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EFFECTS OF NUTRIENT AND LIGHT LIMITATION ON MOUNTAIN HEMLOCK: SUSCEPTIBILITY TO LAMINATED ROOT ROT[1]

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Abstract. Mountain hemlock forests in the Oregon Cascades exhibit wave-form dieback resulting from infection by laminated root rot (*Phellinus weirii*). Although *Phellinus* remains viable in dead roots after the wave of dieback passes, many regenerating mountain hemlock forests do not become immediately reinfected. We measured at least a doubling of nitrogen availability in the dieback and regrowth zones, and thought that this increased availability could improve tree resistance to the fungus. To test this hypothesis, we grew small mountain hemlocks under nutrient and light limitations in a growth room, and then inoculated them with the fungus. Trees growing without added nutrients had significantly greater foliage damage and mortality after *Phellinus* inoculation than did trees growing with nutrients. Shading significantly increased susceptibility whether or not nutrients were added. We believe that increased nitrogen availability and possibly increased light levels after dieback in the field act similarly to increase resistance and prevent reinfection of the regrowing stands.

Foliage damage and susceptibility to infection were related to pool sizes of total nitrogen, phosphorus, and nonstructural carbohydrates. Plants with very low nitrogen reserves (<10 mg per plant), or very low energy reserves (<20 mg starch per plant), were more susceptible. It appears that resistance to *Phellinus* occurs via a defensive pathway that requires resources of both nutrients and carbohydrates.

Key words: nitrogen availability; nonstructural carbohydrates; nutrient reserves; nutrient stress; Oregon Cascades Mountains; Phellinus weirii; Tsuga mertensiana; wave-form dieback.

INTRODUCTION

Relatively pure stands of mountain hemlock (*Tsuga mertensiana* [Bong.] Carr.) in the subalpine zone of the Oregon Cascades die back in distinct waves similar to the fir-waves of New England (Sprugel 1976). The dieback occurs in a radial pattern as root infection and subsequent tree death spread outward from a central infection point (Fig. 1). The pathogen (laminated root rot, *Phellinus* [*Poria*] *weirii* [Murr.] Gilbertson) spreads through woody root contacts in trees of all ages and sizes. Because it remains viable in dead roots and buried stumps for 50 yr or more after tree mortality occurs, trees in regenerating stands can become infected (Hadfield and Johnson 1977, McCauley and Cook 1980). Nevertheless, in many of these mountain hemlock stands, trees in the younger regrowth areas do not exhibit symptoms of *Phellinus* infection, and stand-level reinfection does not occur for 85 yr or more (McCauley and Cook 1980). This suggested to us that some environmental factor that predisposed the trees to infection was reduced or altered after the forest died, allowing the regrowing vegetation to survive.

We examined several dieback areas near Waldo Lake, Oregon, where waves were advancing into 200–250 yr old stands at rates of ≈50 cm/yr (R. D. Boone, *personal communication*). Stands were relatively open, with

densities well below 500 trees/ha. Nitrogen availability, however, was lower than any of the volcanic (Powers 1980) or Mazama ash (Geist 1977) soils previously studied in the region. Nitrogen availability increased two to four fold in the dieback and young regrowth areas, but declined again to old-growth values by ≈85 yr of age (Matson and Boone 1984). We proposed that nutrient limitations in the mature stands predisposed the trees to infection and spread of *Phellinus*, and that the improved conditions in the regrowth areas enabled the regrowing trees to resist infection.

This hypothesis is consistent with a number of studies which have demonstrated that deficiencies in nutrients result in reduced tree vigor and increased susceptibility to diseases, especially to facultative pathogens (Stakman and Harrar 1957, Hare 1966), as well as to some insects (Mattson 1980). However, removing a nutrient limitation may be ineffective when plants are also limited by light, another nutrient, or some other factor (Schoeneweiss 1975, Lambert and Turner 1977, Huber 1980).

