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Tree vigour and the susceptibility of Douglas fir to *Armillaria* root disease

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Summary

In this study the effects of thinning, fertilization and pruning on the vigour of Douglas fir (*Pseudotsuga menziesii*) and its susceptibility to *Armillaria* root disease were explored. Tree vigour was defined as the relative capacity for tree growth, expressed as the above-ground biomass increment per unit of photosynthetic tissue, or growth efficiency (GE). It has been hypothesized that trees with higher GE can better resist pathogen attack, and that GE can be used as a predictor of tree susceptibility to disease. In a previous study, four Douglas fir plantations were thinned, fertilized and pruned in all combinations, and the effects of these treatments on tree vigour were measured after 10 years. Root disease was not a factor in the initial study design, and mortality was ignored until 8 years after the treatments were applied. The results of an earlier study were utilized and the correlation between *Armillaria* root disease incidence and the effects of earlier stand treatments on tree growth was tested. *Armillaria ostoyae* was the primary mortality agent in the study area. Disease incidence on infested subplots ranged from 2 to 20%. *Armillaria ostoyae* incidence was the highest at medium tree density (6.1%), slightly lower on the low density (5.6%), and lowest on the unthinned plots (3.8%). There were no significant correlations between disease incidence and previous tree growth. The vigour of trees that became symptomatic or died by 1993 was not significantly different in 1983–85 from the vigour of trees that remained asymptomatic. On these sites, in areas of infestation, *A. ostoyae* is killing the largest, fastest growing trees, as well as less vigorous trees. *Armillaria* continues to cause mortality, regardless of the growth efficiency or growth rate of the host.

1 Introduction

Armillaria ostoyae (Romagnesi) Herink is a common cause of mortality in coniferous forests of western North America affecting both natural and planted stands (Hadfield et al. 1986). It is commonly stated that *Armillaria* pathogenicity is greater when trees are somehow weakened or stressed (Wargo and Harrington 1991) and forest stand management to improve tree vigour may be an effective way to reduce current and future losses from *Armillaria* infections (Morrison 1981; Hagle and Shaw 1991). In this observational study the effects of thinning, fertilization and pruning on tree vigour and susceptibility to *Armillaria* root disease in Douglas fir in western Oregon were explored.

Tree vigour can be defined as the relative capacity for tree growth, expressed as the above-ground biomass increment per unit of photosynthetic tissue, or growth efficiency (Waring 1983). Growth efficiency (GE) is a function of photosynthetic efficiency and is thought to influence carbon allocation and the ability to produce plant-defence compounds. It has been hypothesized that trees with higher GE can better resist pathogen attack, and that GE can be used as a predictor of tree susceptibility to disease (Waring 1983). In a previous study, four Douglas fir plantations were thinned, fertilized and pruned in all combinations, and the effects of these treatments on tree vigour were measured after 10 years (Velázquez-Martínez et al. 1992). Root disease was not noted when the initial study treatments were

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applied, but within 10 years it forced the abandonment of the original design. In the current study this existing situation was utilized and the correlation between the stand treatments as they influenced tree growth and *Armillaria* root disease incidence was tested.

2 Materials and methods

2.1 Research area

This work was carried out at the H.J. Andrews Experimental Forest (Blue River Ranger District, Willamette National Forest, 44°14′N, 122°20′W), in the central portion of the western Cascade Mountains in Oregon. All sites were situated within the *Tsuga heterophylla* Zone (FRANKLIN and DYRNESS 1973), where western hemlock (*Tsuga heterophylla* Sarg.) and western red cedar (*Thuja plicata* D. Don.) constitute the potential climax tree species, and Douglas fir (*Pseudotsuga menziesii* (Mirb.) Franco var. *menziesii*) occurs in large areas as a dominant, subclimax species. Four sites, previously uncut, were harvested and slash-burned between 1958 and 1965 and planted with Douglas fir seedlings of a local provenance. Seedlings were planted at about 2.5 m spacing. Stand elevations ranged from 700 to 850 m. After planting, natural regeneration of Douglas fir, western red cedar and especially western hemlock occurred. In 1981, when the treatments were applied, the sites were densely stocked (3000–4000 stems/ha).

