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# Secondary Resin Production Increases With Vigor of *Abies grandis* Inoculated With *Trichosporium symbioticum* in Northeastern Oregon

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## Abstract

Thirty grand fir (*Abies grandis* (Dougl. ex D. Don) Lindl.) trees were artificially inoculated with the fungal symbiont, *Trichosporium symbioticum* Wright, to simulate attack by the fir engraver beetle, *Scolytus ventralis* LeConte. Fifteen months after treatment, secondary resin production, necrotic lesion formation, and percentage of wounds with *T. symbioticum* were determined in relation to tree vigor and inoculation density. Resin production was significantly greater in high-vigor trees but was not affected by inoculation density or degree of fungal colonization. Lesions from inoculated wounds were significantly longer than lesions from uninoculated wounds. Neither lesion size nor percentage of wounds with *T. symbioticum* were affected by tree vigor or inoculation density. This study did not establish a threshold of successful attack in grand fir nor did any trees die as a result of treatment. This study is the first attempt to measure secondary resin response in grand fir in relation to two quantified measures of tree vigor and three levels of inoculation density.

Keywords: *Abies grandis*, *Scolytus ventralis*, tree vigor.

## Introduction

When the phloem of coniferous trees is infected with certain fungi, a distinctive reaction zone is formed around the point of infection (Berryman 1969, Reid and others 1967). Within this zone of hypersensitive wound reaction, the parenchyma cells die, and the phloem tissues become impregnated with secondary resin. Provided the reaction is intensive enough, the fungus is arrested within the reaction zone and hence cannot infect surrounding tissues (Wong and Berryman 1977). The resin-impregnated zone may also include some of the underlying xylem.

Some experiments have been done in lodgepole pine (*Pinus contorta* Dougl. ex Loud.) and Norway spruce (*Picea abies* (L.) Karst.) that address how many infections are needed to exhaust this defensive response of the trees; that is, the threshold of successful attack by the fungus. In lodgepole pine, Raffa and Berryman (1983) infected a 30-dm$^2$ area of the stem bark with 2, 4, 8, 16, and 32 inoculations (0.07, 0.13, 0.27, 0.53, and 1.07/dm$^2$, respectively) of the fungus *Europhium* (=*Ceratocystis*) *clavigerum* Robinson and Davidson and measured the amount of secondary

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resin formed in response to the infection. In trees with the highest inoculation densities (0.53 and 1.07/$dm^2$), the quantity of resin per wound was reduced; at the highest density, even the total resin response decreased.

In Norway spruce, Christiansen (1985) inoculated the entire surface of a 0.6-m-long stem section with *Ophiostoma polonicum* Siem. (=*Ceratocystis polonica* (Siem.) C. Moreau) by using 1, 2, 4, and 8 inoculations per $dm^2$. With the two highest densities, a gradual reduction of the resin concentration in the reaction zones was observed; it was accompanied by an increasing success of the fungus in entering the sapwood of the trees. Suppressed trees had less resistance than dominant ones. The experiment indicated that resistance could be related to relative stemwood growth.

Although no specific experiment has been done in grand fir to establish a similar threshold of successful attack, the work of Wright (1935) with *Trichosporium symbioticum* Wright[1] gives a clue to the amount of infection necessary to kill a fir tree. Wright, working with *Abies concolor* (Gord. and Glend.) Lindl.), killed 3 of 10 small trees (diameter breast height [d.b.h] 5-13 cm) after 16 months with the equivalent of 38 to 75 cork-borer inoculations for a tree 20 cm in d.b.h. Inoculation experiments with *T. symbioticum* have also been carried out by Raffa and Berryman (1982a) to clarify basic traits of the hypersensitive wound reaction in conifers. We do not know of experiments or observations showing that this fungus can infect grand fir trees and actually cause their death. Raffa and Berryman note, however, that the wound response to as few as five inoculations per tree was weaker in susceptible trees (that is, trees later killed by *Scolytus ventralis* Le Conte/*T. symbioticum*) than in resistant ones. Suppressed trees responded less to the infection than did dominant or codominant trees.