In this study, we examined the effects of nutrient and light limitations and their interactions on mountain hemlock response to *Phellinus* infection under controlled conditions. Because limitations of nutrients, light, and other environmental factors affect the levels of biochemical reserves that are available for defense and repair (Schoeneweiss 1975, Huber 1980, McLaughlin and Shriner 1980, Bell 1981), we measured the pool sizes of readily available carbohydrates as well as nitrogen and phosphorus in the trees. Our goal was
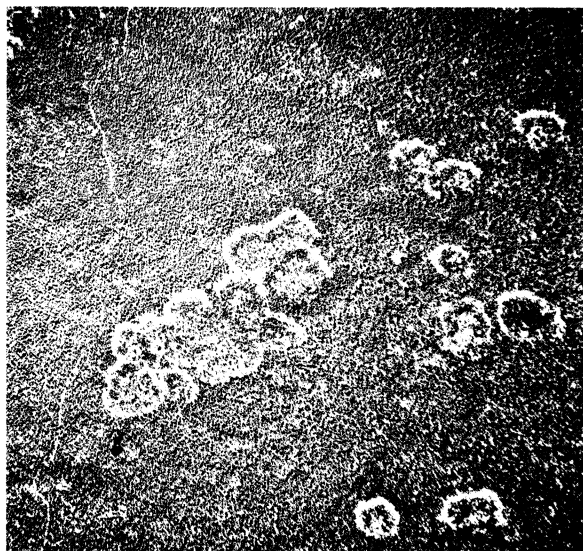
FIG. 1. Aerial photograph of dieback waves in mountain hemlock forests near Waldo Lake, Oregon.

to relate the status of these reserves in plants growing under limitations to the survival of the plants after *Phellinus* infection.

METHODS

Growth room experimental design and treatments

Dormant mountain hemlock trees (15–30 cm tall, 6–8 yr old) were collected from a road-cut near the Waldo Lake dieback area at the time of snowmelt. Trees were held dormant at 2°C for 1 mo, then shipped to the Duke University Phytotron, and planted into pumice soil. They were grown under a 16-h daylength at 600 $\mu E \cdot m^{-2} \cdot s^{-2}$ PAR, with daytime temperature at 20° and nighttime at 14°.

Pumice soil for the growth-room experiment was collected from 0–15 cm depth in an old-growth mountain hemlock stand adjacent to the roadside where the seedlings were collected. The soil was the same as that in the nearby dieback site; it was an Entic Cryorthod in the Winopee series (J. Simonson, *personal communication*), derived from volcanic pumice and ash deposited in the Mazama eruption ≈6600 yr ago, and was 88% sand, 11% silt, and 1% clay.

In September, after buds had broken and growth of aboveground apical meristems was complete, trees were grouped into 10 blocks on the basis of common height, number of branches, and root size. Treatments were then applied in a split-plot design using blocks for replication. Seven nutrient and/or light combinations were the main plot treatments. Each main plot treatment was randomly applied to groups of three trees within each block.

Treatments were designed to alter the degree of nu-

trient or light limitation under which the plants were growing. They originated from nutrient × sugar and nutrient × shade factorials. The main plot treatments were the following: (1) a sugar addition, to stimulate uptake of nutrients by decomposers and reduce availability to the trees (Turner and Olson 1976); (2) a nutrient (nitrogen, phosphorus, sulfur) fertilization; (3) a nutrient plus sugar treatment, to promote microbial activity and increase rates of decomposition, thereby acting as a priming agent for nitrogen mineralization (Bååth et al. 1978); (4) the nonamended field soil control; (5) a shading treatment, which could provide a light limitation in addition to the low nutrient condition of the nonamended field soil; (6) a shade plus nutrient treatment, which would provide inadequate light but adequate nutrients; and (7) a nitrogen alone treatment, to assess if this element was, as we expected, the major deficient nutrient in the soil.

