ARMILLARIA ROOT DISEASE OF *PINUS RADIATA* IN NEW ZEALAND. 6: GROWTH LOSS

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ABSTRACT

Armillaria root disease is widespread in plantations of *Pinus radiata* D. Don in many parts of New Zealand. Data from research trials in three central North Island forests were used to estimate the impact of infection, as assessed by the extent of root collar girdling, on stand growth approximately halfway through the rotation period, assuming a causative relationship between infection severity and growth. Volume loss was determined as a little over 2% at age 13 years, in a second-rotation stand with ca. 20% of trees infected prior to thinning, on a site not previously covered in indigenous forest. This value was calculated from the average stem volume for each of four infection severity classes and the relative numbers of trees in each class. Indices to compensate for competition were included in the model, based on stem diameters and local stocking densities influencing the growth of each tree. Mortality was not a significant factor in this stand. By contrast, mortality induced by Armillaria root disease was typically high in first-rotation stands planted on ex-indigenous forest sites, causing gaps in stocking resulting in substantially greater volume loss. In one such stand a comparison of mid-rotation crop tree volumes with early mortality indicated that a crop volume reduction of 21% had occurred. An additional loss of 4% in this stand was attributable to growth reduction in infected but still living trees. Although comparatively low, growth loss in infected trees may have significant economic impact if the disease becomes generally dispersed in second-rotation plantations.

Keywords: root disease; growth loss; *Armillaria novae-zelandiae*; *Armillaria limonea*; *Pinus radiata*.

INTRODUCTION

Two studies have attempted to estimate the extent to which the root disease fungi *Armillaria novae-zelandiae* (Stevenson) Herink and *A. limonea* (Stevenson) Boesewinkel reduce projected wood volume yields in first-rotation stands of *Pinus radiata* growing on logged indigenous forest sites. Both estimates were based on the growth loss determined on infected trees with different degrees of root collar resinosis. Shaw & Calderon (1977) used measurements of diameter increment reduction in infected living trees to estimate a volume loss of 29–32% for a 15- to 21-year-old pulpwood rotation and a 26-year-old sawlog regime. Their calculations incorporated a 25–30% loss in ground area due to the formation of patches of mortality, and a significant increment decrease among the 15% most severely infected

final-crop trees. MacKenzie (1987) used new data collected from the same stand 9 years later to revise the previous estimate. This was done by making sectional measurements to determine volume increment on selected trees in different root collar resinosis classes. MacKenzie believed that the volume deficit after final thinning was more significant than early mortality loss, which was compensated for by renewed growth on residual trees. By assuming that death and windthrow would continue in the stand, he computed a total potential volume loss of 6–13% for a 28-year-old sawlog regime. These values were used by Self & MacKenzie (1995), assuming the same frequencies of root girdling classes, to quantify the effectiveness of stump removal prior to planting on a site where poison thinning of the previous crop had led to an increase in the level of inoculum.

Since these earlier New Zealand studies, the conversion of logged indigenous forest to pine plantation has declined, and the incidence of early mortality due to Armillaria root disease has decreased substantially. Nevertheless, planting continues, and chronic infection remains widely prevalent, though variable in incidence, throughout much of the exotic resource (Hood & Sandberg 1993a, b; Self *et al.* 1998). The changed character of the problem has created a need for a new estimate of the impact of this disease on stand productivity, and data obtained from three comprehensively monitored central North Island trials were employed for this purpose. Two of these trials were used to monitor the development of the disease in first-rotation *P. radiata* on sites cleared of indigenous forest. Further information gathered from these stands made it possible to revisit the earlier growth loss estimates for this kind of forest. The third trial, in a second-rotation stand on a non-indigenous forest site, characterised a type for which growth loss had not yet been estimated, even though it typified much of the anticipated future forest resource in this country.

MATERIALS AND METHODS

Study Sites

The stand used to estimate the growth impact of Armillaria root disease in second-rotation *P. radiata* on a non-indigenous forest site, was a 3.1-ha trial in Kaingaroa Forest established to determine the effect of thinning on the incidence of infection (Hood & Sandberg 1993a; Hood, Kimberley, Gardner, & Sandberg 2002). Trees were planted in 1985, after the harvesting of a 54-year-old crop of *Pinus nigra* ssp. *laricio* (Poir.) Maire. The original vegetation was tussock and fern, with possibly some scrub, but no trees. Early mortality was low (3% of trees), typical for such a stand, but 22% of living trees were infected at age 6 years. The first pre-commercial thinning was undertaken at age 7 years according to the following prescription:

(1) Unthinned (810 stems/ha, not pruned; control)
(2) Thinned to 250 stems/ha, residual trees pruned
(3) Thinned to 250 stems/ha, by whole-tree extraction using a cable (no thinning stumps), residual trees pruned
(4) Unthinned, pruned (to be thinned to 250 stems/ha at age 13 years)
(5) Thinned to 500 stems/ha, pruned (to be thinned further to 250 stems/ha at age 13 years)

However, for the present study, undertaken before the second thinning, there were effectively only three treatments, unthinned (Treatments 1 and 4), thinned to 250 stems/ha

(Treatments 2 and 3), and thinned to 500 stems/ha (Treatment 5). Trees were pruned between ages 7 and 13 years as previously described (Hood, Kimberley, Gardner, & Sandberg 2002).

