REDUCED EARLY GROWTH RATES OF PINUS RADIATA CAUSED BY DOTHISTROMA PINI

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ABSTRACT

Various infection levels of **Dothistroma pini** Hulbary were induced by artificial inoculation of **Pinus radiata** D. Don trees 1 year after establishment in 1972 in a plantation in Kaingaroa State Forest, and disease levels were recorded quarterly for 8 years. Tree growth between the second and eighth years of the study was examined by stem analysis of a sample of 120 trees from the second thinning and by sectional measurement of the remaining final-crop trees for volume.

Inoculated plots exhibited higher disease levels from 1973 to 1976 and lower annual volume increments from 1975 to 1979 than plots which were sprayed with fungicide. Disease/volume-loss relationships were analysed by multiple regression, and it was shown that volume growth was reduced in proportion to increase in disease level.

INTRODUCTION

Growth of *Pinus radiata* can be affected by severe and protracted defoliation caused by *Dothistroma pini* (Gibson 1974), but study of the relationship between amount of growth loss and level of disease is complicated by a great many factors. The incidence of the epidemic and its effects are probabilistic in nature, with sizable random variations. Differences in site may act as an independent variable to affect growth even in seemingly uniform stands. Because tree growth is associated closely with crown development and competition with neighbouring trees (Assmann 1970), destruction of foliage may affect normal growth patterns in *P. radiata* (Rook & Whyte 1976). However, most stands are never completely free of defoliating fungi, and destruction of moderate amounts of foliage by *D. pini* may be tolerated with little or no detectable effect on growth (Christensen & Gibson 1964; Whyte 1969; Gibson 1974).

Volume loss due to defoliation by *D. pini* can, in principle, be estimated by subtracting the volume produced from the volume expected to be produced during the period in which defoliation occurred. In order to do this, accurate assessments of defoliation levels and delineation of the periods during which they occur are essential. In the North Island *D. pini* usually develops early in the life of the stand and progresses

until the trees develop resistance at the age of about 15 years (Bassett 1972). During the susceptible period annual disease levels may fluctuate grossly and are governed mainly by climate and inoculum potential and, to a lesser extent, by control with fungicides.

In appraisal of growth loss it is necessary first to collect data from field experiments with different levels of disease and to note corresponding differences in growth. Subsequently growth loss models can be developed to describe the relationships between disease and loss of yield.

This study was set up to establish the relationship between growth loss and defoliation levels of young *P. radiata* and to investigate the effect on disease progress of spraying with fungicide. The study was carried out over six consecutive growing seasons starting at tree age 2 years. The results may serve as provisional models for tree stands prior to crown closure.

METHODS

Treatments

The *D. pini* spray experiment was established by J. W. Gilmour and F. H. Crockett on a uniform and flat area of 23.1 ha in Cpt 1251 of Kaingaroa State Forest. The experimental area was planted with 1/0 *P. radiata* seedlings from FRI nursery in July 1972 at a nominal spacing of 4 × 2 m (1250 stems/ha). The compartment was thinned to 625 stems/ha in January 1978 before the canopy had closed and all remaining trees were pruned to 2.0–2.5 m. A second thinning took place in April 1980 to 300 final-crop trees/ha and these were pruned to 4.2 m.

The experiment was laid out in two replicate blocks of approximately 10 and 13 ha which were divided into two sub-blocks, each containing six randomly located plots. Each plot measured 20 × 20 m (0.04 ha) and contained 50 trees at time of planting, 25 trees after the first thinning, and 12 final-crop trees in 1980. In the Sub-block 1 areas two randomly chosen plots were handsprayed in December 1973, 1974, and 1975 with copper oxychloride (4 g/*l* water) to runoff point, to ensure low levels of *D. pini* infection. In December 1972 and March 1973 infected branches were placed against 18 trees dispersed evenly within each of the other four plots. The infected branches were left for 3 months to build up inoculum and to form infection centres within the plots. In the Sub-block 2 areas, four randomly chosen plots were handsprayed and the other two plots were inoculated in the same way as Sub-block 1.