2.2 Silvicultural treatments

The study to determine tree growth response to different silvicultural treatments was initiated by Dr David PERRY and colleagues at Oregon State University (VELÁZQUEZ-MARTÍNEZ et al. 1992). Each of the four harvest units ('sites') was considered a replicate in a split plot design. Each replicate was divided into three plots. One plot was thinned to 300 trees/ha (low density), one thinned to 604 trees/ha (medium density), and one left unthinned (high density), averaging 3459 trees/ha. Douglas fir was favoured in thinned plots. Each plot was then split into four subplots, and the following treatments were applied: (1) fertilized only, (2) pruned only, (3) pruned and fertilized, and (4) untreated control. All subplots initially contained 50 trees, and thus varied in area. Each subplot was surrounded by a 10 m buffer strip. The experimental area of the four sites ranged from 4.1–6.8 ha. Fertilization was carried out by burying slow-release 'complete' fertilizer tablets around each tree (each tablet contained: 4.2 g N, 2.1 g P, 1.0 g K, 0.55 g Ca, 0.34 g S and 0.07 g Fe). The number of tablets per tree varied with diameter, in order to match the fertilization level to tree size. Pruning consisted of removal of the lower branches of the crown. Trees with average diameter or larger had 20–40% of the live crown removed; smaller trees had 0–15% removed. Details of the treatments are in VELÁZQUEZ-MARTÍNEZ et al. (1992).

2.3 Tree growth analysis

Diameters (d.b.h.) of all trees within the subplots were measured in 1983, 1985, 1987 and 1993. Sapwood area was determined for these same years by taking increment core samples (two per tree) of one-third of the trees in each subplot (VELÁZQUEZ-MARTÍNEZ et al. 1992). Regression analyses between d.b.h. and sapwood area were performed in order to estimate the sapwood area of the rest of the trees in each subplot for 1983, 1985 and 1987. Radial growth increment (RG) and growth efficiency (GE) for the periods 1983–85, 1985–87 and 1987–93 were calculated on an individual tree basis. GE (kg/m^2 per year) was calculated

from leaf area (LA = 0.47 × sapwood area; WARING et al. 1982) and biomass estimated from d.b.h. based on equations for Douglas fir proposed by GHOLZ et al. (1979).

Growth Efficiency was calculated as: GE = (biomass year 2 – biomass year 1)/LA year 1 × years.

ANOVA was performed for each time period, in order to determine whether there were differences among treatments (significance level of 0.05) in RG and GE. RG and GE responses were similar in all cases; data is presented for GE only. Means of significant interactions were compared with Bonferroni tests. A logarithmic transformation of the data was necessary to fulfill the test assumptions.

2.4 Disease assessment and analysis

In 1993, all trees on the subplots and within the buffer zone were inspected for above-ground symptoms of *Armillaria* root disease, such as foliage loss, chlorosis, and stem resinosis. Trees were classified as asymptomatic, symptomatic, or dead. Portions of the bark at the base of the stem of all symptomatic and dead trees were removed to look for mycelial fans and rhizomorphs. Main roots of symptomatic trees were excavated up to a radial distance of 1 m from the stem to check for the presence of rhizomorphs or mycelial fans. Lesions on the roots and stem resinosis were used to assess the aggressiveness of the fungal attack. The year of death was estimated from earlier plot records and the general appearance of recently killed trees. The percentage disease incidence in each subplot was calculated as the number of *Armillaria*-affected (symptomatic and dead) trees divided by the total number of trees. All trees which died before 1993 were identified from previous records.

Data from subplots with disease were used in the correlation analysis. Correlations of disease incidence with average GE, RG, and d.b.h. were calculated.

