valves to another water tank and the water supply was monitored with an automatic recording system. This experiment was not successful since resin or other exudates sealed the cut sapwood surface and prevented water uptake. We had not experienced this problem before and recutting the surface did not help. The time of cutting (mid-August) or the complete disruption of sapwood may have been contributing factors.


Tree vigor and stand growth of Douglas-fir as influenced by laminated root rot

RAM OREN
Department of Forest Science, College of Forestry, Oregon State University, Corvallis, OR, U.S.A. 97331

WALTER G. THIES
United States Department of Agriculture, Forest Service, Pacific Northwest Forest and Range Experiment Station, Corvallis, OR, U.S.A. 97331

AND

RICHARD H. WARING
Department of Forest Science, College of Forestry, Oregon State University, Corvallis, OR, U.S.A. 97331

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Total stand sapwood basal area, a measure of competing canopy leaf area, was reduced 30% by laminated root rot induced by Phellinus weirii (Murr.) Gilb. in a heavily infected 40-year-old coastal stand of Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) compared with that of a similar uninfected stand. Annual basal area increment per unit of sapwood area, an index of tree vigor, was expected to decrease in uninfected trees in the infected stand as surrounding trees died from root rot; vigor of the uninfected trees did increase by an average of 30%, offsetting the reduction in canopy leaf area. This increase, although less than might be expected in an evenly spaced thinned stand, was sufficient to maintain stand basal area growth at levels similar to those of unthinned forests. These findings indicate that increased growth by residual trees must be taken into account when the impact of disease-induced mortality on stand production is assessed.

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Dans un peuplement côte de douglas (Pseudotsuga menziesii (Mirb.) Franco) âgé de 40 ans et fortement infecté par Phellinus weirii (Murr.) Gilb., la surface terrière totale d’aulbier, une mesure de la surface foliaire du couvert en compétition, fut réduite de 30% par la pourriture racinaire, par comparaison avec un peuplement témoin. L’accroissement annuel en surface terrière par unité de surface d’aulbier, un indice de la vigueur des arbres, devait normalement augmenter chez les arbres sains du peuplement infecté, à mesure que les arbres abaisonnains mourraient de la pourriture racinaire. La vigueur des arbres non infectés s’est accrue d’environ 30%, compensant pour la réduction de la surface foliaire du couvert. Bien qu’intérieure à celle à laquelle on pouvait s’attendre dans un peuplement éclairci d’aulbier homogène, cette augmentation fut suffisante pour maintenir la croissance en surface terrière à des niveaux semblables à ceux des forêts non éclaircies. Ces observations indiquent qu’on doit tenir compte de la croissance accrue des arbres résiduels lorsqu’on veut déterminer l’impact de la mortalité par maladie sur la production du peuplement.

### Introduction

Short-term detrimental effects of disease and insects on the growth of individual trees or entire stands have been documented (Mattson and Addy 1975; Froelich et al. 1977; Thies 1983). Over the longer term, however, a reduction in canopy as a result of pathogen-caused mortality could induce a thinning response among the surviving trees. In some stands where insect attacks have not been followed by control efforts or salvage logging, growth rates of remaining or reestablished trees have actually surpassed those in unattacked stands (Mattson and Addy 1975; Wickman 1978, 1980; Gausser et al. 1983).

We hypothesized that healthy trees in a forest affected by root rot would exhibit increased vigor, as indicated by wood production per unit of leaf area (Waring et al. 1980), because of reduced competition. To test our hypothesis, we compared the vigor of healthy Douglas-fir (Pseudotsuga menziesii (Mirb.) Franco) trees growing in a stand infected with Phellinus weirii (Murr.) Gilb., the causal agent of laminated root rot, with that of trees in a similar, uninfected forest.

### Site description and selection

The main study site (stand A) was a 0.55-ha stand of 40-year-old Douglas-fir located in the Oregon Coast Range (46° N, 125° W). Soil is Olympic silt loam, elevation is 400 m, and slope is 15%. In this maritime climate, annual precipitation exceeds 1500 mm, less than 10% of which falls during summer. Temperatures average 10°C, the minimum rarely falling much below freezing. The growing season is long, extending from April through September (Franklin and Dymess 1973). Such an environment fosters rapid tree growth; the site index (projected height growth at 100 years) was 52 m (Waring et al. 1980).

The stand mortality resulting from laminated root rot occurred in scattered pockets. Some areas had full canopy, whereas others had large openings, providing a wide range of densities. At the time of comparison, stocking in stand A averaged 280 trees/ha, with a total basal area of 30 m²/ha (Waring et al. 1980).

As a control, we used a 0.5-ha Coast Range site (stand B) of similar age, composition, and site index, but without detectable signs of laminated root rot.

### Methods

In stand A, all trees larger than 10 cm in diameter at breast height (1.37 m), totalling 157, were identified with tags, their diameters measured, and their positions mapped (Thies and Hoopes 1979). All trees were then felled in the dormant season and their heights were recorded. A disk was cut from each tree at breast height and the thickness of bark, sapwood, and most recent growth ring was determined by averaging measurements along three radii. The sapwood–heartwood boundary was confirmed by a distinct color difference following application of pH sensitive stain (Kutsch and Sachs 1962).

In a previous study, Thies (1983) determined which trees in this stand had some degree of laminated root rot by excavating the main roots of all trees with a bulldozer, washing the roots, and then examining them for decay or ectotrophic mycelium typical of P. weirii (Backlund et al. 1954; Thies 1984). Infection on stumps or roots was confirmed by culturing material collected from stained or decayed areas and observing specific morphological features (Nelson 1975). Thies (1983) reported that 29% of the trees were infected and exhibited a cumulative reduction of about 10% in height and diameter growth over the last decade.

