A STATISTICAL APPRAISAL OF *ARMILLARIA* ROOT ROT IN NEW ZEALAND PLANTATIONS OF *PINUS RADIATA*

J. B. van der PAS

Forest Research Institute, New Zealand Forest Service,
Private Bag, Rotorua, New Zealand

The development pattern of mortality due to **Armillaria** root rot in first rotation young **Pinus radiata** D. Don, planted on sites cleared of indigenous forest, was analysed statistically. Rates of progress of mortality differed between study sites, but there were no significant relationships between mortality rate and various stump characteristics such as species and stump density. The pattern of mortality increase over 3 years did not support the concept of radially expanding infection centres, but instead resulted largely in consolidation of existing disease patches and the appearance of new disease centres. It is therefore postulated that initial development of mortality does not occur from contact between pines but rather occurs at random within disease patches, which are delineated by the old growth stumps and residual root systems colonised by **Armillaria** root rot.

INTRODUCTION

Armillaria root rot has caused damage to many recently established plantations of *Pinus radiata* D. Don stands in New Zealand (MacKenzie & Shaw 1977) on sites freshly cleared of cutover podocarp/tawa forest in the central part of the North Island (Beveridge 1973, 1974; Beveridge *et al.* 1973). Mortality usually appears in the form of patches of dead trees early in the life of the stand (Beveridge 1973), and may reach more than 50% in the first 5 years after planting (van der Pas, unpubl. data). Such high localised mortality reduces yield and may increase production costs (Shaw & Calderon 1977).

Field dynamics of root rot development around residual stumps of the former indigenous cover is not thoroughly understood (Roth *et al.* 1979). MacKenzie & Shaw (1977) examined patterns of mortality developing within 27 months of planting and found that mortality was grouped around stumps of *Beilschmiedia tawa.* They suggested that mortality could continue in radially expanding infection centres.

More recently Roth *et al.* (1979) re-emphasised the importance of *B. tawa* stumps and also those of species of Podocarpaceae in initiating infection centres, but did not support the earlier suggestion that infection centres expanded radially with time. Rather, they suggested that seedling death from *Armillaria* root rot was concentrated within the area occupied by roots of old tawa or podocarp trees. Within this area mortality was essentially concurrent among seedlings near or away from the stump. An illusion

Received 27 March 1980; revision 2 February 1981

of progressive enlargement of disease foci with time was created through a delayed wave of mortality moving outward from the stump to the limits of its root system.

The objective of this study was to test the above concepts by evaluating mortality and developmental patterns of *Armillaria* root rot in *Pinus radiata* trees planted 5 years earlier on sites freshly cleared of native forest. This included a re-examination of the relative importance to disease development of stumps of *B. tawa* and other species.

TRIAL HISTORY

Study areas

The study site was located on the Mamaku Plateau on an area formerly covered by podocarp/mixed hardwood forest. The area was logged in the early 1950s for usable timber, and the remaining stems were clearfelled and broadcast burnt in 1974. Five plots were established in 1974 to determine levels and patterns of mortality caused by *Armillaria* root rot in *P. radiata* plantations established on sites converted from indigenous forest of different species composition (MacKenzie & Shaw 1977; Shaw & Calderon 1977).

Plot description of plots 1 to 4 are cited, with permission, from the caption to fig. 1 (p. 361) of Shaw & Calderon (1977). Plot 1 (50 × 50 m) was off Leslie Road on an area formerly occupied by high density tawa (*Beilschmiedia tawa*). The plot was planted at 1 × 1 m spacing (2490 trees) in July 1975. Plot 2 (40 × 60 m) was adjacent to the Pukerimu stream off McPherson Road on an area formerly occupied mainly with large, old growth rimu (*Dacrydium cupressinum*). The plot was planted at 1 × 1 m spacing (2398 trees) in July 1975. Plot 3 was off Tunnel Road on an area formerly occupied by mixed hardwoods with approximately 50% tawa. The plot was planted at 1 × 1 m spacing (3597 trees) in July 1974 and is thoroughly described by MacKenzie & Shaw (1977). Plot 4 is adjacent to plot 1, but on an area occupied mainly by *Weinmannia racemosa.* The plot was planted at 1.8 × 2.4 m spacing (538 trees) in July 1975. Plot 5 is adjacent to plot 2, and was planted at 1.8 × 2.4 m spacing (415 trees) in July 1975. The area was formerly occupied mainly with tawa and old growth rimu.