3 Results

3.1 Tree growth analysis

Site had a significant effect on growth efficiency (GE) (Table 1) and radial growth increment (RG) in all time periods. The range among the four sites was 0.18–0.29 kg/m^2 per year for

Table 1. **Split plot ANOVA for effects of thinning (three residual densities), pruning, and fertilization on growth efficiency in Douglas fir plantations at three time intervals after treatment**

		1983–85		1985–87		1987–93	
	df	MS	Pr > F	MS	Pr > F	MS	Pr > F
Site	3	0.16	0.002*	0.25	0.0001*	0.41	0.0001*
Density	2	0.84	0.038*	0.93	0.002*	1.74	0.025*
Error a	6	0.14	0.001*	0.04	0.003*	0.24	0.0001*
Pruning	1	0.02	0.343	0.00	0.864	0.01	0.614
Fertiliz.	1	0.00	0.896	0.02	0.171	0.00	0.644
Pr. × Fert.	1	0.03	0.280	0.01	0.437	0.01	0.518
Dens. × Pr.	2	0.04	0.247	0.01	0.538	0.00	0.932
Dens. × Fert.	2	0.04	0.229	0.01	0.567	0.01	0.755
Dens. × Fert. × Pr.	2	0.11	0.022*	0.03	0.069	0.03	0.243
Error b	27	0.02		0.01		0.02	
Total	47						

* Asterisk indicates significance at the 0.05 level.

GE, and 0.74–0.99 cm/year for RG. Thinning had the most influence on growth response among all the silvicultural treatments. On all sites, lower densities resulted in faster RG and larger tree diameters, as well as greater values of GE across all time periods (Table 2 and Fig. 1). In 1983 the average d.b.h. of the high density treatment equalled 10.1 cm, medium density was 13.9 cm, and low density was 15.7 cm. Ten years later, differences among density

Table 2. **Diameter (first and last year of measurement), growth efficiency (first and last period) and disease incidence (1993) averaged by subplot. Numbers within parentheses are standard errors of the means (n = 4)**

Plot	Subplot	Diameter (cm) 1983	Diameter (cm) 1993	Growth efficiency (kg/m^2 per year) 1983–1985	Growth efficiency (kg/m^2 per year) 1987–1983	Disease incidence (%) 1993
H	C	10.7 (2.22)	17.8 (2.49)	0.14 (0.02)	0.16 (0.06)	1.7
	F	9.6 (2.53)	16.0 (3.19)	0.22 (0.05)	0.17 (0.04)	3.3
	P	9.8 (1.40)	15.8 (1.76)	0.19 (0.04)	0.18 (0.04)	7.5
	P + F	10.4 (2.71)	15.7 (2.80)	0.17 (0.02)	0.14 (0.03)	2.7
Mean		10.1	16.3	0.18	0.16	3.8
M	C	13.8 (2.12)	22.3 (1.83)	0.24 (0.02)	0.25 (0.04)	8.9
	F	14.4 (2.18)	23.1 (1.93)	0.23 (0.01)	0.25 (0.03)	5.2
	P	13.1 (1.62)	21.0 (1.52)	0.24 (0.01)	0.24 (0.03)	5.7
	P + F	14.4 (2.19)	23.2 (1.79)	0.24 (0.01)	0.26 (0.03)	4.7
Mean		13.9	22.4	0.24	0.25	6.1
L	C	16.8 (1.25)	29.0 (1.58)	0.32 (0.02)	0.31 (0.02)	5.3
	F	14.3 (0.37)	24.9 (0.41)	0.28 (0.01)	0.31 (0.01)	4.0
	P	17.0 (2.49)	28.2 (2.99)	0.26 (0.06)	0.30 (0.01)	11.7
	P + F	14.9 (0.71)	25.8 (0.88)	0.26 (0.06)	0.31 (0.02)	1.5
Mean		15.7	27.0	0.28	0.31	5.6

H, high (control) density; M, medium density; L, low density; C, no treatment; F, fertilized; P, pruned; P + F, pruned and fertilized.

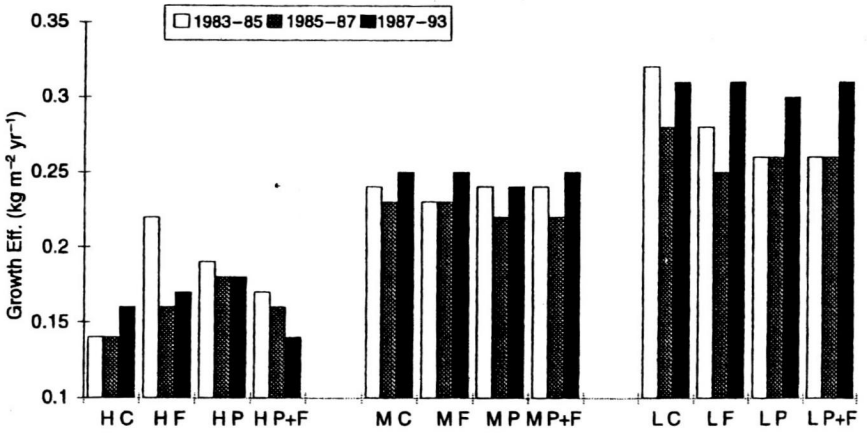

Fig. 1. Tree growth efficiency by density level and treatment. H, high (control) density; M, medium density; L, low density; C, no treatment; F, fertilized; P, pruned, and P + F, pruned and fertilized