In December 1976 the spray treatment was changed. The Sub-block 2 areas were aerially sprayed with 4.16 kg copper oxychloride in 50 *l* water/ha/year, flown twice. Henceforward the four inoculated plots in the Sub-block 2 areas were sprayed aerially each year but the eight handsprayed plots in the Sub-block 1 areas received no further spray treatment.

Experimental Data

Each plot tree was measured and tagged at time of planting. Disease assessment and height and diameter measurement were started in July 1973 and continued at about 3-month intervals. Stem diameter was measured initially at 15 cm above ground level, and at breast height from July 1976. Disease levels were visually scored for each tree

(after Gilmour & Noorderhaven 1973). The percentage of the normal green crown that was defoliated or severely infected by *D. Pini* was estimated in 5% steps by three trained observers whose individual scores were averaged.

In April 1980, 13 trees per plot were selected for the last thinning to waste. The annual disease levels of each tree were calculated and averaged over the period from 1973 to 1980. The trees were ranked according to increasing disease levels (Gilmour & Noorderhaven 1973):

1 = trace to 5%

2 = 6% to 25%

3 = 26% to 50%.

In each rank a random sample of 40 trees was chosen representing the full range of tree sizes, i.e., 10 large, 20 medium, and 10 small trees. Trees with obvious damage of any kind were rejected and replaced. In this way 120 trees were selected for complete stem analysis of past growth. After they were felled, sections were cut at mid-internodes and diameters were measured within each annual shoot (Whyte 1974). Cumulative volumes and annual volume increments were calculated using program package GRANRAD (unpubl. data, available from author).

Final-crop trees were sectionally measured for volume by taping diameters at 0.7 and 1.4 m and at the mid-internodes immediately above these points (Whyte 1971). Volumes were calculated using the truncated cone formula. The average disease levels for each of the final-crop trees were calculated in the same way as for the thinned trees.

Plot volumes were calculated for each year from 1973 to 1980, using annual volume lines constructed from the stem analysis data. The tree volume equations were calculated using the standard model

$$V = b\ D^2H$$

where V = volume

D = diameter at breast height

H = total height

and b = constant,

assuming linearity (Husch *et al.* 1972). The volume lines of 1973 and 1974 were based on the diameter at 15 cm above ground level.

RESULTS AND DISCUSSION

Treatments

The average annual disease levels of the inoculated plots were significantly higher than those of the sprayed plots in 1973–76 (Fig. 1), but there was no difference between treatments in 1977–80. Aerial application of the fungicide commenced in 1976 when infection levels of the sprayed and inoculated plots were 5% and 30% respectively. Dry weather caused disease levels of the inoculated plots to drop to the same low levels as the sprayed plots in the years that followed. Wide guard-strips separated the plots and interplot interference was unlikely to cause cryptic error

(van der Plank 1963) since conidia are dispersed only a short distance by a splash mechanism during wet periods (Gibson *et al.* 1964). Generally it may be preferable to conduct multiple treatment experiments with natural epidemics (James 1978) but since *D. pini* epidemics are strongly dependent on factors such as the weather, artificial inoculation was necessary. This procedure also facilitated sampling since disease levels were uniform within the plots.