Nitrogen, phosphorus, and sulfur were applied in a ratio of 100 : 16 : 8 after Ingestad and Lund (1979); N was provided at 10 mg/kg of dry soil, P at 1.6 mg/kg, and S at 0.8 mg/kg with each treatment. Sugar (sucrose) was added at 300 mg/kg of soil, and the nitrogen alone treatment provided N at 10 mg/kg. These treatments were applied in solution three times weekly, after all pots were watered to remove excess salt and to bring the pots to equal saturation. Shading with two layers of cheesecloth reduced PAR to 65% of the nonshaded treatments. The sugar, control, shade, and shade plus nutrient treatments were considered a priori limitation or stress treatments.

In November, after 2 mo under the limitation treatments, the three trees in each main plot treatment were assigned randomly to inoculation treatments. One tree was grafted with *Phellinus*-infected mountain hemlock roots, which had been collected 7 d earlier from dying trees in the Waldo Lake dieback area. The roots were covered with the white hyphal mat characteristic of *Phellinus* infection (Hadfield and Johnson 1977). *Phellinus weirii* was isolated from this material on a selective medium (Anita Hutchins, *personal communication*). The grafting method was similar to one recently tested at Washington State University (Morse 1979). Trees were partially removed from their pots, the stems just above the first root branch were washed and sterilized with 65% ethanol, and a 2 cm long × 0.5 cm wide section of bark and cambium was removed in each to expose the xylem. Segments of infected roots 2.0–2.5 cm long and 1 cm in diameter also had xylem exposed and a 0.5 cm deep × 0.5 cm wide depression cut. Trees were placed into the groove on the infected roots, and the exposed xylem tissues held in close contact with rubber bands. Trees were then replaced in the pots.

A second tree from each main plot group was treated in the same manner, except that each was grafted with a noninfected root from healthy mountain hemlock trees. These served as wounding controls. Finally, the

third tree was harvested at the time that the others were grafted. This tree provided the material for biomass and biochemical analyses, as it represented the response of nonwounded trees to the limitation treatments.

Nutrient and light treatments were continued for an additional 9 wk until January 1982, during which time the trees were monitored for foliage yellowing and browning or leaf loss. The visual condition of the foliage was rated with scores of 1 through 5 as follows: 1 = no visible foliage damage, 2 = <12% foliage yellowing, browning, or loss; 3 = >12% and <25% foliage damage; 4 = >25% and <50% damage; and 5 = >50% damage. All scores were updated twice weekly and plants attaining a score of 5 were harvested. After 9 wk of foliar damage evaluation, all remaining plants were harvested. The most recent, updated scores were analyzed using analysis of variance (ANOVA) for a split-plot design with two subplot treatments included (*Phellinus*-inoculation and wounding control). Comparisons between inoculation treatments within each limitation treatment and between limitation treatments follow Steel and Torrie (1980: 381). Arcsine transformations of the score data were also done, but because the ANOVA and individual comparisons showed the same significant relationships, the nontransformed data are reported here.

Harvest methods and chemical analyses

At harvest, plants were removed from the pots, and, after the root systems were carefully washed with deionized water, were separated into roots, leaves, and stems. Subsamples of leaves were weighed and specific leaf areas of fresh tissue were measured. Root mycorrhizal cover, root lengths, and numbers of root branches were also measured on subsamples, and stem heights and diameters were recorded. Stems and remaining leaf and root tissue were quick-frozen with liquid nitrogen and stored at $-10°$ until they could be lyophilized. After freeze-drying, samples were weighed, ground to pass through a 425-μm (40-mesh) screen, and stored dry at $-10°$ until analysis.

Chemical analyses were done only for plants harvested in November. Total nitrogen and phosphorus were measured with a Technicon Autoanalyzer II after samples were digested in a Technicon block digestor using a sulfuric acid-mercuric oxide catalyst (Technicon Instruments Corporation 1977). At least one duplicate was run in every 20 samples to ensure uniformity in the method.