Objectives of our study were to (1) establish how many fungal inoculations grand fir trees of widely different vigor can tolerate before their capacity for producing protective secondary resin is reduced and they die; and (2) determine potential effects of tree vigor and inoculation density on secondary resin formation, necrotic lesion formation, and percentage of wounds with *T. symbioticum.*

## Materials and Methods

The study area was about 20 km northwest of Elgin, Oregon, on land managed by the Walla-Walla Ranger District of the Umatilla National Forest. The study area is about 1500 m elevation and is an *Abies grandis/Vaccinium membranaceum* plant community (Johnson and Simon 1987).

In May 1986, *Trichosporium symbioticum* was isolated from pupal chambers of *Scolytus ventralis* in grand fir about 40 km from the study area. The fungus was cultivated on 2 percent malt agar in petri dishes.

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[1] The taxonomy of this species is in question and the fungus probably will be reassigned to the genus *Leptodontium* (personal communication, David Brayford, CAB International Mycological Institute, Kew, Surrey, UK).

Within the study area, 30 grand fir trees were selected in May 1986. Diameter at 1.4 m (d.b.h.) averaged 19.3 cm (range 14.7-26.9). Trees with obvious damage or wounds were not selected. The stand has had light defoliation from western spruce budworm (*Choristoneura occidentalis* Freeman) for the past 2 years. Half of the trees had high vigor (>60 g stemwood/m$^2$ of foliage); the other half low vigor (<25 g/m$^2$), as determined in the field. Tree vigor was estimated by measuring the ratio of current basal area growth to sapwood area as an estimator of stemwood-volume production per unit of leaf area (Waring and others 1980). Current basal area growth and sapwood basal area were calculated after measuring the width of the current growth ring and the width of the sapwood from two increment cores collected at breast height.

Tree vigor also was estimated using a pulsed electric current to measure present cambial electrical resistance (CER). Trees with low electrical resistance are more vigorous than trees with high resistance (Shortle and others 1977, Wargo and Skutt 1975). CER was measured with a Model OZ-67 Shigometer.[2] For each tree, CER was measured at four points around the stem at breast height, and an average CER was calculated for each tree. All CER measurements were taken during the active growing season (July) on consecutive days at about the same time each day to avoid any variation in CER due to temperature.

The entire surface of a stem section 0.8 m long (between 1.1 and 1.9 m above-ground) was marked on each tree. Within these markings, each tree was treated with either 0.5, 1.5, or 2.5 inoculations per dm$^2$, evenly spaced over the bark surface. In a tree 20 cm in d.b.h., the inoculated bark surface amounted to $2.0 \times 3.14 \times 8$ = ca 50 dm$^2$, and the total number of inoculations were about 25, 75, and 125 per tree, respectively. Thus 25 inoculations/50 dm$^2$ = 0.5 inoculations/dm$^2$. Each inoculation density included five high-vigor and five low-vigor trees.

Inoculating was done in July during the most intensive period of growth and the main flight period of *S. ventralis* (Raffa and Berryman 1982a). Holes 4 mm in diameter were made to the cambial zone with a cork borer (Wright 1935). A small piece of agar from the most active growth zone of *T. symbioticum* was inserted into each hole, and the bark plug was returned to its original position. On each tree, five additional inoculations were made as above, but each hole was inoculated with sterile agar. The cork borer was sterilized with 95 percent ethanol but not flamed between inoculations. The five sterile inoculations were in a row about 10 cm above the band of fungal inoculations.