Of the two *P. radiata* first-rotation trials on ex-indigenous forest sites, one consisted of four rectangular plots 36 m × 28–36 m scattered over an area 2 km across in Tuararangaia Forest (Raungaehe Range, east of Te Teko; Hood & Sandberg 1987, 1989, 1993b). These stands were planted at a stocking of 1100–1500 stems/ha in 1985 (Plot 2) and 1986 (remaining plots), within a year of clearfelling and burning of the previous, partially logged, podocarp-hardwood forest. Selected trees were pruned in four lifts to ca. 6.5–7 m height between ages 4 and 9 years, and unpruned trees were operationally felled at age 5 years in Plot 2 only, leaving a stocking of 450 stems/ha. All plots were thinned at age 11 or 12 years to a final stocking ranging from 250 to 360 stems/ha in different plots. Armillaria root disease had killed 22–35% of the original trees by age 5–6 years, leaving significant gaps in the stand, and total infection in all trees ranged from 54 to 64%. In Plot 2, 67% of stumps were colonised by *Armillaria* species 15 months after the first thinning.

The second first-rotation, ex-indigenous forest trial was that used by Shaw & Calderon (1977) and MacKenzie (1987), being Block E of Beveridge (1966) and Site 2 of Shaw & Toes (1977). A site on the Mamaku Plateau near Rotorua was planted in *P. radiata* in 1966, after clearing and burning of the residual, selectively logged, podocarp-hardwood forest. Incidence of trees infected after thinning to 300 stems/ha was 60-62% at age 10 years, and 56% (including 6% trees that had since died) at age 19 years. The stand was clearfelled at age 29 years.

Field Data

The positions of all trees in the Kaingaroa trial were mapped using aerial photography at the time of trial establishment prior to crown closure (Hood & Sandberg 1993a). Severity of infection was evaluated on all standing trees at ages 6 and 12.5 years (Fig. 2 and 3 in Hood, Kimberley, Gardner, & Sandberg 2002). Diameter at breast height (dbh), tree height, height to the base of the green crown, and pruned height were measured on all trees in July 1998 (age 13 years), prior to a final thinning at age 13.5 years. However, growth data were not taken from trees in the outer row around the inside of each plot, which was treated as a buffer zone, and five trees that had died after the first thinning were also not measured.

The positions of all trees in the Tuararangaia trial were mapped after planting, and mortality was monitored during subsequent years. Severity of infection was estimated on all standing trees in December 1991 (ages 5, 6 years), prior to the first thinning (Fig. 1–4 Hood & Sandberg 1993b), and again, after the final thinning, in November and December 1998 at ages 12, 13 years (Fig. 1). Dbh, tree height, height to the base of the green crown, and pruned height were measured on all standing trees at mid-rotation in September 1998 (ages 12, 13 years), less than 12 months after the final thinning (all residual trees being still living).

Residual trees in the Mamaku trial were evaluated for infection, height, and dbh in 1995 (age 29 years), immediately prior to harvest. Archival dbh and infection data collected from this trial at ages 10 and 19 years were also available to augment information from the other trials (MacKenzie 1987).

Diameters were measured with a diameter tape, and heights optically with a "Forester Vertex". Infection severity was estimated using a light, short-handled grubber to expose the