levels were even greater, with d.b.h. averaging 16.3, 22.4 and 27.0 cm, for high, medium and low density, respectively (Table 2). The 1987–93 GE means were 0.16, 0.25 and 0.31 kg/m^2 per year for high, medium and low density, respectively. Comparing the first with the last time period, GE experienced an overall reduction at high density (from 0.18–0.16 kg/m^2 per year), a slight increase at medium density, from 0.24–0.25 kg/m^2 per year and a larger increase at low density, from 0.28–0.31 kg/m^2 per year (Table 2). Neither pruning nor fertilization had a significant effect on GE or RG.

Comparisons of treatment means were performed for the time periods that showed significant treatment interactions (Fig. 2). The GE means of thinning–pruning–fertilization treatments were compared for the 1983–85 period (Prob. sig. interaction = 0.02, Table 1) and for the 1985–87 period ($p = 0.07$). The 1987–93 period was not included in the comparison of means because the three treatments combined did not have a significant effect on GE ($p = 0.24$, Table 1).

In 1983–85, all high density treatments had significantly lower ($p = 0.05$) GE than all low density treatments, and lower GE than medium density treatments in 12 of the 16 comparisons (Fig. 2). In 1985–87, GE of high density treatments was different from low density treatments in 10 of 16 comparisons and from medium density treatments in only three of 16 cases (Fig. 2).

3.2 Disease assessment and analysis

Armillaria ostoyae was the primary mortality agent in the study area. Identification was confirmed by pairing tissue isolates with known haploid testers. None of the 12 subplots on one site had infected trees, although *A. ostoyae* was found in buffer areas. On the remaining three sites, 23 of the 36 subplots were infected. Disease incidence on these subplots ranged from 2 to 20%. Plot mortality rates for all sites and plots combined ranged from 2 to 3.5 trees/year through 1987. After 1987, rates increased to 7.6 trees/year.

Armillaria incidence was the highest at medium density (6.1%), slightly lower on the low density (5.6%), and lowest on the high density plots (3.8%) (Table 2). All incidence values showed a relatively high variation among treatments within density levels. There were no significant correlations between disease incidence on subplots in 1993 and tree growth

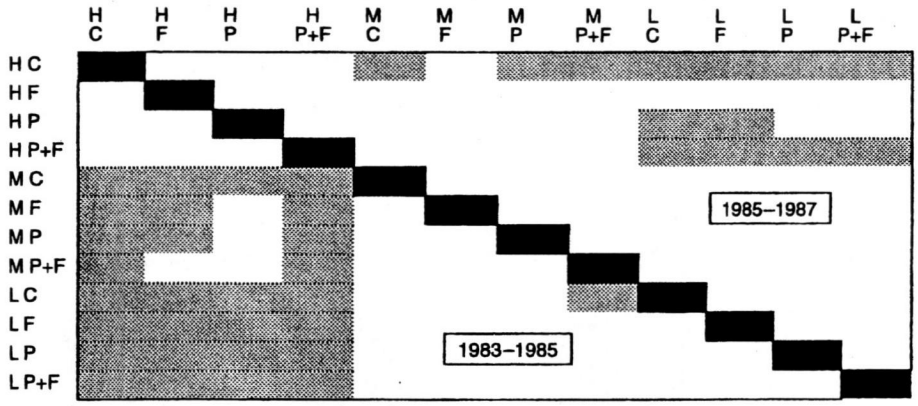

Fig. 2. Comparison of density-fertilization-pruning treatment means for GE (log transformation) for 1983–85 (right side of the diagonal), and 1985–87 (left side of the diagonal). Shaded cells indicate that the means compared are significantly different at the 0.05 level. H, M and L, high, medium and low density; C, no treatment; F, fertilized; P, pruned, P + F, pruned and fertilized

