easy to detect at low concentrations) and two other fumigants. Based on the sizes of stumps in the first study, a standard dosage of 6.7 ml of chloropicrin per kilogram of treated stump and root biomass was established for the second study. The second study refined application

techniques and demonstrated that a dosage of 3.3 ml of chloropicrin per kilogram of treated biomass eliminated *P. weirii* from the stump and most of the large root biomass. In a related study (Thies and Nelson, 1996), chloropicrin applied to living Douglas-fir at the same dosage eliminated *P. weirii* from most of the belowground biomass. To evaluate effectiveness of the fumigation applications in the first two studies, the stumps and root systems were excavated and evaluated for elimination of the fungus. In the third stump treatment study in the series, reported here, chloropicrin was applied operationally to a harvested infested stand; the stumps were left in the soil; and evaluation was based on the appearance of LRR-caused mortality in seedlings of the replacement stand.

Chloropicrin applied to stumps did not have a detectable impact on LRR-caused mortality in the replacement stand. In the earlier studies in this series the fungus was eliminated from the stump and most of the large roots, but some residual fungus remained alive. Stumps and roots in this study were not disturbed and attempts were not made to evaluate the volume or size of root biomass in which the fungus remained alive. We hypothesize that the fungus remained alive in small roots in enough locations to reintroduce the disease to the replacement stand. We find it curious that even though the fungus was likely eliminated from the stump and most of the large root biomass, there was no detectable reduction in the infection rate on the treated plots as compared to the nontreated plots.

Neither II (an indicator of the biomass of inoculum) nor a simple count of known infected stumps (an estimator of distribution or density) was a reliable predictor of LRR-caused mortality in the replacement stand. This demonstrates the difficulty of surveying for LRR and in using only known inoculum to predict appearance of LRR in the next stand. Further, the disease did not always reappear in the replacement stand where inoculum was known to be present in the parent stand. We found no evidence that chloropicrin influenced LRR-caused mortality on treated plots. Yet of 32 plots known to have inoculum present, the disease did not reappear on 8 (25%). This rate of failure of the disease to reappear is similar to the rate reported in a much larger study (Thies and Westlind, 2005). In that study, examination of all plots without a treatment effect found that of 94 0.04-ha plots with LRR in the parent stand, 29 plots (31%) were without LRR in the replacement stand.

As a predictor, II explains only a small amount of the variation in LRR-caused mortality, indicating that other variables may be important for predicting LRR infection. Our observations from this study and others (Thies and Nelson, 1997; Thies and Westlind, 2005) that reappearance of LRR is not predictable by either the presence or volume of known inoculum adds to our conviction that the infection process is more complex than previously thought and needs to be further examined to be well understood.

There was no evidence that the fumigant influenced the growth of the Douglas-fir seedlings. Growth of the seedlings as measured by their height in 1995 or their height and diameter in 2005 was not measurably influenced by the treatments. Even growth of the tallest 15 trees in each plot did not reflect a treatment effect.

Although we have evidence that chloropicrin remains in the treated belowground biomass for an extended time (Thies and Nelson, 1996), both this study and other observations from the same study area suggest that application of chloropicrin to stumps had a barely detectable impact on specific fumigant-sensitive flora and fauna. Chloropicrin-sensitive tomato and alfalfa seedlings, planted 2 m from stumps to monitor the release of chloropicrin from stumps, grew normally suggesting no significant release of chloropicrin in the first year. There was little impact on soil biota in the first growing season (Ingham and Thies, 1996), chloropicrin had little effect on surrounding vegetation in the first 3 years following application (Luoma and Thies, 1997), and 5 years after harvest and fumigation, no significant differences in vegetation composition or cover were found (Wilson et al., 1999). On seedlings planted adjacent to treated stumps neither the abundance nor type of ectomycorrhizae on Douglas-fir roots was significantly changed either in two growing seasons (Castellano et al., 1993) or in 4 or 5 years (Massicotte et al., 1998) after application of the fumigant chloropicrin. Seventeen months after application of chloropicrin, significant alteration of the invertebrate communities was detected in the immediate vicinity (ca. 2 m) of treated stumps, but not at a scale so small as a 0.02-ha plot (Moldenke and Thies, 1996). We conclude that application of chloropicrin at labeled rates has little detectable impact on the organisms living on the treated site. Given that chloropicrin is a toxic general biocide, we presume that this method of application causes the fumigant to be released slowly over such a long period that the concentrations are too low to have a significant impact on the diversity or population of nontarget organisms on the treated area.

5. Conclusions

In this study we found:

1. Applying chloropicrin to stumps at labeled rates did not reduce LRR-caused mortality in the replacement stand.
2. Both the relative inoculum potential (index) and a count of *P. weirii*-infected stumps in a parent stand may have little value in predicting LRR-caused mortality of Douglas-fir in a replacement stand.

Considering results from this study and others conducted on the same study area, applying chloropicrin to stumps at labeled rates did not have a significant impact on any of the organisms included in our observations.

Acknowledgements

The authors wish to thank the Simpson Timber Company for their efforts in establishing and maintaining the chloropicrin application study site and for access to their lands. We also thank Great Lakes Chemical Corporation (West Lafayette, IN) for supplying chloropicrin, advice, and equipment. We thank the National Agricultural Pesticide Impact Assessment Program for the support of this work as a part of study PNW-32.

Thanks are extended to Greg Brenner of Pacific Analytics for statistical consulting and support on this paper. We appreciate the effort and offer thanks to members of the field crews for 17 years of effort, especially crew leaders Mike McWilliams and Bob Merola. Finally, we thank Drs. Mike McWilliams, Pete Angwin, and Connie Harrington for reviews of earlier versions of this manuscript and several anonymous reviewers for careful consideration of the manuscript and useful comments.

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