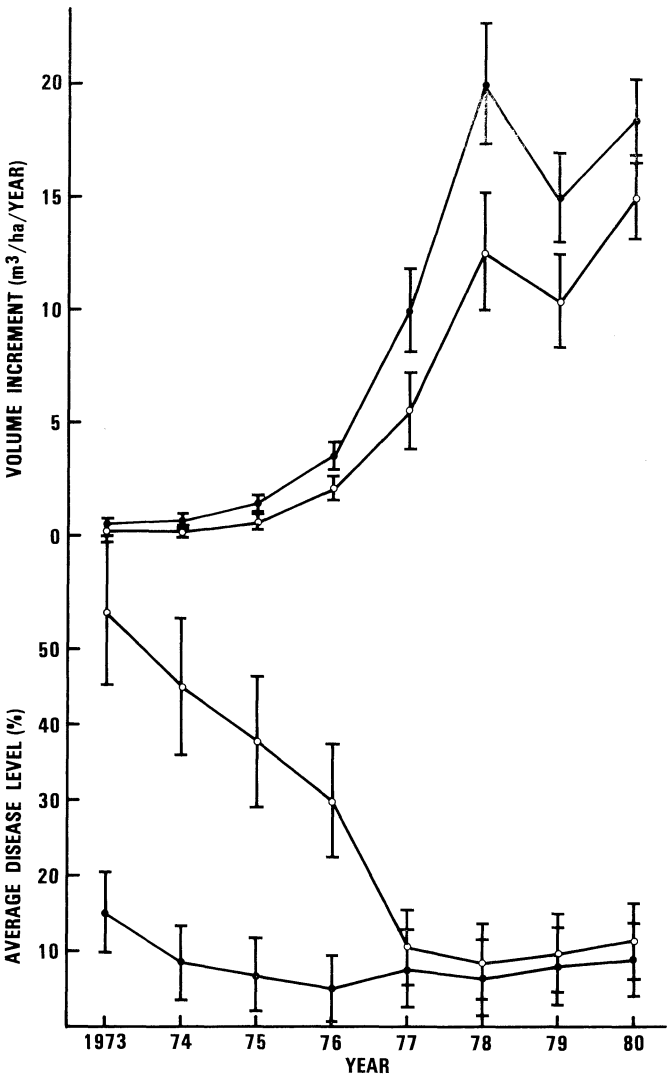

FIG. 1—Annual disease levels and volume increments of inoculated and sprayed plots from 1973 to 1980 (o = inoculated plots, ● = sprayed plots). Vertical bars show the 95% confidence limits

Average annual volume increments of the sprayed plots were significantly higher in 1975–79 than those of the inoculated plots in the same period. (The apparent drop in volume increments in 1979 was caused by the thinning in 1978.) It was clear that growth response lagged behind disease progress (Fig. 1). This may be explained by the additive effect of reduced photosynthetic capacity caused by gradual defoliation during subsequent growing seasons, and a decrease of photosynthates translocated to the roots. The latter can cause a delayed inhibition of volume increment since roots are very good sinks in young *P. radiata.* Thus *D. pini* may have a direct effect on photosynthesis but also a secondary effect similar to that of a root pathogen, causing growth reduction that lags behind the occurrence of defoliation.

Thinning

Between thinned and final-crop trees there were no significant differences in frequencies of trees with light, medium, and high average disease levels (Table 1), nor in height. The volumes and diameters at breast height of final-crop trees were significantly greater than those of the thinned trees. This implies that thinning was non-selective for height and disease level, but that trees with smaller diameters at breast height and with less volume were thinned out. Thus thinning favoured higher volumes with more taper, i.e., trees that were less affected by *D. pini.* This suggests strongly an increase of disease tolerance (Schafer 1971) in final-crop trees which may be heritable.

TABLE 1—Effect of thinning on disease levels and tree variables

Variable	Thinned trees	Final-crop trees
Disease level 0–5%		
Frequency (%)	12	15
Height (m)	10.8	11.3
Diameter (cm)	14.5	16.8
Volume (m^3)	0.11	0.14
Disease level 6–25%		
Frequency (%)	34	33
Height (m)	10.0	10.6
Diameter (cm)	13.4	15.2
Volume (m^3)	0.09	0.12
Disease level 26–50%		
Frequency (%)	54	52
Height (m)	9.6	10.1
Diameter (cm)	11.0	13.2
Volume (m^3)	0.08	0.10

Horizontal lines indicate treatments not different at the 5% significance level

Disease/Growth Relationships

A stepwise regression analysis of the annual volume increments on annual disease levels was performed for the results of the stem analysis. Average disease levels of previous years were successively added to the regression equations in order of decreasing significance until the added variable failed to make a significant contribution at the 5% level. Orthogonalisation and transformation of the data gave no satisfactory distributional improvements. The best-fit models are given by adding two variables, i.e., the average annual disease levels of the first and second years prior to the year of the volume increment (Table 2). Since disease levels were not assessed in 1978, the average disease levels in 1979 and 1977 and in 1977 and 1976 respectively have been taken for the volume increments of 1980 and 1979. The second disease variable removed virtually no variation of the volume increments except for 1977 and 1978. The current annual disease level was also a highly significant variable but removed less variation than the average disease levels of the penultimate year. Successive average annual disease levels were highly correlated during the years from 1974 to 1977; this was exhibited by the partial F values before addition into the regression equations (not given) and by the correlation coefficients (Table 3).