Nonstructural carbohydrates were measured using methods of Haissig and Dickson (1979). Tissue samples 20 mg in mass were extracted in a methanol-chloroform-water (MCW) solution (6 : 2.5 : 1.5, V : V : V), with the supernatant containing soluble sugars, pigments, phenolics, and other solubles (Dickson 1979). Sugars were separated from pigments and lipids by adding 3 mL of water per 5 mL MCW, followed by

centrifugation and separation of the water-alcohol phase from the chloroform phase. Sucrose was hydrolyzed to glucose and fructose with 0.1 mol/L HCl, neutralized with 0.1 mol/L NaOH, and measured by mixing 0.5 mL of diluted sample with 5 mL peroxidase glucose oxidase *o*-dianisidine dihydrochloride reagent (Sigma Chemical Company, St. Louis, Missouri). Sucrose standards were included in each group of 20 to determine percent recovery. Absorbance was measured at 450 nm after 30 min incubation at 30°. While the glucose oxidase reagent has a much higher affinity for glucose, some fructose may also have reacted (Sigma Chemical Company Bulletin 510-A). Recovery of our sucrose standards suggest that, assuming all of the glucose moiety of sucrose reacted first, an additional 30% of the fructose is included in the results.

After MCW extraction, the starch-containing residue was dried at 50° overnight. After 0.2 mL 95% ethanol and 4 mL water were added to each sample, tubes were capped and placed in boiling water for 10 min. Each tube, including water blanks, received 1 mL purified enzyme solution; tissue blanks received only buffer. The enzyme solution was a combination of purified Diazyme L-150 (alpha 1,4 glucan glucohydrolase, Miles Laboratories, Elkhart, Indiana) and Mylase 100 (alpha-amylase, G. B. Fermentation Industries, Des Plaines, Illinois) in concentrations of 10 mg/mL and 5 mg/mL, respectively. Tubes were capped tightly, mixed, and placed in an incubator at 50° for 24 h. After enzymatic hydrolysis, starch was measured as glucose, using the glucose oxidase method described above. Duplicates were run for two samples in each group of 20, and two to five tissue blanks, one enzyme blank, and three starch standards were included with each group.

Total amino acids were analyzed in root tissues from two replicates for four of the treatments (control, nutrient, shade, and shade plus nutrient). The analyses were completed in the Department of Biochemistry at Oregon State University, and followed the procedure of Spakman et al. (1958).

Chemical data were analyzed with ANOVA for a randomized block design using the ANOVA procedure of the SAS statistical programs package (Statistical Analysis System 1979). Log transformations were used when necessary to equalize variances. However, all data are reported here in the nontransformed form. In general, individual comparisons were made using Tukey's honestly significant difference test (HSD) (Steel and Torrie 1980:185). Correlations were done using the GLM procedure of SAS.

RESULTS

Foliage damage scores

Analysis of variance for a split-plot design for the final score of foliage damage showed highly significant effects for both the main plot (limitation) treatments ($P < .0001$) and subplot (inoculation) treatments ($P <$

TABLE 1. Foliar damage score for *Phellinus* inoculation and wounded controls. Low scores indicate no damage, high scores indicate severe damage (see Methods). Each value is the mean ± SE of 10 replicates. Asterisks indicate significant differences between columns only (* $P < .05$, *** $P < .001$); common superscript letters indicate no significant differences within columns.