In October 1987, after 15 months or two growing seasons, sample trees were examined for secondary resin production, lesion formation, percentage of wounds with *T. symbioticum*, tree mortality, and attack by *S. ventralis.* The length of resin issuing from 10 randomly selected inoculation wounds and all 5 uninoculated wounds was measured for each tree. Resin length (nearest mm) was measured from the point of inoculation to the lowest point of resin flow on the bark. Also, we removed the outer bark from the same 10 inoculated wounds per tree and the 5 uninoculated

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[2] Shigometer is a registered trademark of the Osmose Wood Preserving Co. of America, Inc.

wounds. The length (nearest mm) and width of each necrotic lesion was measured. Each of the 15 lesions was then excised from the tree, and the entire lesion was rinsed with sterile water and aseptically placed on 2 percent malt agar to attempt to recover *T. symbioticum*.

For both inoculated and uninoculated wounds, mean resin length per tree, the percentage of wounds with *T. symbioticum* per tree, and mean lesion length, width, and area per tree were subjected to a two-way analysis of variance to detect significant treatment differences and interactions between tree vigor class and inoculation density using the MINITAB program (Ryan and others 1985). Analysis of variance was also used to detect significant differences between inoculated and uninoculated wounds for (1) resin length, (2) lesion length and width, and (3) percentage of wounds with *T. symbioticum*. Percentages were analyzed after arcsin transformation. Regression analysis was used to develop equations and determine coefficients of determination among several dependent and independent variables.

## Results and Discussion

### Tree Mortality and Fir Engraver Attack

Fifteen months (two growing seasons) after inoculation, none of the 30 trees had died or showed evidence of attack by *S. ventralis*. This differs from the observation of Wright (1935) where 3 of 10 trees died 16 months after treatment; all dead trees had been attacked by *S. ventralis*. We used more inoculations than did Wright, but failed to kill trees. The most likely explanation lies in the spatial arrangement of the inoculations.

Wright placed his inoculations in rings around the trunk with a distance of only 1.3 to 2.5 cm between the cork borer holes. This produced a girdling effect; the stains coalescing into a solid band extending completely around the trunk, forming an effective girdle.

*Trichosporium symbioticum* does not penetrate the sapwood of grand fir trees, and the defence reaction is also confined to the phloem and cambium and the outermost annual ring of the xylem (Berryman 1969). For a tree to die, the fungus-infected areas and wound lesions have to coalesce around the entire stem to produce a complete girdle that will effectively block the flux of assimilates from the crown. This will then inevitably lead to a starvation of the roots and the slow death of the tree. As noted by Berryman (1972), the horizontal maternal gallery of the fir engraver is a more significant contribution to a full girdle than is a vertical gallery.

Other fungi (for example, *O. polonicum* which attacks Norway spruce) may penetrate the entire sapwood within a short period of time and effectively block the water transportation to the crown, leading to rapid death of the tree (Horntvedt and others 1983). This fungus can grow under extremely oxygen-poor conditions, compared to other Ophiostomaceae, for example, and is able to penetrate sapwood with a high water content.[3]

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[3] Personal communication, H. Solheim, forest pathologist, Norwegian Forest Research Institute, Division of Forest Protection, Section of Forest Pathology, P.O. Box 62, 1432 Ås-NLH (Norway).

Other reasons for our different results may be the tree species involved (*A. concolor* vs. *A. grandis*); tree size inoculated (5-13 cm vs. 16-26 cm d.b.h.); the isolate of *T. symbioticum* used (California vs. Oregon); or tree vigor. Wright reports that two killed trees had medium vigor and one had poor vigor. No quantitative vigor measure was given.