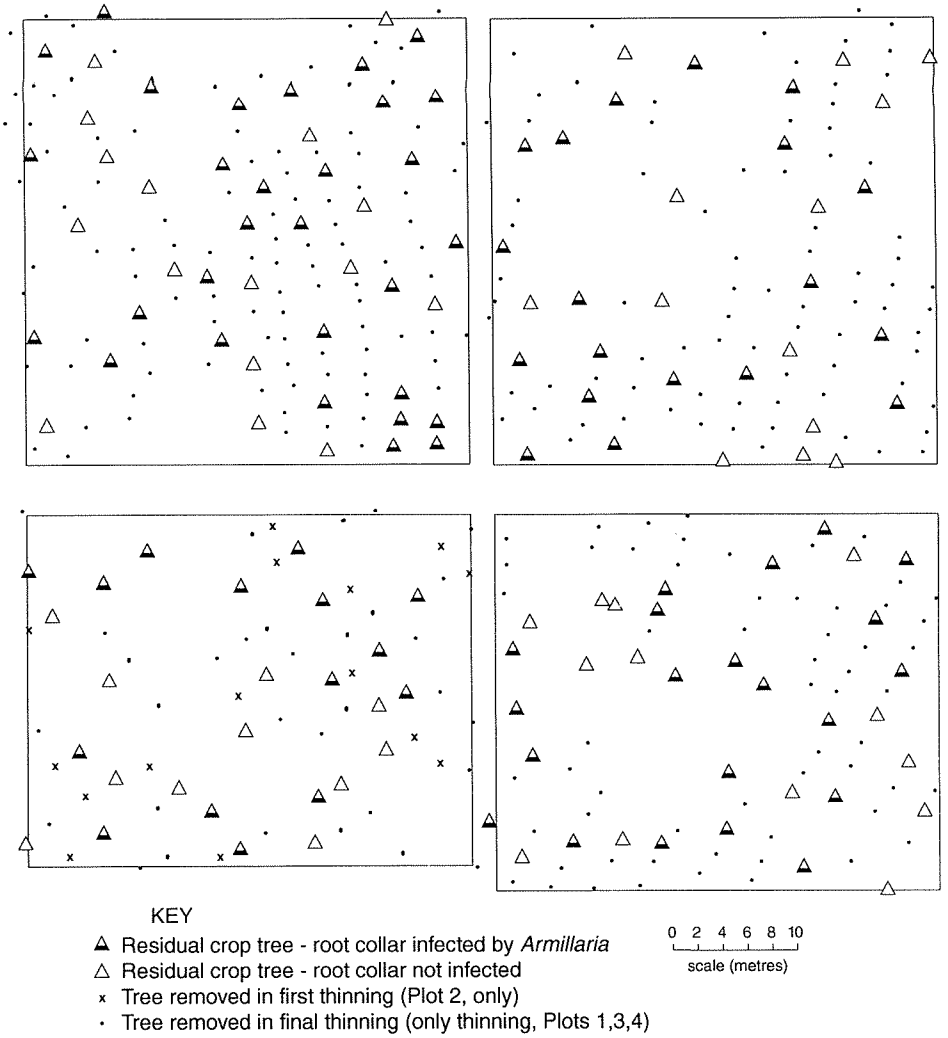

FIG. 1–Residual trees available for operational selection in the Tuararangaia trial after early mortality caused by *Armillaria* species (as at age 5 years in Plots 1, 3, 4; 6 years in Plot 2). Symbols distinguish crop trees retained by forest management (including those infected at the root collar after thinning), trees removed in a single thinning at age 12 years (Plots 1, 3, 4), and trees removed in two thinnings at ages 5 and 13 years (Plot 2). Clockwise from top left: Plots 1, 3, 4, 2 (cf. Figs. 1–4 in Hood & Sandberg 1993b).

root collar in order to estimate the extent of girdling by a zone of resinosis, accompanied by the presence of rhizomorphs. Trees were assigned to one of five girdling classes (0 = 0%; 1 = 1–25%; 2 = 26–50%; 3 = 51–100%; 4 = dead, with *Armillaria*). This technique has been used in previous studies (Shaw & Calderon 1977; MacKenzie 1987), and is believed to give a reliable estimate of true infection of *P. radiata* by *Armillaria* species in New Zealand (Hood, Kimberley, Gardner, & Sandberg 2002). A slightly different scale was used for infection in the Mamaku trial (*see* Results section).

Analyses

The effect of mortality on volume production

Knowledge of the tree positions in the Tuararangaia trial was made use of to assess the impact of the high incidence of early mortality, typical of first-rotation ex-indigenous forest stands, on final-crop volume. Stem volume (m^3) was estimated from height (m) and dbh (cm) using the following general volume equation, which provides good estimates for New Zealand-grown *P. radiata* (A. Gordon pers. comm.):

$$\text{Stem volume} = 0.00004446\ \text{dbh}^{1.829}\ \text{ht}^{1.0319} \quad (1)$$

A SAS programme (SAS Institute Inc. 1989) was developed which sampled both mortality and crop volume by repeatedly placing circular subplots randomly within the trial. The early mortality attributable to Armillaria root disease, and the volume of the crop trees at mid-rotation, were determined within each subplot. The following nonlinear regression function was then fitted using the SAS procedure NLIN:

$$V = a\left[\frac{N_2}{N_1}\right]^b \quad (2)$$

where V is crop volume (m^3/ha), N_1 and N_2 are the stocking density (stems/ha) at planting and after mortality induced by *Armillaria* species, and a and b are regression coefficients. The coefficient a gives the volume corresponding to zero mortality, while the coefficient b determines the effect of mortality on crop volume. For example, $b = 1$ implies that crop volume reduction is directly proportional to mortality, while $b = 0$ implies that mortality has no effect on crop volume. This regression equation was fitted separately for each of the four plots in the trial, providing four independent estimates of the regression coefficients and volume loss. A general equation was also obtained with a global b parameter but separate a parameters fitted for each plot using dummy variables. A sufficient number of simulated subplots was generated to obtain regression parameters with adequate precision for each plot. The simulation was repeated for a range of subplot sizes with radii of 2.5, 5, 7.5, and 10 m.

The mean percentage reduction in volume within a subplot is given by:

$$\begin{aligned}\text{Percentage volume loss} &= 100(1 - {}^{V}/_{a}) \\ &= 100(1 - ({}^{N_2}/_{N_1})^b) \\ &= 100(1 - (1 - {}^{M}/_{100})^b)\end{aligned} \quad (3)$$

where M is the percentage mortality in the subplot. Within each of the four plots in the trial, the mean percentage reduction in volume of the selected crop trees at mid-rotation resulting from early mortality was estimated using the a coefficients from the global b model by:

$$\text{Percentage volume loss} = 100(a - \text{mean}(V))/a \quad (4)$$

Equation 3 was then fitted as a regression relating estimated plot mean volume loss to mortality. Because this regression was fitted to plot means, it was believed to provide the most reliable relationship between the loss in crop volume and mortality.