Before planting, unburnt logs and slash were removed from the plot areas of plots 1, 2, and 3; debris was not removed in plots 4 and 5. The densely planted plots (1, 2, and 3) were gridded with stakes to facilitate planting as accurately as possible at a spacing of 1 × 1 m.

METHODS

Recording of mortality

Tree mortality was recorded quarterly up to 30 months after planting and annually thereafter as described by MacKenzie & Shaw (1977). Mortality percentages for the first 2 years of plots 1–4 are given in fig. 1 of Shaw & Calderon (1977). Plots were thinned approximately 2½ years after planting. Every second tree of the original planting was scheduled for thinning and those still living were removed (MacKenzie & Shaw 1977).

Indigenous stumps were measured for diameter at stump base. Stumps greater than 10 cm in diameter and all pine trees were located on 1 : 78 scale plot maps. Residual

stumps were categorised by species: tawa or non-tawa. The number of species in the non-tawa category ranged from 10 to 16 per plot. Stumps within 1 m of each other and which were of the same species category were treated as being one stump. Positions of pine trees killed by *Armillaria* root rot were entered on maps by an integer representing the assessment period during which they died (Fig. 2). Thinned trees which were infected were not considered in the analyses, but percentages are given for each plot with the summarised plot data in Table 1.

TABLE 1—Summary of plot data

Characteristic	Plot				
	1	2	3	4	5
Plot area (m^2)	2500	2400	3600	2500	2400
No. of planted **P. radiata**	2490	2398	3597	538	415
No. of tawa stumps	44	6	69	27	11
No. of non-tawa stumps	45	34	89	15	27
Total no. of stumps	89	40	158	42	38
Stump basal area (m^2) — tawa	8.98	1.90	13.30	4.92	6.79
Stump basal area (m^2) — non-tawa	3.04	13.65	8.15	5.07	7.40
Total stump basal area (m^2)	12.02	15.55	21.45	9.99	14.19
Total stump basal area (m^2/ha)	48.08	64.79	59.58	39.96	59.13
Infection of thinned trees (%)	24	23	28	22	26

Mortality incidence and progress rate

The mortality incidence (x) for each assessment period before thinning was calculated as the proportion of total trees planted.

The formula $x = n/N$ was used, where x = mortality incidence, n = number of trees killed by *Armillaria* root rot, and N = number of trees planted less the number of dead trees due to other causes. To calculate mortality incidence for assessment periods after thinning the following formula was developed:

$$x = (x_1 + x_2)/2, \text{ where } x_1 = x/N \text{ and } x_2 = \frac{2n - n'}{N},$$

n' = number of trees killed 24 months after planting.

As the sum of x_1 and x_2 is respectively an under and over estimate of x, it was halved to give a realistic estimate of mortality incidence in successive assessment periods after thinning. Mortality progress rate was computed as the increase of mortality over time after procedures as described by Van der Plank (1963). Mortality progress rates were fitted to linear regressions. Regression equations were tested by analysis of covariance and differences compared by Student-Newman-Keuls' test.

Association of mortality with tawa and non-tawa stumps

Distances between each living and dead tree and nearest tawa and non-tawa stumps were measured on plot maps and ground checked where necessary. Mean distances to tawa and non-tawa stumps were calculated for killed and healthy trees in each assessment

period for each plot and compared by t-test. Trees within equal distance from stumps of different species category, i.e., less than 50-cm difference, were excluded from analysis.

The variation of distance of killed trees from nearest stump with time after planting was examined by regression analysis. Linear regressions in the form of $Y = b_0 + b_1 X$ were used, where Y = distance of killed tree from nearest stump, X = time, and b_1 = regression coefficient. The slope of the regression line expresses quantitatively the dependence of distance on time. Change of slope with time was examined by t-test for significance. Mortality frequency around tawa and non-tawa stumps was examined from joint occurrences of stumps and killed trees in 100 randomly selected 4 × 4 m sample quadrats per plot. Observed frequecies of stump category and the associated number of killed trees were tested to their expected frequencies by χ^2 goodness of fit. This procedure was repeated for each successive assessment period.

