

Survival of Douglas-fir Injected with the Fumigants Chloropicrin, Methylisothiocyanate or Vorlex

Abstract

Most Douglas-fir (*Pseudotsuga menziesii* (Mirb.) Franco) trees injected with the fumigants chloropicrin, methylisothiocyanate (MIT), or Vorlex were alive and growing 2 and 3 years following treatment. Fumigants were introduced through holes drilled near the base of the tree down into the root collar; the dosages approximated and bracketed those shown earlier to eradicate *Phellinus weirii* (Murr.) Gilb. from infested stumps and roots of harvested trees. After 3 growing seasons, 21 of 45 treated with chloropicrin and all 45 trees treated with MIT were still living. After two growing seasons, the crown condition of trees treated with Vorlex were found to be poorer than crowns of check trees while the crowns of trees treated with MIT were about the same as those of check trees. Although all the fumigants tested are broad spectrum biocides, most Douglas-fir survived the treatments at dosages previously demonstrated to eradicate *P. weirii* from stumps. Conventional wisdom regarding the use of phytotoxic chemicals to control plant pathogens may not always apply when application is made to the heartwood of a tree.

Introduction

Phellinus weirii (Murr.) Gilb., cause of laminated root rot, infects nearly all commercially important conifer species in western North America, where it reduces forest productivity by about 4.4 million m³ annually (Nelson *et al.* 1981). The disease, its distribution and impact, and current research on control have been discussed previously (Hadfield 1985, Nelson *et al.* 1981, Thies 1984).

Several soil fumigants have been reported to eradicate fungi from utility poles (Graham and Corden 1980), from infested wood buried in soil when applied to soil (Bliss 1951, Godfrey 1936, Houston and Eno 1969), and from stumps when placed in stumps (Filip and Roth 1977, Thies and Nelson 1982). Live Douglas-fir were shown to tolerate chloropicrin internally when eight chloropicrin-treated trees remained alive after 4 years and were reported to have growth comparable to that of control trees (Goodell *et al.* 1984).

The discoveries that chloropicrin could be used to eradicate *P. weirii* from infested stumps and roots and that trees can survive injection with this fumigant suggested the possibility of therapeutic application of fumigants to Douglas-fir infected by *P. weirii*. A major question remains: Can trees tolerate levels of fumigant necessary to kill wood-inhabiting fungi in roots and stems? In this paper we report findings 30 months (three growing seasons) after injecting Douglas-fir with the fumigants chloropicrin¹

or methylisothiocyanate (MIT), and 17 months (two growing seasons) after injecting Douglas-fir with Vorlex (v/v 20 percent MIT, 80 percent chlorinated C₃ hydrocarbons) and chloropicrin. Survival of the fungus, requiring destructive sampling of trees, can be determined only at the conclusion of this long term study or as individual trees die.

Methods and Materials

The study area, in the Oregon Coast Ranges near Apiary, Oregon (46° 1' N. latitude, 123° 4' W. longitude), had the following characteristics: elevation 420 m, slope 0-35 percent, mean annual precipitation 145 cm (U.S. Weather Bureau 1965), and Olympic silt loam soil. The 47-year-old, naturally regenerated stand of predominantly Douglas-fir growing on the site indicated a rating of site II (McArdle *et al.* 1949). Only dominant and codominant Douglas-fir, with clearly visible crowns, were selected for fumigation. Trees showing severe distress symptoms were rejected.

Selection of trees

Because tolerance to fumigants could depend upon presence and cause of stem and root decay, we selected trees in 3 classes of probable decay

¹This paper reports the results of research only. Mention of a pesticide does not constitute a recommendation for use by the U.S. Department of Agriculture, nor does it imply registration under The Federal Insecticide, Fungicide, and Rodenticide Act as amended. Also, mention of a commercial or proprietary product does not constitute recommendation or endorsement by the U.S. Department of Agriculture.

caused by *P. weirii*. Candidate trees were examined for the presence of *P. weirii* by carefully exposing major lateral roots within 60 cm of the base of the tree, examining the root surface for characteristic ectotrophic mycelium, and boring with an increment borer to detect stained or decayed root wood. Each tree was tagged, and measured, then, based on examination results, assigned to one of three infection classes: I, Infected; II, Probably infected, with crown symptoms and inoculum within 5 m but *P. weirii* not found on subject tree; III, Probably uninfected, no symptoms and no identified inoculum source within 25 m of subject tree.

Each infection class contained 45 trees separated into 5 groups of 9 based on similarities of diameter at breast height (dbh) and tree location. Selected trees ranged in dbh from 27.4 cm to 62.2 cm. Treatments were randomly assigned to trees within each group. Crown condition of individual trees was not recorded prior to treatment. As a result of the blocking and random assignment of treatments within blocks, we assumed that the average crown condition for the trees in each treatment was the same at the time of treatment.