Table 3. **Correlation between disease incidence of subplots in 1993, and tree growth variables (only subplots with *Armillaria* mortality were included in the analysis). Pearson coefficient, n = 23. None of the coefficients were significant at the 0.05 level**

Variable	Correlation coefficient
Growth efficiency 1983–85	0.30
Growth efficiency 1987–93	0.11
Radial growth 1983–85	0.15
Radial growth 1987–93	0.20
d.b.h. 1983	0.13
d.b.h. 1985	0.14
d.b.h. 1987	0.16
d.b.h. 1993	0.16

responses in the first or the last periods of the experiment. Correlation coefficients ranged from 0.13–0.30 (Table 3).

3.3 Disease and individual tree vigour

To study the relationship between earlier tree growth and current disease, trees within subplots were grouped based on their condition in 1993 as: (A) asymptomatic, (B) live with symptoms caused by *Armillaria* or (C) killed by *Armillaria* after 1987 (trees that died before 1987 were excluded from this analysis because of uncertainty over cause of death). The means of GE and RG were calculated for each group of trees for each time interval, and the means of log-transformed data of groups B and C were compared with the asymptomatic group mean (group A), using t-tests (Fig. 3).

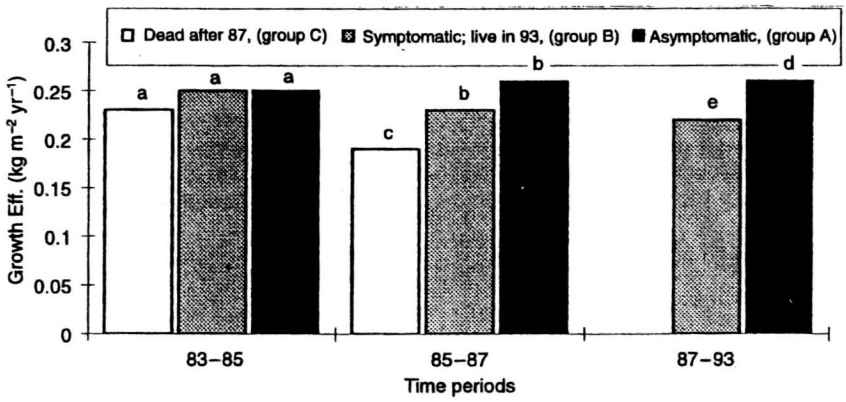

Fig. 3. GE in 1983–85, 1985–87, and 1987–93 for trees asymptomatic in 1993 (Group A), trees alive in 1993 but symptomatic and infected with *Armillaria* (Group B), and trees killed by *Armillaria* after 1987 (Group C). Comparisons were made within each time period between Group A and Groups B and C, but not between Group B and Group C. Means within the same time period with the same letters do not differ significantly at 0.05 level. Tests of significance were done with log-transformed data

The GE of trees that were killed after 1987 (group C) declined from 0.22 kg/m^2 per year in the 1983–85 period to 0.19 kg/m^2 per year in the 1985–87 period. The GE of trees symptomatic in 1993 (group B) was 0.25, 0.23, and 0.22 kg/m^2 per year in the 1983–85, 1985–87, and 1987–93 periods, respectively, and the GE of trees without symptoms in 1993 (group A) was 0.25, 0.26, and 0.26 kg/m^2 per year in the same periods. The GE mean of the recently dead trees (group C) did not differ significantly from the asymptomatic group (group A) until 1985–87 ($p = 0.004$), and GE of currently symptomatic trees (group B) differed significantly from asymptomatic trees (group A) only in 1987–93 ($p = 0.036$). Changes in RG (radial growth increment) followed the same pattern.