## Wound Resin Production

Resin exudation was positively correlated ($F=34.43$, $P<0.01$) with tree vigor. Resin length showed some correlations with vigor readings using basal area growth/sapwood area ($r^2=0.55$) and pulsed electric current ($r^2=0.37$). Vigor using basal area growth/sapwood area showed good correlations with vigor using a pulsed electric current ($r^2=0.67$). Exudation of resin on the bark surface is a sign of an active wound response (Berryman 1969). Semiquantitative measures of exudation have been used as indicators of the resin concentration of the reaction zone (Christiansen 1985, Christiansen and Ericsson 1986, Horntvedt 1988). Resin concentration or concentration of chemical constituents such as monoterpenes is crucial for the efficiency of the wound reaction (Christiansen 1985; Raffa and Berryman 1982a, 1983). The efficiency of the reaction seems related to tree vigor (Christiansen 1985; Raffa and Berryman 1982a, 1982b). Our results agree with this general description of the defense reaction.

Our study showed no significant changes in resin exudation with inoculation density (table 1). This is probably because even the highest inoculation density of our experiment never reached the threshold of successful attack. Studies have indicated that a tree's capacity for forming secondary wound resin depends on the translocation of current photosynthate to the infected area (Christiansen and Ericsson 1986, Miller and Berryman 1986, Raffa and Berryman 1982a, Wright and others 1979). During the growth season, keen competition for carbon among a multitude of different sinks in the tree probably occurs; several of these sinks may have a higher priority than production of defensive chemicals. Field studies have confirmed the existence of a threshold of successful attack for bark beetles. At this number of infections, the resin concentration of the individual reaction zones decreases, which is paralleled by increasing fungal invasion (Christiansen 1985, Raffa and Berryman 1983). The threshold seems dependent on tree vigor (Christiansen 1985, Mulock and Christiansen 1986, Waring and Pitman 1985), tree age, and degree of suppression (Raffa and Berryman 1982a). In some of these experiments, the vigor of the trees was manipulated by silvicultural treatments, such as thinning and fertilization.

## Wound Lesion Formation

We found that lesion development occurred both in inoculated and uninoculated wounds similar to those described by Raffa and Berryman (1982a) and Wong and Berryman (1977). Lesions from inoculated wounds were significantly ($F=8.03$, $P<0.01$) longer but not wider than lesions from uninoculated wounds (table 2, fig. 1). We found no correlation between resin length and either lesion length or width. This agrees with the observations of Raffa and Berryman (1982a) who state that "lesion formation and terpene synthesis represent at least two independent activities during the wound response."

**Table 1—Effects of tree vigor and inoculation density on secondary resin production, lesion formation, and percentage of wounds with *Trichosporium symbioticum* (Ts) in 30 grand fir trees in northeastern Oregon**

| | | Number of inoculations per dm$^2$ | | | | | |
|---|---|---|---|---|---|---|---|
| | | Low | | Mid | | High | |
| Tree vigor[a] | Wound characteristics | 0.5[b] | sd | 1.5 | sd | 2.5 | sd |
| Low | | | | | | | |
| | Resin length (mm) | 13 | 13.2 | 13 | 4.5 | 40 | 22.5 |
| | Lesion length (mm) | 32 | 15.1 | 40 | 35.0 | 37 | 8.0 |
| | Lesion width (mm) | 13 | 1.6 | 11 | 1.3 | 12 | 2.4 |
| | Wounds with Ts (%) | 100 | 0 | 98 | 4.5 | 96 | 8.9 |
| High | | | | | | | |
| | Resin length (mm) | 115 | 57.7 | 80 | 16.8 | 67 | 35.3 |
| | Lesion length (mm) | 30 | 9.8 | 30 | 5.1 | 31 | 8.2 |
| | Lesion width (mm) | 15 | 2.8 | 15 | 3.0 | 12 | 1.5 |
| | Wounds with Ts (%) | 88 | 26.8 | 92 | 11.0 | 84 | 26.1 |

[a] Low vigor: stemwood growth/leaf area <25g/m$^2$ or cambial electrical resistance >9 Kohm. High vigor: >60g/m$^2$ or <8 Kohm. Each mean represents 5 trees, 10 wounds/tree.

[b] 25 inoculations/50 dm$^2$ = 0.5/dm$^2$; sd = standard deviations.