Pattern of stumps and trees

Association between stump categories and killed and healthy trees was examined by methods described by Pielou (1961) for 2-species populations as follows. Segregation was approached by nearest neighbour relationships which was examined for each stump and for 200 randomly selected trees per plot. For stump populations, 4 classes of nearest neighbour relationships were distinguished: tawa : tawa, tawa : non-tawa, non-tawa : tawa, and non-tawa : non-tawa. For tree populations, the 4 classes were: killed : killed, killed : healthy, healthy : killed, and healthy : healthy. Frequencies of occurrence were tested by χ^2 test with one degree of freedom and Yates correction (Pielou 1961).

Randomness of stump location was tested using the "coefficient of dispersion" (Greig-Smith 1964). Test parameters were calculated from 100 randomly-thrown 4 × 4 m sample quadrats per plot. The Poisson series with the observed mean of either category of stump were then calculated and observed numbers of quadrats containing 0, 1, 2, . . ., n individuals were compared with their random expectation. The mean : variance ratio was calculated and the difference from unity examined by t-test following Skellam (1952).

The ratio test was used to test the randomness of location of both stump categories (Holgate 1965a, b). The test statistic, Z, was based on the sample mean of the ratio of the squares of the distances to the nearest and second-nearest stump from 100 random sample points. Randomness was rejected when the sample mean ratio differed from its expectation by more than 1.96 times its sampling standard deviation.

Spread of mortality

Increases in area of patches of killed trees with time were examined by methods suggested by Pielou (1963). These methods assume that a certain proportion (π) of the site consists of patches, within which a proportion (v) of trees is killed. The remainder of the site, i.e., the gaps, is assumed to contain no killed trees. To estimate π and v, a grid of 3 × 3 m contiguous quadrats was layed over the plot maps and the number of killed and living trees within each quadrat recorded. The method requires that quadrats must be sufficiently smaller than average patch size of killed trees to assure that most quadrats lie wholly inside or wholly outside a patch. It also assumes that a tree's chance of being killed is unaffected by its degree of isolation from its

neighbours. This prerequisite was verified for each plot by a χ^2 test comparing difference between "sparse" and "dense" quadrats.

Distributions of killed trees within quadrats were fitted to a binomial expectation which was derived in terms of π and v. From this, the maximum likelihood estimates of π and v and their approximate variances were obtained by an iterative method.

In all plots some scattered mortality occurred outside the diseased patches, i.e., in gaps. Hence the model was adapted to estimate also the proportion of mortality in gaps (w). Using this procedure π, v, and w were estimated for 1976 and 1980. An increase in π over this period indicates that patches of killed trees had increased in area or that new patches had emerged.

An increase in v or w indicates that the proportion of diseased trees had increased within patches and gaps respectively.

RESULTS

Tree mortality began approximately 6 months after planting and continued during following years (Table 2). Mortality incidence was highest in plots 1 and 5 (a densely and normally planted plot respectively) (Table 2). Mortality was lowest in plot 4, but no differences between rate of progress of mortality could be demonstrated between plots 2 and 3 (Table 3).

In all plots there was a very good fit of mortality progress with time to linear regression (Table 3), demonstrating that mortality progress rates were constant before and after thinning, but were not constant between periods. Mortality progress rates dropped significantly in all plots after thinning with the greatest reduction occurring in densely planted plots (Fig. 1). Mortality progress rate increased fastest in plot 5 during both periods before and after thinning.

TABLE 2—Incidence of mortality of **P. radiata** caused by **Armillaria** root rot from 8 to 60 months after planting in plots 1–5

Months after planting	Plot 1		2		3		4		5	
	n*	1/1-x†	n	1/1-x	n	1/1-x	n	1/1-x	n	1/1-x
8	218	1.09	82	1.03	144	1.04	8	1.008	16	1.04
10	300	1.14	151	1.05	216	1.06	12	1.02	24	1.06
13	366	1.17	192	1.08	252	1.07	17	1.029	33	1.086
16	434	1.21	208	1.09	306	1.09	24	1.04	52	1.14
20	583	1.30	334	1.16	432	1.14	46	1.08	77	1.227
24	638	1.34	395	1.20	569	1.19	56	1.10	93	1.288
Thinning										
29	692	1.40	415	1.21	597	1.20	64	1.12	94	1.29
36	702	1.42	432	1.23	630	1.23	73	1.15	105	1.36
39	728	1.45	449	1.25	648	1.24	80	1.20	109	1.38
48	771	1.51	473	1.27	669	1.26	93	1.22	119	1.48
60	796	1.56	481	1.28	686	1.27	111	1.26	127	1.53