The effect of infection on tree growth

A regression model was fitted using the SAS procedure GLM to test for significant differences in the growth of individual living trees between Armillaria root disease infection

severity classes in all three trials. The growth variables employed were dbh, height, and stem volume estimated using Equation 1. Data from all trials were processed using the same basic analysis of variance model, which included the following factors:

- A plot effect which accounted for site and silvicultural effects on growth (thinning and pruning)
- Where possible, two competition indices (described below)
- The infection severity score.

To improve the sensitivity of the model, local competition effects were accommodated using x, y grid co-ordinates identifying the locations of all trees in the Kaingaroa and Tuararangaia trials, for which these data were available. Two competition indices were employed. The first, COMP1, a measure of local stocking, was the number of trees within a fixed radius of the subject tree. The second, COMP2, was the average stem volume of all trees within the same fixed radius of the subject tree. A radius of 6 m was used because the testing of a range of values indicated no further improvement in the model fit beyond this distance. The competition indices for the Kaingaroa trial were calculated using the 13-year, post-thinning data. Because positions but not diameters were known for the buffer trees between the measurement plots at Kaingaroa, COMP1 could be determined accurately. However, COMP2 could only be calculated using the non-buffer plot trees, and was therefore determined only approximately for trees near plot edges. In the Tuararangaia trial, only COMP1 was calculated, since the positions, but not the diameters were available for trees removed by thinning at ages 12 and 13 years. No indices could be calculated for the Mamaku trial for which tree position data were not available.

One concern with the above analysis is that the reduction in growth of diseased trees could be exaggerated by competition from neighbouring, disease-free trees. The competition terms in the regression model fitted to the Kaingaroa trial made it possible to correct for this effect.

Firstly, it should be noted that the mean stem volume (m^3) without any adjustment for competition, can be expressed as:

$$V = \mu - k\bar{C} - \sum_{i=1}^{3} l_i p_i \qquad (5)$$

where,

μ is the mean of model effects including the COMP1 term applied for the average stocking, but excluding COMP2 and assuming an infection severity of zero,

k is the coefficient of the competition index term, COMP2, at average local stocking,

$\bar{C}$ is the mean value of COMP2 in the trial

l_i is the coefficient indicating the reduction in volume associated with severity class i (with $i = 1$, 2, or 3),

p_i is the proportion of trees in severity class i.

This formula can be used to predict mean volume, without adjustment for competition, for a stand containing any proportion of trees in each severity class.

To adjust for competition, it is only necessary to notice that the mean of COMP2 will always equal the mean stem volume in the stand. Thus the mean stem volume adjusted for competition is:

$$V = \mu - kV - \sum_{i=1}^{3} l_i p_i \qquad (6)$$

or, $$V = \frac{\mu - \sum_{i=1}^{3} l_i p_i}{1 + k}$$

For example, for a disease-free stand (i.e., $p_i = 0$ for all i), competition-adjusted volume is estimated by:

$$V = \frac{\mu}{1 + k} \quad (7)$$

A hypothetical stand consisting entirely of trees in severity class 1, would have a mean volume adjusted for competition of:

$$V = \frac{\mu - l_1}{1 + k} \quad (8)$$

The mean volume adjusted for competition can be estimated by a weighted mean of terms such as those given in Equations 7 and 8 using the proportion of trees in each severity class as weights. An estimate of the percentage loss caused by the disease can then be obtained by comparing the adjusted mean volume with the adjusted disease-free volume (Equation 7).

RESULTS

The coefficients of the regression equation relating mid-rotation volume of crop trees to mortality caused by *Armillaria* species (Equation 2) derived from the Tuararangaia trial are given in Table 1. These coefficients show that mortality is associated with a reduction in crop volume. For example, for 5-m-radius subplots, the parameter *b* had a mean value across the four plots in the trial of 0.55 with a standard error of 0.10. The hypothesis that there is no association between mortality and crop volume (i.e., $b = 0$), is rejected ($t_3 = 5.66$; $p = 0.011$). Regressions using a global *b* parameter and separate *a* parameters for each plot gave a value of $b = 0.47$ for 5-m-radius subplots (Table 1). These coefficients were used to estimate the loss in crop volume for each plot using Equation 4 (Fig. 2). Equation 3 was then fitted as a regression relating estimated plot mean volume loss to mortality. The coefficients for this model varied slightly with subplot size (Table 1, Fig. 2), and there appears to be little obvious rationale for preferring any particular subplot size. However, because the subplots with radius of 5 m gave the smallest standard errors (Table 1), these were considered to give the most reliable results. The value for *b* using these subplots was close to 0.5. This leads to the following simple rule-of-thumb: "Volume loss equals one minus the square root of survival", where both survival and volume loss are expressed as proportions. At Tuararangaia, the

TABLE 1–Analysis of the effect of mortality caused by *Armillaria* species on crop volume in Tuararangaia trial. Estimated values of the *b* coefficient (Equation 2) for a range of subplot sizes.