Treatments

One of nine treatments was applied to each tree in the study: chloropicrin and MIT were applied at several dosages, and Vorlex was applied at a single dosage. Both chloropicrin and Vorlex have been shown to eradicate *P. weirii* from infested stumps (Thies and Nelson 1982). MIT is an ingredient in Vorlex. At room temperature Vorlex and chloropicrin are liquids, whereas MIT is a white, waxy solid. Holes drilled in the trees to apply the fumigant were part of the treatment. Check trees had neither holes drilled nor fumigants applied.

Test dosages were based on a dosage expected to kill *P. weirii* in treated wood. Earlier work (Thies and Nelson 1982) suggested that a dose of 6.7 ml of either chloropicrin or Vorlex per kg of stump and root biomass was sufficient to eradicate *P. weirii*. For this study, a standard dosage (D) was 6.7 ml of chloropicrin, or 6.7 ml of Vorlex or 1.5 g of MIT per kg of treated biomass. The dosage for MIT was based on the concentration of MIT in Vorlex (232 g MIT per 1.0 l Vorlex). For ease of application, the dosages

were rounded upward to the next quarter liter of chloropicrin or Vorlex and to the next even increment of 58 g of MIT. We assumed that the dose tolerated by a tree would vary linearly with the estimated biomass of its major roots and lower bole. The dose applied to each tree was based on treated biomass, estimated from the dbh. The nine treatments were defined in terms of standard dosages (Table 1).

TABLE 1. Number of live study trees and crown rating in mid-September 1984, following treatment with 3 fumigants in living Douglas-fir in western Oregon.

Treatment	Live trees ^a	Frequency of trees by Crown-Condition ^b				
		0	1-3	4-6	7-9	10
Applied March 1982:						
Check	14	1	1	—	8	5
Chloropicrin 1D ^c	3	12	1	—	2	—
Chloropicrin 0.5D	6	9	3	—	3	—
Chloropicrin 0.25D	12	3	5	1	6	—
MIT D	15	—	—	—	13	2
MIT 0.5D	15	—	—	—	13	2
MIT 0.25D	15	—	—	—	9	6
Applied April 1983:						
Chloropicrin 0.125D	15	—	—	—	13	2
Vorlex 0.5D	14	1	4	3	6	1

^aNumber of live trees remaining out of 15 trees treated.

^bCrown-Condition rating of live trees. Based on an 11-point scale where 0 = dead and 10 = vigorous.

^cD = standard dosage of 6.7 ml of chloropicrin or Vorlex, or 1.5 g of MIT per kg of treated biomass.

Each of the 9 treatments was applied to 15 trees, 5 trees in each of 3 infection classes. Treatments were applied in March 1982, except for chloropicrin 0.125D and Vorlex 0.5D, which were applied in April 1983.

Estimating biomass of wood treated

Based on fumigant movement of 2.4 m or more in poles (Graham and Cordon 1980) at wood moisture contents of about 30 to 40 percent (ovendry weight)—which is typical of heartwood of living Douglas-fir trees (Forest Products Laboratory 1973), we assumed that fumigant vapors would diffuse 2.4 m from the application holes.

We estimated treated biomass by 2.5-cm dbh classes from the following relationships: (1) Stem biomass (to 2.4-m height): $Y = 0.0007128 + 0.0002716 X$; where Y = stem volume in m^3 , X = basal area (breast high) in cm^2 ($n = 47$, $r^2 = 0.993$; sample trees ranged from 17.0- to 61.7-cm dbh). Data were from Douglas-fir in a single stand in the Coast Ranges near Apiary, Oregon (Thies, unpublished data). (2) Below ground stem and root biomass: $\ln Y = -4.6961 + 2.6929 \ln X$; where Y = below ground biomass in kg, X = dbh in cm, \ln = logarithms to the base e ($n = 26$, $r^2 = 0.96$; trees ranged from 2.3- to 23.0-cm dbh and from 94- to 135-cm dbh). Biomass was based on all roots having a diameter equal to or greater than 10 mm. Data were from two stands on the west slope of the Cascade Range (Gholz *et al.* 1979). Wood density was assumed to be 0.44 g per cm^3 . For example: a small tree with a dbh of 15 cm has an estimated treatable biomass of 34 kg and would receive a D dose of 0.25 l of chloropicrin or Vorlex, or 58 g of MIT; a large tree with a dbh of 60 cm has an estimated treatable biomass of 898 kg and would receive a D dose of 6.0 l chloropicrin or Vorlex, or 1362 g of MIT.