Growth condition treatment	Inoculation treatment	
	Wounded control	*Phellinus*
	Foliar damage score	
Nonstress treatments		
Nitrogen	1.3 ± 0.21^{ab}	1.6 ± 0.42^c
Nutrient	1.1 ± 0.10^a	1.3 ± 0.15^c
Sugar plus nutrient	1.0 ± 0^a	1.2 ± 0.13^c
Stress treatments		
Shade plus nutrient	1.4 ± 0.40^{ab} *	2.3 ± 0.50^d
Control	1.8 ± 0.20^{ab} *	2.7 ± 0.45^{de}
Sugar	2.1 ± 0.45^b *	3.3 ± 0.37^{ef}
Shade	2.2 ± 0.29^b ***	3.8 ± 0.42^f

.0001), with no significant interaction. In the control, sugar, shade, and shade-plus-nutrient treatments, which were our a priori stress treatments, the plants inoculated with *Phellinus* had significantly more severe foliage damage and mortality than did their paired wounding controls (Table 1), indicating that the damage in these plants was *Phellinus*-induced.

Comparisons between limitation treatments for the *Phellinus*-inoculated plants showed that the nutrient, sugar-plus-nutrient, and nitrogen treatments had significantly lower scores and apparently less susceptibility than did the stress treatments (Table 1). The shading treatment had the most foliage damage resulting from *Phellinus* infection, while the sugar-alone and control treatments produced slightly less damage. The shade-plus-nutrient treatment produced a significantly higher mean score than the nutrient-only treatment, but a significantly lower score than the other a priori stress treatments.

Because scores were assigned using a visual estimate of damage, a correlation of the mean score vs. the mean ratio of total leaf mass to stem mass was used to quantify the amount of leaf damage. Trees from the November harvest had approximately the same ratio regardless of treatment, so variations in the ratio for the January harvest should indicate foliage loss or browning due to infection or wounding, and should therefore correlate with the assigned scores. The correlation coefficient was $-.74$ ($P < .01$); ratios ranged from 0.6 to 1.0, and trees with higher visual damage scores had lower ratios of leaves to stems, indicating that they had more severe foliage damage.

Biomass

Plants harvested in November did not differ significantly in total dry mass when analyzed by ANOVA (Table 2). Root mass, however, did differ among treatments; significantly larger root mass was produced in the nutrient treatment (Table 2). Root : shoot and root : foliage ratios were not significantly different between treatments. The fact that growth response to treatment was evident only in the roots is not surprising, considering that mountain hemlock is a determinate, slow-growing tree. Shoots did not flush during the course of the experiment.

Total nitrogen and phosphorus

Because plant tissue masses were not generally different for the plants harvested in November, differences in total plant tissue nutrients primarily reflected differences in tissue nutrient concentrations. As expected, increasing the nitrogen supply to the roots significantly increased N concentrations and contents in all plant parts (Fig. 2). In the treatments which included nutrient additions, N concentrations averaged 1.1% in leaves, 0.54% in stems, and 0.94% in roots. In contrast, nutrient-limited plants had 0.40% in leaves, 0.23% in stems, and 0.36% in roots. Shading had no significant effect on N concentration or contents in any tissue.

TABLE 2. Dry biomass for plants harvested in November, after 2 mo growth under treatments. Each value is the mean ± SE of 10 replicates. Comparisons of means using HSD were done only when the ANOVA F was significant.