TABLE 2—Summary of regression analysis of volume increment on initial size and disease levels

Year	b_0	b_1	b_2	b_3	R^2
1980	8.9304	0.5629***	—0.2932**	—0.0374	0.851
1979	7.1091	0.7438***	—0.2211**	—0.0365	0.860
1978	4.3439	1.0744***	—0.1056**	—0.0119*	0.864
1977	2.0639	1.6919***	—0.0302**	—0.0241**	0.942
1976	0.7306	2.4604***	—0.0209**	—0.0029	0.891
1975	0.3397	3.8433***	—0.0039**	—0.0005	0.795
1974	0.0030	0.9548***	—0.0002*	—0.0001	0.697

Regression coefficients computed from $Y = b_0 + b_1X_1 + b_2X_2 + \ldots . b_tX_t$
where Y = annual volume increment,
X_1 = the corresponding size of the tree of the previous year,
X_2–X_t = the average disease level of the current or previous years respectively.

* = significant at the 5% level
** = significant at the 1% level
*** = significant at the 0.1% level

TABLE 3—Correlation coefficients between annual disease levels from 1973 to 1979

Year	1974	1975	1976	1977	1978	1979
1973	0.8397	0.8038	0.7184	0.2435		0.0001
1974		0.8842	0.8501	0.3461		0.0004
1975			0.9266	0.4194		0.0033
1976				0.5539		0.0973
1977						0.2394

r = 0.6063 significant at the 1% level
r = 0.4824 significant at the 5% level

Regression analysis is a useful technique for translating disease damage into growth loss (Butt & Royle 1974) and multiple-point models (James 1978) were employed by measuring disease levels at regular intervals during the epidemic. This seems appropriate since the epidemic develops over a relatively long period with variable infection rates, and growth response is a prolonged process. The high correlation between successive annual disease levels in 1973–76 was caused by a high carry-over of inoculum from one year to the next and also by the fact that the infected needles of one year were contributing to the defoliation percentage of the next. This is less true after pruning when part of the crown with some inoculum is removed. The regression equations of annual volume increments on initial size and annual disease levels (Table 2) for that reason have no predictive value. It was demonstrated that disease levels were time-related and interdependent, and that the effect of disease level on growth was greatest 1 year after occurrence. The growth response relationships exhibit inverse correlations whereby the earlier discussed root-sink effect may play an important role. Another point to consider is the manner in which infection of needles progresses throughout the growing season, gradually inhibiting the photosynthetic capacity. This can affect current and later growth (*see* Rook & Whyte 1976), since needles are a strong sink and they are important for storage of reserves particularly when foliage volume is considerable in relation to structural components (Glerum 1980).

Final-crop Trees

Stem form

Form curves were constructed for trees with high (41%) and low (2%) average disease levels (1973–80), using a sample of 30 trees for each group. Profiles of the ratios of diameters measured at 10% steps of the total height of the tree, to diameter at 20% of the total height were constructed and the two groups were compared (Table 4). Real changes in the form of the tree caused by *D. pini* consisted mainly of a decrease at the base of the stem since only the two lowest relative diameters of the series were significantly different. This agrees with findings by Rook & Whyte (1976) who reported that artificial defoliation treatments produced less stem taper, with diameter growth being inhibited more at the base of the tree than at the top.

Compensation of growth (Zadoks & Schein 1979) of the selected trees was thought to be negligible since disease incidence was uniform within plots and growth measurements were taken before crown closure. It may be argued that the disease may affect growth processes not directly attributable to loss of leaf area. Predisposition (Yarwood 1959) of the host to pathogen development may confuse growth response for two reasons – because of variability of early growth, and because of differences in the onset of adult resistance with age, a point on which there is at present little documented evidence.