Subplot radius (m)	Models fitted separately to each plot		Common *b* model	Model fitted to plot means
	mean(*b*)	s.e.(*b*)		
2.5	0.44	0.13	0.34	0.70
5.0	0.55	0.10	0.47	0.52
7.5	0.51	0.13	0.48	0.50
10.0	0.36	0.14	0.42	0.43

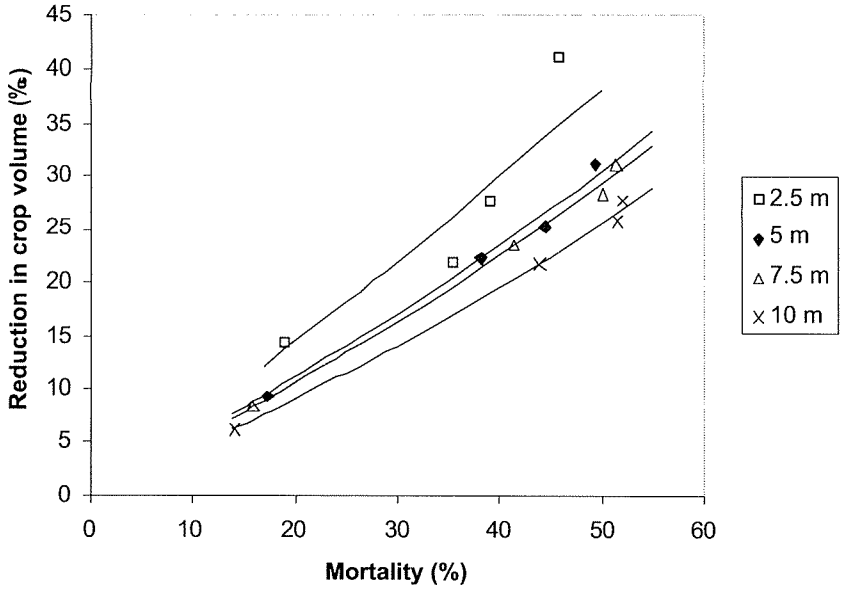

FIG. 2–Reduction in crop volume associated with mortality at the Tuararangaia trial. Results are shown for subplots of four different radii. Regression lines are of the form given in Equation 4.

mean survival after mortality caused by *Armillaria* species was 63%. Using the above rule-of-thumb, predicted volume loss would be $100(1 - \sqrt{0.63})$, or 21%.

Tree growth was significantly associated with severity of infection in two of the trials, Kaingaroa and Mamaku. This relationship is demonstrated for Kaingaroa, for instance, in the analysis of variance table for stem volume (Table 2). Both localised stocking (COMP1) and size of neighbouring trees (COMP2) were included as covariates in this analysis along with their interaction, and all proved to be statistically significant. It was noted that COMP1 and COMP2 were themselves largely unrelated (pooled within-treatment correlation = –0.06), and they could therefore both be validly included in the model. The signs of the coefficients for these terms indicated that both higher local stocking and larger surrounding trees reduced

TABLE 2–Analysis of variance for 13-year stem volume, Kaingaroa trial

Source	d.f.	Sum of squares*	Mean square	F-value	prob > F
Block	3	0.28	0.094	0.81	0.51
Thinning treatment	2	7.22	3.612	31.14	<0.0001
Plot(Block)	14	1.62	0.116		
Comp2	1	0.68	0.682	22.73	<0.0001
Comp1	1	0.21	0.208	6.93	0.0086
Comp1 × Comp2	1	0.19	0.187	6.24	0.0126
Infection score (12.5 yr)	3	0.45	0.179	4.97	0.0020
Residual	1094	32.83	0.030		

*Sums of squares are Type 1

stem volume of a subject tree. The coefficient for the interaction term was negative, indicating that the competing effect of larger neighbouring trees was less pronounced at lower stockings. The interaction between severity of infection and stocking was also tested, but was not statistically significant for any growth variable.

Values for all growth values (least squares means and standard errors) are compared for each infection severity class in Table 3, for each of the three trials. In both the Kaingaroa and

TABLE 3–Least squares means and standard errors for trees in different infection classes*

(a) Kaingaroa trial

Infection score (12.5 yr)	Volume (13 years) (m^3)		dbh (13 years) (cm)		dbh increment (6–13 yr) (cm/yr)		Height (13 years) (m)	
	mean	s.e.	mean	s.e.	mean	s.e.	mean	s.e.
0	0.487 a	0.011	29.5 a	0.4	2.23 a	0.03	19.1 a	0.1
1	0.455 b	0.014	28.6 b	0.5	2.12 b	0.04	18.9 ab	0.2
2	0.438 b	0.019	28.2 bc	0.6	2.12 bc	0.06	18.7 ab	0.3
3	0.418 b	0.027	26.7 c	0.9	1.93 c	0.08	18.4 b	0.4

Infection score (6 yr)	Volume (13 years) (m^3)		dbh (13 years) (cm)		dbh increment (6–13 yr) (cm/yr)		Height (13 years) (m)	
	mean	s.e.	mean	s.e.	mean	s.e.	mean	s.e.
0	0.475 a	0.10	29.2 a	0.4	2.01 a	0.07	19.0 a	0.1
1	0.486 a	0.21	29.7 a	0.7	2.19 a	0.03	19.3 a	0.3
2	0.460 ab	0.23	28.9 ab	0.8	2.21 ab	0.06	19.0 ab	0.3
3	0.409 b	0.22	27.2 b	0.7	2.15 b	0.07	18.4 b	0.3