* n is the number of trees killed by **Armillaria** root rot

† x is the proportion of trees killed by **Armillaria** root rot

TABLE 3—Analysis of covariance testing for differences in average mortality progress rates between plots in the periods before and after thinning

Plot	b_0	b_1 [1]	S_b [2]	r^2 [3]	SNK-test [4] Slope [2]	SNK-test [4] Level
A. Before thinning						
5	0.08	0.166	0.009	0.986		
1	0.01	0.155	0.007	0.987		
2	0.05	0.114	0.005	0.991		
3	0.02	0.090	0.005	0.964		
4	0.04	0.074	0.004	0.959		
B. After thinning						
5	0.125	0.062	0.003	0.979		
4	0.129	0.051	0.002	0.966		
1	0.239	0.041	0.002	0.959		
2	0.153	0.020	0.001	0.927		
3	0.152	0.016	0.001	0.967		

(1) Regression coefficient and standard error computed from $y = b_0 + b_1 x$ where $y = \log_e [1/(1-x)]$, x = time

(2) Slopes before and after thinning were significantly different for each plot

(3) r^2 values significant at the 1% significance level

(4) Vertical lines indicate regression coefficients and adjusted means for $\log_e [1/(1-x)]$ not different at the 5% significance level.

Association of mortality around stumps

The mean distance of healthy trees to the nearest stump of each category was significantly higher than that of killed trees in all plots demonstrating a concentration of mortality around stumps. On the other hand, there was no significant difference between the mean distance of either killed or healthy trees to the nearest tawa or non-tawa stump (Table 4a).

Mean distances of killed trees to nearest stump did not differ within successive assessment periods thus no change of distance of killed trees from stumps with time could be demonstrated.

On all plots mortality frequency around tawa stumps did not differ from that around non-tawa stumps within each successive assessment period (Table 4b).

Pattern of stumps and trees

Stump density differed greatly among plots and was highest in plot 3 (438 stumps/ha) and lowest in plot 5 (158 stumps/ha). Basal area of stumps was highest in plot 3 (36.9 m^2/ha) and lowest in plot 2 (7.9 m^2/ha).