**Table 2—Characteristics of 30 grand fir trees by inoculation density class (dens) and tree vigor class (vig) with artificial inoculations by *Trichosporium symbioticum* (Ts) in northeastern Oregon**

| Treatment density and vigor[a] | DBH | Growth/ area | CER | Inoculated wounds[b] | | | | Uninoculated wounds | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|
| | | | | Ts | Resin length | Lesion length | Lesion width | Ts | Resin length | Lesion length | Lesion width |
| | cm | g/m$^2$ | Kohm | % | - - - - - - - mm - - - - - - - | | | % | - - - - - - - -mm - - - - - - - | | |
| Low/low | 16.8 | 20.7 | 13.2 | 100 | 13 | 32 | 13 | 84 | 21 | 25 | 16 |
| Low/high | 20.9 | 75.3 | 6.2 | 88 | 115 | 30 | 15 | 76 | 100 | 26 | 16 |
| Mid/low | 18.3 | 17.7 | 12.8 | 98 | 13 | 40 | 11 | 78 | 9 | 20 | 12 |
| Mid/high | 20.8 | 74.0 | 5.9 | 92 | 80 | 30 | 15 | 87 | 96 | 26 | 15 |
| High/low | 18.6 | 16.6 | 11.2 | 96 | 40 | 37 | 12 | 84 | 38 | 25 | 12 |
| High/high | 20.3 | 73.9 | 6.7 | 84 | 67 | 31 | 12 | 45 | 84 | 27 | 14 |
| Mean | 19.3 | 46.4 | 8.6 | 93 | 55 | 33 | 13 | 76 | 58 | 25 | 14 |

[a] Low, mid, and high inoculation densities = 0.5, 1.5, and 2.5 inoculations/dm$^2$ of bark, respectively. Low vigor: stemwood growth/leaf area< 25g/m$^2$or cambial electrical resistance >9 Kohm. High vigor: >60g/m$^2$; <8 Kohm. Each treatment represents 5 trees.

[b] 10 wounds per tree sampled for inoculated wounds; 5 for uninoculated wounds.

Figure 1—Lesions (arrow) formed by artificial inoculation with *Trichosporium symbioticum* on grand fir.

We found no significant differences in lesion length or width between vigor classes or among inoculation density classes. With an increasing number of inoculations, the lesion size will probably remain unchanged until the threshold of successful attack is approached. Then the fungus can break out of the "enclosures" erected by the tree and spread to a larger area of phloem and xylem (Christiansen and others 1987). In this study, however, this level of infection was not reached.

## Percentage of Wounds with *T. Symbioticum*

*Trichosporium symbioticum* was recovered from 93 percent of the inoculated wounds 15 months after treatment. Even though fungal recovery differed significantly (F=12.47, P<0.01) between inoculated and uninoculated wounds, *T. symbioticum* was recovered from 76 percent of the uninoculated wounds. We speculate that (1) because wounds were not sealed with tape, they became contaminated or (2) our inoculation technique was not adequate to prevent contamination. No significant differences occurred in percentage of wounds with *T. symbioticum* (either inoculated or uninoculated wounds) between tree vigor classes or among inoculation density classes.

Although most of the lesions in our study had viable fungus after 15 months, in all cases the host appeared to compartmentalize the fungus. This is in agreement with Wong and Berryman (1977) who state that "resinous and phenolic materials which rapidly accumulate in the reaction zone do not drastically affect the viability or pathogenicity of the fungus." Exactly what causes the fungus to spread laterally to kill part or all of the cambium as observed by Wright (1935) is unknown. Because *T. symbioticum* spreads more rapidly along the vertical axis than radially or circumferentially, overlapping horizontal wounds (as occur naturally) rather than point wounds seem potentially to be a more lethal method of inoculation. This agrees with the suggestion that a horizontal bark beetle gallery secures a better tangential spreading of the fungal symbiont (Berryman 1972).

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