4 Discussion

Armillaria root disease is generally less damaging in the rapidly growing Douglas fir forests west of the Cascade mountain range in the Pacific Northwest than in the drier, slower growing forests east of the Cascades (HADFIELD et al. 1986; MCDONALD 1990; MORRISON et al. 1985; MORRISON 1981). The difference in disease severity is often attributed to differences in tree vigour. Mortality in westside plantations is usually scattered and closely associated with large stumps from the previous stand (FILIP 1979). In addition, mortality declines after about age 15 years on most westside sites, and trees are seldom killed after age 25–30 years (HADFIELD et al. 1986; MORRISON et al. 1985; JOHNSON et al. 1972; FOSTER and BARANYAY 1971). Locally, however, as in the present study area, the disease is more severe, and mortality may continue as the stand ages. The reasons are unclear.

On the sites used in the present study, incidence of *Armillaria* root disease in Douglas fir is evidently not limited by tree growth. Thinning produced a significant increase in tree vigour, even at the first time period, but enhanced tree vigour at low tree densities did not correspond with low root disease incidence on either plots or subplots. Rather, the lowest amount of root disease was on the low vigour, unthinned plots (Table 2). The uneven distribution of inoculum limits interpretations in this observational study, but analysis based on individual trees known to have been infected by *Armillaria* also supports the conclusion. Trees that were symptomatic or recently dead in 1993 had been growing 8–10 years earlier with the same efficiency and at the same rate as trees that remained asymptomatic (Fig. 3). Any decline in GE (growth efficiency) or RG (radial growth increment), presumably the result of advancing infection (MORRISON et al. 1991; VAN DER KAMP 1993), occurred only in the years immediately preceding death in these young trees. On these sites, in areas of infestation, *A. ostoyae* is killing the largest, fastest growing trees, as well as less vigorous trees. Larger trees do have more extensive root systems, and are thus more likely to contact below-ground inoculum.

The amount, condition, and distribution of inoculum (old infected roots and stumps) influences incidence of *Armillaria* root disease (REDFERN and FILIP 1991). The inoculum potential or distribution in the study areas used were not measured, but there were large (1–2 m) old-growth stumps from the previous stand on all plots. In addition, clonal analysis of *A. ostoyae* isolates (data not shown) indicated that on each of the four sites a single fungal individual spanned most of the area, although mortality was not uniformly distributed. The large size of most of these clones indicates that the infections were well established in the previous old-growth stand. The old clones are now discontinuous, perhaps explaining the scattered distribution of current mortality.

Many trees were thinned to waste in the initial stand spacing, creating many small stumps in the thinned plots. While it is possible that the fungus increased on this new food base and spread from the stumps to attack and kill the surrounding live trees, this seems unlikely. The stumps were small (< 10 cm), and pre-commercial thinning has been a common practice in Douglas fir plantations, with no observed increase in *Armillaria* root disease.

Some other studies in the western United States have examined the effects of thinning at young age on *Armillaria* root disease. FILIP and GOHEEN (1995) found no differences in mortality between thinned and unthinned Douglas fir and western hemlock plots 10 years after precommercial thinning. FILIP et al. (1989) applied similar treatments in Ponderosa pine stands in central Oregon. After 20 years they found higher mortality in unthinned plots. ENTRY et al. (1991) inoculated Douglas fir (*P. menziesii* var. *glauca*) in plantations 10 years after plots were thinned, thinned and fertilized or left untreated. Inoculation success was greatest on the largest trees with the highest GE, in the thinned and fertilized treatment. Inoculation success in that study was not related to GE, but was correlated with the ratio of phenolic compounds to sugars in the roots. Higher leaf area and stem growth in thinned and fertilized trees were hypothesized to have increased the ratio of sugars to phenolic compounds in the roots, thus favouring the fungus.

In synthesis, *Armillaria* in the sites used in the present study continues to cause mortality, regardless of the growth efficiency or growth rate of the host. Under some conditions, *A. ostoyae* acts as an aggressive pathogen, killing vigorous trees. Silvicultural treatments to increase tree growth may not reduce disease levels under these circumstances. In order to explain the local distribution of severe *Armillaria* root disease, more information is needed about microsite factors influencing distribution and inoculum potential of the pathogen and changes in the physiological status of host roots.