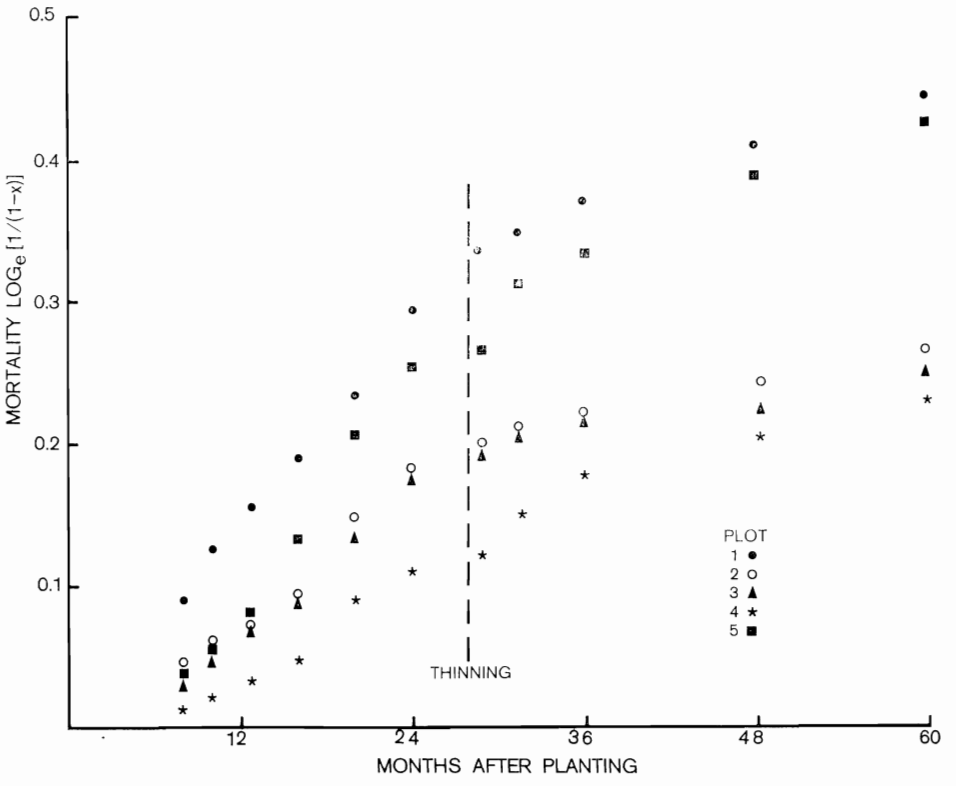

FIG. 1—The progress of mortality in **Pinus radiata** caused by **Armillaria** root rot up to 5 years after planting.

Tawa and non-tawa stumps were randomly distributed within plots except in plot 2, where non-tawa stumps seemed to be clustered (Table 5). However, segregation could not be demonstrated in this plot (see also Fig. 2). Tawa and non-tawa stumps were unsegregated within plot areas except in plot 3 where segregation was highly significant.

In plots 1, 3, and 5 killed and healthy trees were unsegregated or more coloquially, mingled together, whereas they were partially segregated or less mingled in plots 2 and 4.

Spread of mortality

In the period from 1976 to 1980 the proportion of killed trees within patches and gaps increased significantly in plots 1, 4, and 5 (Table 6). In plots 2 and 3, a significant increase of the proportion of plot area consisting of patches was caused by an increase in the area of existing patches and appearance of new patch areas. The proportion of patch area increased only marginally in plots 1, 4, and 5.

Increase of plot mortality in plots 1, 3, 4, and 5 was affected mainly by a significant increase in the proportion of dead trees within existing patches.

In plots 4 and 5 the increase of proportion of killed trees in gaps was marginally significant.

TABLE 4A—Mean distance (in m) of healthy trees ($\bar{d}_h$) and killed trees ($\bar{d}_k$) to nearest stump and their test criteria

Plot	Tawa		Non-tawa		Regression of d_k on time	
					Tawa	Non tawa
	$\bar{d}_h$	$\bar{d}_k$	$\bar{d}_h$	$\bar{d}_k$	t_{b_1} (1)	t_{b_1}
1	3.2	2.7	2.9	2.4	0.327	0.536
2	3.9	2.9	4.3	3.0	0.695	0.763
3	3.5	2.5	3.4	2.1	0.129	0.429
4	4.4	3.2	5.1	3.5	0.631	0.784
5	5.4	3.7	5.1	3.9	0.775	0.812
(2) $t_{\bar{d}_k - \bar{d}_h}$	5.584**		6.457**			
(3) $t_{\bar{d}_h}$	0.398					
(4) $t_{\bar{d}_k}$	0.144					

(1) t-test of regression coefficients

(2) paired t-test between $\bar{d}_h$ and $\bar{d}_k$ of tawa and non-tawa stumps

(3) paired t-test of $\bar{d}_h$ between tawa and non-tawa stumps

(4) paired t-test of $\bar{d}_k$ between tawa and non-tawa stumps

** significant at the 1% significance level

TABLE 4B—Test criteria of difference of mortality frequency around tawa and non-tawa stumps ($\Sigma\chi^2$ is the sum of χ^2 values of 12 assessment periods)

		Plot				
	df	1	2	3	4	5
$\Sigma\chi^2$	11	12.11	11.29	13.54	12.76	10.84

TABLE 5—Summary of test criteria on the pattern of the residual stumps and killed and healthy trees within plots

	Plot				
	1	2	3	4	5
Segregation test (stumps)					
χ^2	3.78	1.37	7.32**	0.30	0.10
Segregation test (trees)					
χ^2	2.92	8.24**	3.65	9.38**	2.17
Coefficient of dispersion (stumps)					
Mean : variance	1.40	1.81	1.18	1.29	1.06
t	1.52	3.04**	0.69	1.80	1.53
Ratio test (stumps)					
tawa $\bar{Z}_{12}$	0.501	0.863*	0.602	0.630	0.593
non-tawa $\bar{Z}_{12}$	0.532	0.932*	0.609	0.498	0.610

* significant at the 5% level ** significant at the 1% level

FIG. 2—Tree mortality around tawa and non-tawa stumps from 1975 to 1980 in plot 2.