(b) Tuararangaia trial

Infection score (12 yr)	Volume (12 years) (m^3)		dbh (12 years) (cm)		Height (12 years) (m)	
	mean	s.e.	mean	s.e.	mean	s.e.
0	0.476 a	0.028	28.6 a	0.8	19.9 a	0.4
1	0.490 a	0.034	29.0 a	0.9	20.4 a	0.5
2	0.433 a	0.045	27.1 a	1.2	19.7 a	0.6
3	0.526 a	0.041	29.4 a	1.1	20.7 a	0.5

(c) Mamaku trial†

Infection score (19 yr)	dbh (10 years) (m^3)		dbh (19 years) (cm)		dbh increment (10–19 yr) (cm/yr)	
	mean	s.e.	mean	s.e.	mean	s.e.
0	18.0 a	0.2	39.9 a	0.4	2.42 a	0.04
1	18.1 a	0.3	40.0 a	0.7	2.43 a	0.07
2	18.7 a	0.3	39.3 ab	0.6	2.30 ab	0.06
3	18.1 a	0.4	37.5 b	0.8	2.15 b	0.07
4	18.5 a	0.5	33.8	5.5	1.64	0.52

* Values followed by the same letter within a column do not differ significantly (p=0.05)

† Archival raw data analysed without edge trees; score categories (proportion of root collar girdled, scale shifted for ease of comparison): 0 = 0%; 1 = < 5 cm circumference; 2 = >5 cm to <50%; 3 = > 50%; 4 = dead or windthrown

Mamaku trials there was a trend indicating a significant decrease in dbh, dbh increment, height, and volume with increasing severity of infection, the dbh increments being of similar order in both trials. In the Kaingaroa trial, the growth trends were more pronounced for the 12.5-year severity values evaluated after the first thinning, than for the earlier 6-year values. These trends were not apparent with the data from the Tuararangaia trial.

The values in Table 3 are not adjusted for the effect of competition. Percentage reductions in volume of diseased trees compared with disease-free trees, with and without competition adjustment, are shown in Table 4 for the Kaingaroa trial. Values are calculated for each severity class and for the mix of severity classes in the stand, and the effects of the competition adjustment are shown in Fig. 3. Without adjustment for competition, the predicted loss in volume associated with Armillaria root disease was 3.15%, but after adjustment for competition this was reduced to 2.35%.

TABLE 4–Percentage reduction in volume of infected compared with infection-free trees in Kaingaroa trial. Values are shown for each severity class, and across all classes as represented in the trial. Reductions are calculated with and without competition adjustment.

Infection score (12.5 years)	Unadjusted		Adjusted	
	mean	s.e.	mean	s.e.
1	6.37	2.72	4.79	2.04
2	9.87	3.69	7.42	2.77
3	13.98	5.32	10.50	3.99
Average score*	3.15	0.79	2.35	0.59

* Calculated using the average proportions of trees in each severity class in the trial with even weighting given to each plot.

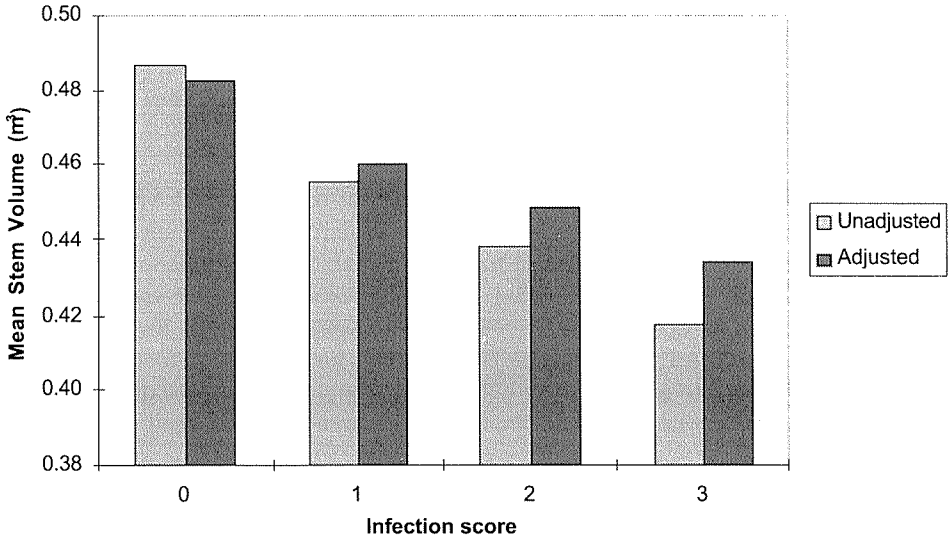

FIG. 3–Mean stem volume at 13 years, for each 12.5-year root collar infection class, Kaingaroa trial data. Estimates are given with and without adjustment for competition.