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Résumé

Vigueur du Douglas et sensibilité à l'Armillaire

Nous avons examiné l'effet de l'éclaircie, de la fertilisation et de l'élagage sur la vigueur du Douglas et sur sa sensibilité à l'Armillaire. La vigueur a été définie comme l'aptitude relative à la croissance exprimée par l'augmentation de biomasse aérienne par unité de tissus photosynthétiques: l'efficience de croissance (GE). On a pris comme hypothèse que les arbres présentant une plus grande GE peuvent mieux résister à l'attaque du pathogène et que GE peut être utilisé comme un indicateur de la sensibilité à la maladie. Dans une précédente étude, quatre plantations de Douglas avaient été éclaircies, fertilisées et élaguées dans toutes les combinaisons et l'effet de ces traitements sur la vigueur avait été mesuré au bout de 10 ans. La maladie n'ayant pas été prévue dans le protocole initial, la mortalité n'avait pas été prise en compte pendant les 8 premières années. Nous avons utilisé cette étude pour tester la liaison entre l'incidence de l'Armillaire et les effets des traitements antérieurs sur la croissance des arbres. *Armillaria ostoyae* était l'agent primaire de la mortalité dans la zone étudiée. L'incidence de la maladie variait de 2 à 20% selon les placeaux. Elle était plus élevée (6,1%) aux densités moyennes, un peu plus faible (5,6%) aux densités faibles et plus basse (3,8%) dans les placeaux non éclaircis. Il n'y avait pas de liaison significative entre l'incidence de la maladie et la croissance antérieure des arbres. En 1983–85, la vigueur des arbres n'était pas significativement différente chez les arbres apparemment sains et ceux qui présentaient des symptômes ou qui étaient morts en 1993. Dans les zones infectées de ces sites, *A. ostoyae* tue aussi bien les arbres les plus gros, ceux qui poussent le plus vite, ou les moins vigoureux. *A. ostoyae* est une cause de mortalité permanente, indépendante de l'efficience de croissance et de l'accroissement de l'hôte.

Zusammenfassung

Vitalität und Anfälligkeit der Douglasie gegen Armillaria Wurzelfäule

Es wurden die Einflüsse von Durchforstung, Düngung und Astung auf die Vitalität von Douglasien und ihre Anfälligkeit gegen einen *Armillaria*-Befall untersucht. Die Baumvitalität wurde definiert als relative Wachstumskapazität, ausgedrückt als oberirdische Biomasse pro Einheit photosynthetisch aktiven Gewebes (Wachstumseffizienz, GE). Es wurde die Hypothese aufgestellt, dass Bäume mit einer höheren GE gegen den Angriff des Pathogens resistenter sind und dass die GE zur Vorhersage der Anfälligkeit benutzt werden kann. In einem früheren Experiment waren vier Douglasien-Plantagen durchforstet, gedüngt und geastet worden (in allen Kombinationen), und die Auswirkungen dieser Behandlungen auf die Baumvitalität wurden nach 10 Jahren gemessen. Wurzelkrankheiten wurden in das anfängliche Versuchsdesign nicht einbezogen, und das Absterben von Bäumen wurde in den ersten 8 Jahren nach den Behandlungen nicht berücksichtigt. In der vorliegenden Untersuchung wurden auf der Basis dieser früheren Aufnahmen Korrelation zwischen dem Auftreten der *Armillaria*-Wurzelfäule und den Effekten der früheren Behandlungen auf das Baumwachstum geprüft. *Armillaria ostoyae* war in den Versuchsflächen die primäre Ursache für das Absterben von Bäumen. Die Krankheitshäufigkeit erreichte in den infizierten Versuchsflächen 2–20%. *A. ostoyae* kam am häufigsten auf den Flächen mit mittlerer Baumdichte vor (6,1%), die Krankheit trat auf den Flächen mit niedriger Baumdichte etwas weniger in Erscheinung (5,6%) und war auf den nicht durchforsteten Flächen am geringsten (3,8%). Zwischen der Krankheitshäufigkeit und dem vorangehenden Baumwachstum ergab sich keine signifikante Korrelation. Die Vitalität der Bäume, die erkrankten oder bis 1993 abstarben, war nicht signifikant verschieden von der in den Jahren 1983 bis 1985 gemessenen Vitalität symptomloser Bäume. In den untersuchten Beständen tötet *A. ostoyae* die grössten und wüchsigsten Bäume ebenso wie weniger vitale. *Armillaria* führt weiterhin zum Absterben von Bäumen, unabhängig von ihrer Wachstumskapazität oder ihrem Zuwachs.

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