TABLE 6—Maximum likelihood estimates of π, v, and w in 1976 and 1980 for plots 1–5

	Plot									
	1		2		3		4		5	
	1976	1980	1976	1980	1976	1980	1976	1980	1976	1980
π	0.20	0.32	0.29	0.49*	0.39	0.53*	0.09	0.15	0.39	0.44
v	0.59	0.76*	0.36	0.35	0.32	0.52*	0.20	0.57*	0.32	0.68*
w	0.23	0.41*	0.06	0.07	0.06	0.07	0.04	0.15*	0.03	0.13*

* difference beween 1976 and 1980 significant at the 5% significance level

DISCUSSION

The existence of residual indigenous stumps was considered the influencing factor for occurrence of *Armillaria* root rot in planted *P. radiata* trees. The detection and analyses of the pattern of location of residual stumps was a prerequisite to some test statistics, and a starting point for determining the factors associated with the spread of the disease.

Stump and tree populations were examined by techniques commonly used in quantitative plant ecology. The validity of the methods may be limited as was detailed by Greig-Smith (1964) and Gounot (1969).

Patch size and proportion of killed trees within patches during consecutive periods were first examined using random line transects. The results using stochastic models (Pielou 1965; Vithayasai 1971) did not lead to constructive interpretation and were conflicting with results obtained by quadrat sampling. The transect method was apparently misleading because certain assumptions, notably a geometrical distribution of run lengths, were not met. The quadrat method, using modified binomial expectation, was likewise not completely free from objections since it was dependent on assumptions on the nature of patches. For instance, there was no real evidence that the number of individuals per patch was distributed randomly since patch size was undefined. Therefore choice of 3 × 3 m size quadrats was arbitrary and could only be selected after testing of several quadrat sizes. Yet validity of inferences may be subject to reservation and could be judged only in relation to other relevant test results.

Mortality was related to stumps of tawa and non-tawa species. A further breakdown of the non-tawa category for empirical association between mortality and single species proved unrewarding and was aborted. Mortality around stumps was examined using distance to nearest stump rather than by series of equidistant contours around stump centres (Shaw & McKenzie 1977). Since such stump-centred contours were overlapping at a distance of 3 m, it was questioned that the contour approach may lead to bias within stump populations of uneven densities (plots 2, 4, and 5) or when different kinds of stumps were segregated (plot 3). Selection of isolated stumps on the other hand (cf Roth *et al.* 1979) may give a partial sample with potential for unknown bias.

Mortality appeared to be aggregated around tawa and non-tawa stumps quantitatively to the same extent. This was compatible with findings by Leach (1939) and Gibson (1960) in Africa, who showed that nearly all common species in an indigenous mixed forest were susceptible to *Armillaria* and that their role as a food base was largely determined by the rate at which stump roots perish in the soil. Earlier findings by

MacKenzie & Shaw (1977) also do not conflict since they indicate that mortality was strongly associated with sporophore-bearing stumps rather than with *B. tawa per se*. MacKenzie & Shaw (1977) found that approximately 40% of all stumps were bearing sporophores 2 years after planting in plot 3. Unfortunately no assessments were made of location of infected indigenous stumps in other plots and consequently no comparisons could be made.

The existence of any significant causal relationship between density of tawa or non-tawa stumps and mortality could not be established (see Tables 1 and 2). Plot 1 with high density tawa showed higher incidence of mortality than plot 2 with low density tawa. Mortality incidence and increase of mortality rate in plots 2 and 3 exhibited no difference, whereas tawa density in plot 3 closely matched that of plot 1 (Table 1). However, stump basal area, composition, and pattern differed highly between plots 2 and 3 (Tables 1 and 4a). After thinning, the mortality progress rate increased faster in plots 5 and 4 relative to the densely planted plots 1, 2, and 3. This was probably attributable to unburnt logs and slash in these plot areas, which served as additional inoculum. This in turn may have increased mortality in gaps (Table 6) which may have resulted from food bases too small to generate distinct patches. Small stumps, debris, or even non-woody plants, such as *Cortaderia fulvida* (see Shaw *et al.* 1976b), may play a role, however minor, in the infection process of *Armillaria* root rot. MacKenzie & Shaw (1977) confirm that this debris was colonised by *Armillaria* and associated with early seedling mortality. However, this experiment did not allow further quantification of stump and root biomass on amount or location of inoculum.