Although there was no significant interaction between thinning and infection severity score in these analyses, it was thought worthwhile to confirm this by fitting separate regression models to the thinned and unthinned plots in the trial. As expected, the competition adjustment term, k, and the corresponding effect of the competition adjustment, were smaller for the thinned than the unthinned plots (Table 5). Because disease scores were higher on average in the thinned plots, the estimated reduction in volume was greater for these plots (2.53%) than for the unthinned plots (2.00%). To compare directly the two treatments at similar disease levels, volume losses were predicted using the trial mean severity score distribution for both treatments. Under this scenario, predicted volume losses were found to be very similar at 2.48% for unthinned and 2.22% for thinned plots (Table 5).

TABLE 5–Percentage reduction in volume of infected compared with infection-free trees in the Kaingaroa trial evaluated separately for thinned and unthinned treatments. Estimates are given for the actual distribution of scores found in each treatment, and for the average distribution in the trial. Reductions are calculated with and without competition adjustment.

Distribution of infection scores	Treatment	Unadjusted		Adjusted	
		mean	s.e.	mean	s.e.
Treatment mean	Unthinned	3.18	1.04	2.00	0.65
	Thinned	2.94	1.08	2.53	0.93
Trial mean	Unthinned	3.91	1.39	2.48	0.88
	Thinned	2.56	0.92	2.22	0.80

One potential source of bias in this analysis stems from the use of a single assessment of disease severity (at age 12.5 years), and the assumption that all trees that showed no symptoms of the disease were disease-free. It is possible, however, that some of these apparently healthy trees had been infected at some earlier period, and had suffered some reduction in growth. This would tend to lead to an under-prediction of the loss in growth. To test this, the above analysis was repeated but it was assumed that only trees with no symptoms in both assessments (ages 6 and 12.5 years) were truly disease-free. The predicted loss in volume growth was slightly greater than the analysis based on the single assessment, although the difference was not statistically significant. The estimated volume loss adjusted for competition using both assessments was 2.71% (se=0.88) compared with 2.35% (se=0.65) in the single assessment analysis.

The Kaingaroa trial gave the most precise estimates of volume growth loss associated with infection score, as is clearly evident from the standard errors in Table 3. This was primarily because of the large number of trees in the trial, and also because competition indices could be calculated. The competition-adjusted volume reduction estimates from Table 4 can therefore be considered the most reliable estimates currently available of the association between disease level and growth loss. Growth loss predictions for the two, first-rotation, ex-indigenous forest sites, were obtained by applying these to the distribution of infection scores in these trials. The resultant volume loss estimates ranged from 3.85 to 4.43% (Table 6).

DISCUSSION

Various approaches have been used to estimate the effects of root disease fungi on tree growth. In North America, Froelich *et al.* (1977) determined that trees of *Pinus elliottii*

TABLE 6–Predicted volume growth loss caused by disease in the three trials, using a generalised adjustment based on the Kaingaroa trial. This prediction takes no account of the effects of mortality.

Site and treatment	Predicted loss in volume (%)
Kaingaroa trial	2.35
Tuararangaia trial	4.43
Mamaku trial	3.85

Engelmann suffered significant losses in basal area growth if more than half their roots were infected by *Heterobasidion annosum* (Fr.) Bref. These workers deliberately inoculated thinning stumps to induce infection, but in practice also chose infected trees from plots that had not been treated. Other researchers have relied purely on the natural occurrence of root diseases, quantifying growth losses by relating tree size to some measure of disease severity. Thiess (1983) found significant reductions in stem volume in *Pseudotsuga menziesii* (Mirb.) Franco infected by *Phellinus weirii* (Murrill) R.L. Gilbertson. Infected trees were identified by culturing from the stain or decay visible in the stump after felling, and stumps were uprooted to quantify infection. Bloomberg & Morrison (1989) classified infection by *Armillaria ostoyae* (Romagnesi) Herink in stands of *Ps. menziesii* according to the extent of resinosis at the tree base. Volume growth was found to be lower in trees in higher resinosis classes. Lewis (1997) examined the impact of *Inonotus tomentosus* (Fr.:Fr.) S. Teng. on the growth of spruce trees (*Picea* spp.) by sampling roots and grouping trees according to the number of infected roots. With this disease only a slight decline in stem volume growth was associated with infection. The analysis was complicated by a tendency for larger trees to be more commonly infected than smaller individuals.

In New Zealand, Shaw & Calderon (1977) and MacKenzie (1987) also used a significant relationship between a decline in volume growth and the extent of resinosis at the base of *Pinus radiata* trees infected by *Armillaria* species in order to quantify stand growth loss estimates. The study reported here matches the findings of Shaw & Calderon (1977). They believed that in first-rotation stands of *P. radiata* growing on logged indigenous forest sites, the loss in wood production associated with infection by *Armillaria* species was likely to be caused primarily by the formation of mortality patches and the consequent under-utilisation of ground area. They also suggested that the contribution to volume loss due to growth reduction in infected but living trees was lower but still significant. Our mid-rotation analysis of the Tuararangaia trial indicated a volume loss of 21% associated with the early mortality at this site, and an additional loss of just over 4% associated with reduced growth of infected but living trees. The combined volume loss figure of about 25% is slightly less than the value of 29–32% predicted by Shaw & Calderon. There will be additional loss resulting from reduced tree quality caused by the coarser branching associated with low stocking, and from the poorer choice of crop trees due to the lower selection ratio at thinning (Fig. 1). However, this may be partly compensated for in returns from grazing, if cattle are introduced to mortality gaps before they become dominated by unpalatable hardwood shrubs such as wineberry (*Aristotelia serrata* (J.R. & G. Forst.) W.R.B. Oliver). The gaps in stocking due to the high incidence of early mortality are quite obvious in this stand (Fig.1), and clearly explain the resultant loss in volume. These plots were established prior to the clearing of the original indigenous forest cover, before the precise distribution of mortality in the subsequent pine crop could be known, so there is no reason to believe that the gaps are atypical.