| Treatments	Biomass (g)			
	Total	Leaves	Stems	Roots
Nonstress treatments				
Nitrogen	4.06 ± 0.63	1.02 ± 0.17	1.13 ± 0.18	1.91 ± 0.32
Nutrient	4.42 ± 0.57	1.08 ± 0.15	1.01 ± 0.15	2.33 ± 0.28
Sugar plus nutrient	3.54 ± 0.55	0.86 ± 0.14	0.99 ± 0.21	1.69 ± 0.21
Stress treatments				
Shade plus nutrient	3.53 ± 0.54	0.89 ± 0.14	0.93 ± 0.18	1.71 ± 0.23
Control	3.15 ± 0.36	0.79 ± 0.09	0.88 ± 0.10	1.48 ± 0.22
Sugar	3.69 ± 0.53	1.00 ± 0.18	0.99 ± 0.14	1.70 ± 0.23
Shade	3.16 ± 0.16	0.84 ± 0.16	0.96 ± 0.14	1.36 ± 0.16
HSD				0.764
ANOVA significance	$\cdots$	$\cdots$	$\cdots$	$P \leq .001$
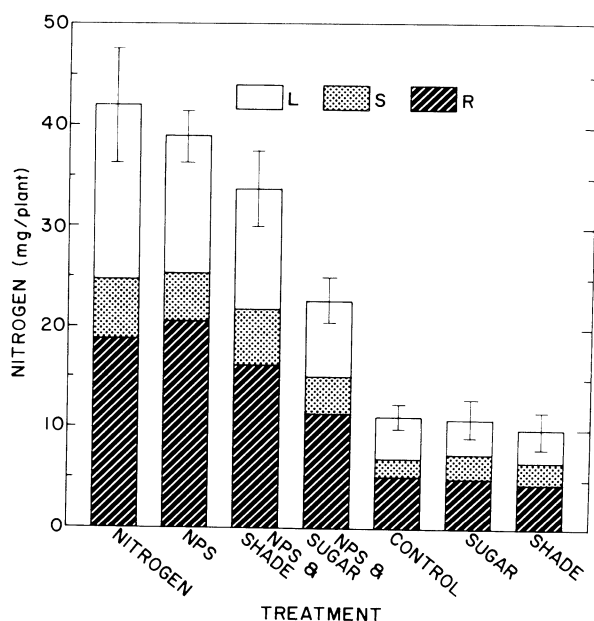
FIG. 2. Total plant nitrogen (mg/plant), divided into leaves (L), stems (S), and roots (R). Upper lines are total plant nitrogen means (±SE) of 10 replicates. NPS = nitrogen, phosphorus, sulfur (the nutrient addition).

Sugar additions to the nonamended soil also had no effect. However, there was significantly less N in the leaves, stems, and roots of the sugar-plus-nutrient treatment plants when compared to the nutrient treatment mean ($P < .05$). Apparently, instead of increasing N availability to the plant through a priming effect on decomposition, the sugar-plus-nutrient treatment provided an accessible carbon source for the decomposers, thereby increasing their N requirement and

leading to N immobilization in microbial biomass. Relative allocation of nitrogen to plant tissues did not differ significantly between any of the treatments (Fig. 2).

When amino acid values were grouped into nutrient treatment and nutrient limitation means (i.e., nutrient and shade-plus-nutrient treatments vs. control and shade treatments), the nutrient-treated plants had three times more total amino acids than did the nutrient-limited plants (*t* test, $P < .05$). In addition, the nutrient-treated plants put ≈30% of this pool into arginine, a common storage amino acid (Van den Driessche and Webber 1975, 1977). In contrast, the nutrient-limited plants had only 11% of their amino acid pool as arginine (*t* test, $P < .05$).

Total plant phosphorus was significantly greater in the nitrogen and the NPS treatment plants than in all other treatments except the shade-plus-nutrient treatment ($P < .05$, Fig. 3). Similarly, P concentrations in the roots were significantly greater in the nutrient-treated plants than in the nutrient-limited plants (0.11 vs. 0.08% respectively, $P < .05$). Relative allocation of P to plant tissues varied, with the plants growing without nutrients or nitrogen allocating a greater proportion of their total P to leaves ($P < .01$, Fig. 3). These differences in P uptake and allocation cannot be attributed simply to P fertilization, because differences were not found between the nutrient (NPS) and the nitrogen-alone treatments.