Application of fumigants

A series of equally spaced 3.2-cm-diameter holes was drilled in each tree receiving fumigant. Holes were drilled at approximately a 45-degree angle below horizontal and extending past the center of the tree. For the lowest dosage the holes were equally spaced around the tree approximately 30 cm above the litter layer. For larger dosages the holes were drilled 15 cm apart on a spiral that started approximately 15 cm above the litter layer and moved approximately 30 cm up the bole with each turn around the tree, for a maximum of two turns. A dose of chloropicrin or Vorlex was distributed (poured) equally into all holes in a tree; MIT was applied as a solid in small polyethylene sacks. Each sack was broken as it was pushed into a hole. Each hole was tightly plugged with a 12.5-cm-long by 3.3-cm-diameter hemlock dowel. Plugs were beveled at one end to facilitate driving them into the holes, then the beveled end was dipped into resorcinol glue (to resist passage of the fumigant through the dowel) and allowed to harden before use.

Data collection

The crown of each study tree was examined, photographed, and visually rated in mid-August

1982, mid-September 1983, and mid-September 1984. Particular note was made of crown condition. Crowns were rated on an 11-point scale of crown condition (C-C), from dead (0) to vigorous (10). The rating was based on needle complement, needle length and color, number and distribution of dead needles and branches, internodal growth, and number and growth of new branches.

In October 1983 and September 1984 dead subject trees were felled and their stumps and roots removed and cleaned. All roots recovered were cut at 30-cm intervals. When stain or decay typical of *P. weirii* infection was found a 5-cm thick disk was removed. Each disk was split longitudinally and five chips were removed aseptically from the split face in areas of typical stain or advanced decay and placed on 1.5% malt agar slants in culture tubes. Culture tubes were incubated for 14 days at room temperature. Presence of *P. weirii* was determined from morphological features of developing colonies (Nelson 1975).

Results

Of 120 Douglas-fir treated with fumigants, 95 trees were still alive at the time of our evaluation in mid-September 1984. After three growing seasons (30 months), trees treated in March 1982 showed differences in crown condition among treatments. At the end of the first growing season there were no dead trees and only relatively minor indications of a decline in crown condition, but by the end of the second growing season, 22 trees had died and differences among treatments in appearance of the crowns had become evident. By the end of the third growing season 25 treated trees were dead (Table 1) but crowns of remaining trees showed little change in crown condition from the second growing season.

All trees treated with 0.5D Vorlex or 0.125D chloropicrin in April 1983 were alive in September 1983. After one growing season, crowns of Vorlex-treated trees but not chloropicrin-treated trees were rated as less vigorous than crowns of check trees. At the end of the second growing season crown condition of chloropicrin-treated trees and check trees was unchanged. Trees treated with Vorlex had average crown ratings lower than checks and one of the 15 treated trees was dead. Vorlex at 0.5D and MIT

at 0.5D (administered 1 year earlier), contained the same amount of MIT, but after three growing seasons all 15 trees receiving MIT at 0.5D were alive and they had better C-C ratings than trees receiving Vorlex at 0.5D.

We can draw conclusions about the relative toxicity of the chemicals to the trees; however, since we have not examined the roots of the living treated trees, we are unable to determine if dosages non-lethal to trees were effective in killing wood inhabiting fungi.

Isolations attempted from stumps and roots of 26 dead trees recovered *P. weirii* from all disks from the check tree, but from only 22 percent of the root disks from the 25 chloropicrin- or Vorlex-treated trees. However, since we have not examined the roots of 94 of the treated trees we are unable to compare the individual treatments based on reduced viability of *P. weirii* inoculum.

We did not find meaningful differences in mortality or crown conditions among infection classes.

Discussion

Although all the fumigants are toxic, broad spectrum biocides, 73 percent of the treated trees were still living three growing seasons after treatment. Chloropicrin appeared more toxic to Douglas-fir than MIT: of 25 treated trees that died, 24 were treated with chloropicrin and there had been a decline in the crown condition of surviving trees. In contrast, after three growing seasons the crown condition of MIT-treated trees did not appear different from the check trees.

Although limited to two seasons of exposure, crown condition of check trees was similar to trees treated with chloropicrin at the lowest dosage (0.125D). This dosage approximates the highest dosage tested in an earlier study (Goodell *et al.* 1984) in which eight chloropicrin-treated

healthy Douglas-fir survived and attained growth comparable to check trees 4 years after treatment.

For a given dosage of MIT, there was more adverse impact on Douglas-fir if the fumigant was delivered as Vorlex rather than as MIT. Because we did not apply the Vorlex carrier as a treatment without the MIT, we cannot rule out the possibility of a synergistic effect on the tree. Nevertheless, if our primary goal is to maintain or improve the health of the tree, and the stand in general, then MIT alone was less harmful.

Although limited to the roots of killed trees, isolation results confirmed that fumigants can reduce *P. weirii* in roots of Douglas-fir trees as well as stumps. A comparison of treatment effectiveness will not be possible until all trees in the study can be felled and their roots examined.

The results from this study suggest that host trees can be treated with fumigant in quantities large enough to expect a reduction in inoculum potential of a root rot pathogen without killing the host. These results encourage additional research to identify other potential fumigants and to determine the efficacy of any fumigant to reduce residual inoculum.

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