However, mortality was insignificant in the Kaingaroa trial situated in a second-crop stand on a site not originally covered in indigenous forest in which ca. 20% of trees were chronically or non-symptomatically infected prior to thinning. Early mortality is usually negligible in second-crop plantations on non-indigenous forest sites, and so any loss in wood volume will be primarily due to a decrease in the incremental growth of infected trees. We calculate the loss in wood volume in this stand to be a little over 2%. This value appears comparatively low, but the economic impact may nevertheless be substantial when spread over larger areas (Hood, Marshall, & Kimberley 2002). The overall growth effect, at the end of the rotation, may also be influenced by the much larger second-thinning stumps, which have potential for boosting infection through their role as a substrate for *Armillaria* species. This remains to be seen.

The model used in the analysis to derive these growth loss values incorporated a correction to account for competition bias. In an uninfected stand, the trees would be conceptually larger than those observed, competition would therefore be more intense, and the mean stem volume would actually be somewhat lower than that predicted by a model where competition was not accounted for. The reliability of the incremental growth loss figures also presupposes that root collar resinosis is an appropriate measure of the overall infection of the root system. Growth loss may be under-estimated, particularly if there is significant undetected infection on trees classed as free from infection (girdling class, 0). However, this is unlikely to be so, as discussed by Hood, Kimberley, Gardner, & Sandberg (2002). The contribution to stand growth loss of Category 1 trees is already comparatively small, suggesting that Class 0 trees are even less likely to have a significant input, even if some root infection should go undetected.

A related issue is the transference of trees between the different infection severity categories with time (MacKenzie 1987; Hood, Kimberley, Gardner, & Sandberg 2002). In this study, estimates of the effects of the disease were generally based on measures of growth determined for each category at single points in time (Table 3, 4, and 5). Potentially this could lead to biased results, particularly as there is an underlying assumption in this analysis that Class 0 trees have been free of the disease at all times prior to the assessment. However, when the Kaingaroa trial was analysed assuming that only trees without symptoms in both 6- and 12.5-year assessments were truly disease-free, the estimated loss in volume of 2.7% was only slightly, and not significantly, higher than the analysis based on the single 12.5-year assessment of 2.35%. This analysis does suggest that, if anything, the volume loss estimates given in Table 4, are likely to be somewhat conservative

The conclusions also assume a causative relationship between infection and growth. Alternatively, the same association might occur in whole or part, regardless of any effect of Armillaria root disease, if smaller trees may simply become infected more readily than larger trees. However, a genuine causative effect is implied by, for instance, results from the dendrometer band study of Shaw & Toes (1977). These authors found steadily diverging diameter growth between 10-year-old infected and healthy *P. radiata* trees of similar crown depth, carefully selected to have comparable average diameter and height (means and variance) at the start of the monitoring period (cf. Fig. 1 MacKenzie 1987). The analysis of thinned and unthinned plots in the Kaingaroa trial also provides some indirect evidence for a causative relationship between infection and growth. The unthinned plots will contain suppressed trees at mid-rotation, but few will be present in thinned plots. Under the

assumption that the relationship is not causative, it might be expected that the greater availability of smaller, suppressed, supposedly more readily infected trees in the unthinned plots would result in a larger apparent volume reduction, compared with disease-free trees, than in thinned plots. However, after adjustment for competition effects, the association between growth loss and infection was similar for both treatments (Table 5).

The estimates of growth loss from the two, first-rotation, ex-indigenous forest trials were slightly more speculative than those obtained from the Kaingaroa trial, mainly because of the smaller numbers of trees. This is probably also the reason why the growth trends apparent in the other trials were not evident in the Tuararangaia trial, where growth data were available only for the trees remaining after the final thinning. The standard errors of growth estimates for this trial were much higher than for the other two trials, particularly the Kaingaroa trial which gave the most precise values (Table 3). However, as with the Kaingaroa trial, it should be possible to make further use of the Tuararangaia trial nearer the end of the rotation period. It proved impractical to analyse the final data-set from the Mamaku trial collected in 1995 (age 29 years), just prior to felling. Some tags were missing, a number of trees could no longer be identified, and the earlier purposely non-random selection of a large proportion of the most heavily infected individuals for stem analysis had biased the data from the residual trees for possible subsequent use. However, useful comparisons were made using the 10- and 19-year-old data.

At present, options are limited for the treatment of sites affected by Armillaria root disease, whether or not the current or succeeding stand is considered. Several potential methods are under investigation, which involve reducing or denying the pathogen access to the stump-wood substrate, and using planting stock that is physiologically or genetically less susceptible. The intention is to develop an integrated management procedure for reducing the growth impact of Armillaria root disease and for rehabilitating sites that are more severely infested.

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