Plant nonstructural carbohydrates

Plants growing without added nutrients generally had higher concentrations and accumulated significantly greater quantities of starch than did those treated with nutrients (Fig. 4). Plants grown without nutrient additions had 6.14, 2.27, and 4.1% starch in leaves, stems, and roots respectively. In contrast, plants grown with
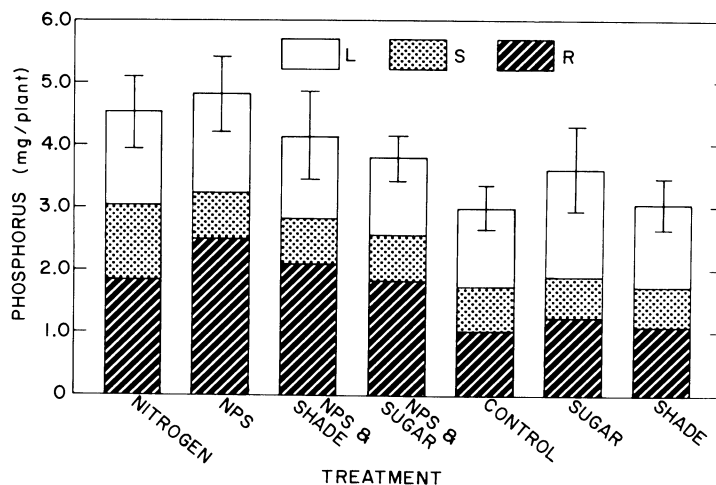

FIG. 3. Total plant phosphorus (mg/plant), divided into leaves (L), stems (S), and roots (R). Upper line indicates total plant means (±SE) of 10 replicates. NPS = nitrogen, phosphorus, sulfur (the nutrient addition).
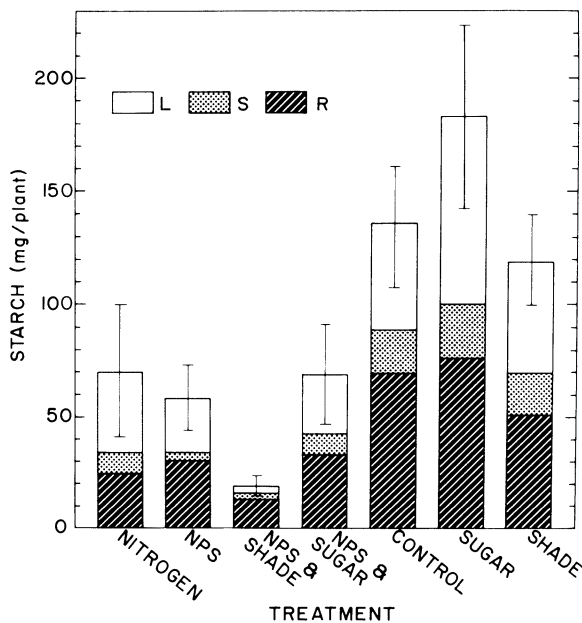
FIG. 4. Total plant starch (mg/plant), divided into leaves (L), stems (S), and roots (R). Upper line indicates total plant starch means (±SE) of 10 replicates. NPS = nitrogen, phosphorus, sulfur (the nutrient addition).

nutrient additions averaged 1.6, 0.51, and 1.36%, respectively.

Total tissue quantities of the measured sugars were not different between treatments. However, concentrations of sugars in leaves were significantly lower in all of the nutrient-treated plants than in those growing without nutrient additions ($P < .05$ for each pairwise comparison between groups). Means for leaves of nutrient-treated and nutrient-limited groups were 2.1 and 3.1%, respectively. Mean root concentrations were 2.2 and 2.7%, respectively. As mentioned earlier, these measurements do not include all sugars, but only give a relative estimate of the contributions of glucose and sucrose. Sucrose is in general the sugar that varies most with season and nutrient status in conifers (Krueger and Trappe 1967, Geiger 1979), and glucose and sucrose have key roles in transport and the regulation of transport of carbon (Kramer and Kozlowski 1979).