Since mortality rates increased linearly it appears that killing was associated with the initial inoculum, rather than by disease spread through root contact between pines. This would indicate that the epidemic of the disease did not generate its own inoculum during the period of observation. Since $\log_e [1/(1-x)]$ was plotted against time it follows that the slope of the curve measured the absolute rate of increase of mortality. This does not imply that the source of inoculum remained constant but rather that the fungus did not spread in significant amounts from tree to tree. This agrees with findings by Roth *et al.* (1979), who consider such pine-to-pine spread limited, if present at all. Root systems of residual stumps were assumed to be the only source of inoculum, and there was no spread by spores. Based on these assumptions, the proportion of trees killed during a certain time interval may therefore be expected to be proportional to the amount of initial inoculum. Hence the slope of the curve will increase with more inoculum and the curve would rise more steeply (e.g., plot 5). Plot differences in the mortality progress rate may therefore be caused in the first instance by differences in the amount of initial inoculum. This again may be related directly to the quantity of localised infected roots of living indigenous trees before felling.

After felling of the indigenous trees, some of the initial inoculum may die out; alternatively, there may be a buildup through growth of rhizomorphs, or by subsequent colonisation of the entire root system (Shaw 1980). It may be understood that foci or patches of concentration of mortality appear because of uneven distribution of concentrations of the fungus in the plot area. Consolidation of the inner part of the patch *in sensu* (Roth *et al.* 1979), may proceed concurrently near and away from the stumps in a random fashion, since there was no gradient of the distance to stumps with time. Reference may be made to Roth *et al.* (1979) who give detailed views and hypotheses

of the mechanisms of patch development in and beyond the inner patch, which are partly in agreement with above findings.

Results obtained elsewhere report of radially progressing patches in many instances caused by tree-to-tree spread from roots. Shaw *et al.* (1976a) report radially progressing patches by root-to-root contact which were predominantly found in naturally regenerated *Pinus ponderosa* in the USA. In ponderosa pine, Shaw (1980) considered that the disease centres continued to enlarge by spread from pine to pine. Huntly *et al.* (1961), in Canada, suggested radial spread in *Pinus resinosa* and *Pinus strobus* from the infected source. Swift (1972) demonstrated that killing by spread from pine to pine increased logarithmically in a *Pinus elliottii* stand which may be partially attributed to a very high incidence of original inoculum.

In this study, spread through root contact was not expected to have taken place before thinning. There may have been some root-to-root contact in densely planted plots just before thinning but this could not be verified, since root systems of removed trees exhibited no grafting at the time of thinning.

A rather abrupt decrease of mortality rates after thinning may result from compound effects of removing the infected trees (Table 1), decrease of potential hosts, and gradual increase of disease tolerance with age, as suggested by MacKenzie & Shaw (1977).

Elongating lateral roots of pines will likely be in contact throughout the study stands in the next few years. As a result, the disease may then continue by spread from pine to pine and spread may become compounded rather than simple (Van der Plank 1963).

This view is compatible with that of Roth *et al.* (1979), who suggested that enlargement of the patch may occur beyond the old root zone to form an outer part. If that occurs then spread will be continued by a different means, i.e., radially from pine to pine and therefore probably at a different rate (i.e., logarithmically). It may be anticipated, though, that killing of the trees will be impeded by increasing tolerance to the fungus with tree age.

CONCLUSIONS

1. Mortality of *Pinus radiata* caused by *Armillaria* root rot was aggregated around stumps of tawa and non-tawa species to the same extent.
2. Relationships between levels of mortality and stump basal area or stump density were not apparent.
3. Mortality during 1977–1980 resulted from increases in the proportion of killed trees within existing disease patches, the appearance of new patches, and, to a lesser extent, increases in proportion of killed trees in gaps.
4. Within disease patches the spread of mortality occurred at random. At any one time mortality was not more prevalent near stumps than away from them.
5. Mortality progress rates decreased after thinning. This possibly resulted from the decrease of potential hosts, removal of infected trees, and increased tree tolerance with age.
6. Future spread of mortality may take place by root-to-root contact between pine trees and not only from contact with primary inoculum. If so, the patches will increase radially at a different rate from that found here.