In stand B, standing trees were visually assessed as healthy and no dead trees or infected stumps were noted within 20 m of the stand.

Canopy competition may be indicated by total canopy leaf area or, because of the correlation between leaf and sapwood areas (Waring et al. 1982), by the sum of the sapwood basal area at the base of the live crown for all trees in a specified area. The shade from ground to live crown varies among trees, depending on dominance class and stand history. In this study, we lacked data on distance to the base of the live crown and could not estimate the sapwood area, so we quantified competition in terms of the sum of the sapwood basal area at breast height for all trees in a plot or stand (complete sapwood basal area; square metres per hectare) for stands A and B. Individual-tree vigor was expressed as the ratio of current annual basal area increment to sapwood basal area for each of the 104 uninfected trees evaluated in stand A (Waring et al. 1980). For both stands A and B, the annual stand growth was expressed as current increase of basal area (in square metres) of all trees in the stand per hectare per year.

We first delineated at random 20 circular 200-m² plots on the map of stand A (Fig. 1) and related the average vigor of healthy dominant and codominant trees in each plot to the total competing sapwood basal area of surrounding trees on that plot. As a variant to random selection, we evaluated the vigor of each healthy tree positioned at the center of its own 200-m² plot. Data for the randomly and nonrandomly selected plots were grouped separately and both data sets were analyzed with least-squares regression techniques. The analyses considered possible differences between dominant and codominant, intermediate, and suppressed.

### Results and discussion

The total sapwood basal area in stand A was nearly 30% lower than that in stand B (10.9 vs. 15.4 m²/ha; Table 1). Nevertheless, the annual stand growth of stand A was within

### Table 1. Characteristics of a healthy Douglas-fir stand (stand B) contrasted with those of a stand with mortality induced by laminated root rot (stand A)

<table>
<thead>
<tr>
<th>Stand</th>
<th>Basal area (m²/ha)</th>
<th>Sapwood basal area (m²/ha)</th>
<th>Basal area growth (m² ha⁻¹year⁻¹)</th>
<th>Growth efficiency (m² year⁻¹ m⁻² sapwood)</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>30</td>
<td>10.9</td>
<td>0.75</td>
<td>0.07±0.002</td>
</tr>
<tr>
<td>B</td>
<td>57</td>
<td>15.4</td>
<td>0.77</td>
<td>0.05±0.004</td>
</tr>
</tbody>
</table>
Fig. 1. Map of individual tree locations in stand A. Random and nonrandom 200-m² circular plots were distributed over six quadrants (0.55 ha); of the three plots shown, one is random (dashed circle) and the other two are nonrandom (solid circles; note tree in center of each). Relative sizes of circles reflect differing tree diameters and dominance classes; ×, dead tree. In total, the tree vigor of 194 uninfected trees of all dominance classes was evaluated.

3% of that of stand B, largely because tree vigor averaged nearly 30% greater in stand A (Table 1).

Tree vigor and competing sapwood basal area were not significantly related ($p < 0.05$) when growth responses to release were compared for dominant and codominant trees in stand A. Surprisingly, we found that vigor of dominant and codominant trees was not significantly related ($p < 0.05$) to competing sapwood basal area, regardless of where trees were located within the 200-m² plots.

We sought to understand why a relationship between tree

Fig. 2. Tree vigor (BA1/SA) of an uninfected, 50-year-old Douglas-fir stand in Dunn Forest near Corvallis, OR, increases as the competing sapwood basal area (SSA) decreases. Values are means of trees on 200-m² plots (R. H. Waring, unpublished data).

Fig. 3. Tree vigor (BA1/SA) increases as competing sapwood basal area (SSA) decreases for (a) the '04 individual trees in stand A and (b) an uninfected, 36-year-old, experimentally thinned Douglas-fir stand near Hoskins, OR (after Waring et al. 1981). The lower curve (a) represents the maximum potential tree vigor ± absolute measurement errors (Oren 1981) for the individual trees; the upper curve (b) represents mean values for 30 trees.

vigor and competing sapwood basal area was lacking. A good relationship between the two was evident in an uninfected, 50-year-old, 0.5-ha Douglas-fir stand in Dunn Forest near Corvallis, OR, whose site index was poorer (35 m) than that of stand A (R. H. Waring, unpublished data) (Fig. 2). The ongoing thinning resulting from pathogen-induced mortality might keep a stable relationship from developing because the canopy would be continuously changing. In examining release further, we analyzed only trees with the highest vigor for a given level of competing sapwood basal area (Fig. 3). We found that tree vigor in stand A declined as competition increased (lower curve, Fig. 3) in a manner similar to that reported for a 36-year-old, experimentally thinned, 1.2-ha Douglas-fir stand near Hoskins, OR, with a similar site index (52 m) (Waring et al. 1981; Fig. 3, b).

We do not know exactly how long it takes for a tree to fully respond to release from competition, but few trees in stand A had reached their potential at the time of harvest, as suggested by the upper boundary for vigor (Fig. 3, a). Nevertheless, the annual basal area growth in stand A did increase over the decade before harvest, elevating current wood production to a level comparable with that of stand B, which had 20% higher stocking and a 30% denser canopy. The harvestable material lost to laminated root rot, while measurable, might be equivalent to that from density-related mortality. However, our findings indicate that stand growth after release partially offsets mortality and must be taken into account so that losses associated with the disease are not overestimated. Frequent commercial thinning, as practiced in the experimentally thinned stand of Fig. 3, notably increased stand growth and could improve tree vigor sufficiently to reduce substantial losses attributed to laminated root rot observed in unmanaged stands.