The similarity of patterns between these sugars and starch suggest that nonstructural carbohydrates in general accumulate under limited nutrient availability. This is in agreement with a number of studies of both agricultural and wild plants (Smith 1973, Ericsson 1979, Ericsson and Persson 1980, and Shaver and Chapin 1981), and presumably occurs because without adequate nutrients for growth the carbon fixed in photosynthesis is in excess of the demands of the plant (Mattson 1980, Webb 1981).

When trees received nutrients, shading significantly lowered starch concentrations and accumulation in the

leaves ($P < .05$, Fig. 4). For plants growing without nutrients, however, starch accumulation with shading was not significantly different from the control. The ANOVA for root : shoot ratios of starch did not show a significant treatment effect. For the shade-plus-nutrient treatment, however, the plants apparently allocated a much greater proportion of the total plant starch to the roots (Fig. 4). Root pools and concentrations of starch and sugar were not significantly different between the shade-plus-nutrient and nutrient-alone treatments.

DISCUSSION

Trees that were grown under nutrient limitation were more susceptible to *Phellinus* infection than trees receiving nutrients. These more susceptible plants in general had low nitrogen and phosphorus contents and high starch storage. The availability of stored carbohydrates suggests that the production of carbon-based secondary chemicals such as phenolics may not have been quantitatively important in defense. Phenolic levels are normally highest when low nitrogen availability limits the production of proteins, thus leaving their common precursor, phenylalanine, available for use in phenolic synthesis (Phillips and Henshaw 1977).

Shading trees grown without adequate nutrients results in even higher infection scores and greater susceptibility to *Phellinus*. This increased susceptibility to infection under a combination of light and nutrient limitation, however, could not be related to the measured plant reserves or allocation of reserves, as the trees that received shading without nutrients had nutrient and carbohydrate reserves similar to the nutrient-limited control plants. Carbon reserves were not reduced by shading, probably because when nutrients are inadequate, carbon fixation and storage is more limited by a lack of N-containing compounds such as chlorophyll and RuBP carboxylase than by light availability. Qualitative as well as quantitative variations in soluble sugars, amino acids, or organic acids could be caused by shading (Durzan 1971), and could influence susceptibility of trees to attack by *Phellinus* (Li and Bollen 1975).

Among trees receiving nutrient additions, shading significantly increased the trees' susceptibility to the pathogen. In this case, however, shading did reduce the amounts, concentrations, and allocation of nonstructural carbohydrates without affecting plant nutrient levels. When nutrients are adequate, reduction in light intensity does influence carbon fixation and storage (Little and Loach 1973, Smith 1973, Magnussen 1981). Increased susceptibility to infection under adequate nutrients but low light could indicate that some lower threshold of carbon necessary for allocation to defense or repair had been exceeded. Alternatively, this result could suggest that N was in excess relative to carbon, resulting in alterations in the biochemical components which influence susceptibility to

the pathogen. Excess N has apparently resulted in greater susceptibility to insects and disease in a number of studies (Stakman and Harrar 1957, Hesterberg and Jurgensen 1972, Lambert and Turner 1977, Onuf et al. 1977).

Together, these results suggest that resistance to the spread of *Phellinus* in mountain hemlock occurs via a defensive pathway that requires adequate resources of both nitrogen and carbon. One such pathway, the building of morphological barriers, is a relatively common response to wounding and invasion by decay fungi and rusts (Hare 1966, Shigo et al. 1977, Bell 1981), and could have functioned in resistance to *Phellinus*. However, resistance may depend on more than one biochemical and physical component acting through more than one defensive pathway (Levin 1976, McLaughlin and Shriner 1980, Bell 1981).

Whatever the pathway of defense, our results show that plants growing without adequate nutrients were more susceptible to infection by *Phellinus* than were nutrient-treated plants. These results may explain the field observation that while mountain hemlock trees growing in the old-growth stands are susceptible to infection, trees in the regrowth zone do not show infection and mortality. The increased nutrient availability after dieback could lead to increased resistance and prevent reinfection of the regrowing stands (Matson and Boone 1984).

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