ACKNOWLEDGMENTS

The author gratefully acknowledges Mr M. MacKenzie and Dr C. G. Shaw III who initiated and established these experimental plots. I would like to thank Mr M. O. Kimberley for assistance with the statistical analysis. I am indebted to the official referees and particularly to Dr C. G. Shaw III for his useful criticism and helpful suggestions of the draft of the manuscript. The assistance of FRI technicians who carried out most of the field work is also gratefully acknowledged.

REFERENCES

BEVERIDGE, A. E. 1973: Mortality in young plantations on sites cleared of indigenous forest. Pp. 32–3. **In** "New Zealand Forest Service Report". Forest Research Institute, 1972.

——— 1974: **In** "New Zealand Forest Service Report". Forest Research Institute, 1973.

BEVERIDGE, A. E.; KLOMP, B. K.; KNOWLES, R. L. 1973: Grazing in young plantations of radiata pine established after clearing logged and reverted indigenous forest. **New Zealand Journal of Forestry 18:** 152–6.

GIBSON, I. A. S. 1960: **Armillaria** root rot in Kenya pine plantations. **Empire Forestry Review 39:** 94–9.

GOUNOT, M. 1969: "Methodes d'Etude Quantitative de la Vegetation". Masson et Ae. Paris.

GREIG-SMITH, P. 1964: "Quantitative Plant Ecology". 2nd edn. Butterworths, London.

HOLGATE, P. 1965a: Some new tests of randomness. **Journal of Ecology 53:** 261–6.

——— 1965b: Tests of randomness based on distance methods. **Biometrics 52:** 345–53.

HUNTLY, J. H.; CAFLEY, J. D.; JORGENSEN, E. 1961: **Armillaria** root rot in Ontario. **Forestry Chronicle 37:** 228–36.

LEACH, R. 1939: Biological control and ecology of **Armillaria mellea** (Vahl.) Fr. **Transactions of the British Mycological Society 23:** 320–9.

MacKENZIE, M.; SHAW, C. G. III 1977: Spatial relationships between **Armillaria** root rot of **Pinus radiata** seedlings and the stumps of indigenous trees. **New Zealand Journal of Forestry Science 7:** 374–83.

PIELOU, E. C. 1961: Segregation and symmetry in two species populations as studied by nearest-neighbour relationships. **Journal of Ecology 49:** 255–69.

——— 1963: The distribution of diseased trees with respect to healthy ones in a patchily infected forest. **Biometrics 19:** 450–9.

——— 1965: The spread of disease in patchily-infected forest stands. **Forestry Science 11:** 18–26.

ROTH, L. F.; SHAW, C. G.; MacKENZIE, M.; CROCKETT, F. H. 1979: Early patterns of **Armillaria** root rot in New Zealand pine plantations converted from indigenous forest — An alternative interpretation. **New Zealand Journal of Forestry Science 9:** 316–23.

SHAW, C. G. III 1980: Characteristics of **Armillaria mellea** on pine root systems in expanding centres of root rot. **Northwest Science 54:** 137–45.

SHAW, C. G. III; CALDERON, S. 1977: Impact of **Armillaria** root rot in plantations of **Pinus radiata** established on sites converted from indigenous forest. **New Zealand Journal of Forestry Science 7:** 359–73.

SHAW, C. G. III; ROTH, L. F.; ROLPH, L.; HUNT, J. S. 1976a: Dynamics of pine and pathogen as they relate to damage in a forest attacked by **Armillaria**. **Plant Disease Reporter 60:** 214–18.

SHAW, C. G. III; SIJNJA, D.; MacKENZIE, M. 1976b: Toe toe (**Cortaderia fulvida**), a new graminaceous host for **Armillaria** root rot. **New Zealand Journal of Forestry 21:** 263–8.

SKELLAM, J. G. 1952: Studies in statistical ecology. 1 Spatial pattern. **Biometrika 39:** 346–62.

SWIFT, M. J. 1972: The ecology of **Armillaria mellea** Vahl (ex Fries) in the indigenous and exotic woodlands of Rhodesia. **Forestry 45:** 67–86.

VAN DER PLANK, J. E. 1963: "Plant Diseases. Epidemics and Control". Academic Press, London.

VITHAYASAI, C. 1971: Runs of healthy and diseased trees in transects through an infected forest; statistical and computational methods. **Biometrics 27:** 1003–16.