Loss

The volume, diameter, and height of the final-crop trees in 1980 were related to the average disease levels (1973–80). The best-fit models were linear and scattergrams are given of the percentage loss of increment in height, diameter, and volume as related to average disease level (Fig. 2). The trends are linear and the lines were conditioned to pass through the origin. The original regression equations were used

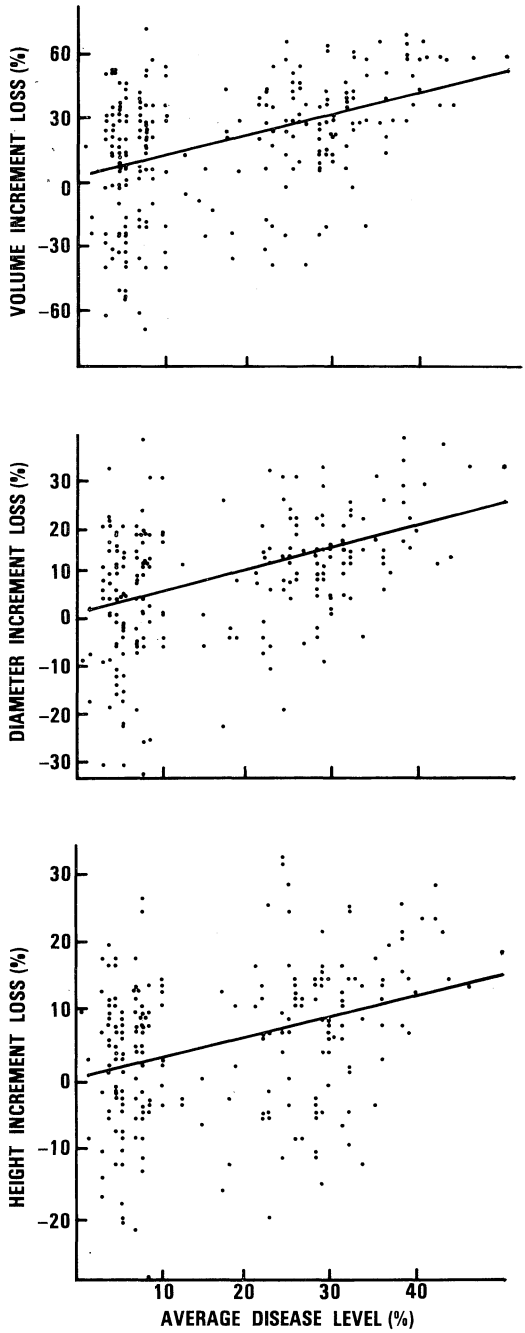

FIG. 2—Percentage increment loss of final-crop trees as related to average disease level. Regression lines are of the form $Y = b_1X$ where Y = increment loss (%), b_1 = -100 b/a (see Table 5), and X = average disease level (%), for height $Y = 0.2916\ (\pm 0.1422)\ X$,
diameter $Y = 0.4707\ (\pm 0.3210)\ X$,
volume $Y = 0.9679\ (\pm 0.2831)\ X$

to estimate the confidence intervals for percentage loss for a given average disease level following the method of Kendall & Stuart (1961). Percentage loss of volume, diameter, and height were calculated for 10% intervals of increasing disease levels up to 50% (Table 5). Volume loss is proportional to average disease level, but height loss is less severe than diameter loss (Fig. 2). It may be reasonable to expect non-linearity at higher disease levels and after crown closure.

The disease loss estimates showed standard errors of the order of 30%. High bias was possibly caused by defective disease measurement procedures. What is to be measured in the field may be defined as the percentage of the foliage normally green at the time of the assessment that has been rendered inoperative for photosynthesis, or is no longer green by reason of the disease (Large 1955, 1966). A major difficulty in assessing plantation trees is the variation in the level of the lower green foliage which is affected by age, spacing, and pruning. In order to determine the loss of green foliage

TABLE 4—Profile series of trees with high and low disease levels

Height as percentage of total height	Mean relative diameter (%)[1]		t-value
	Average disease level 41%	Average disease level 2%	
0	123	135	4.17**
10	109	113	3.39**
20	100	100	
30	87	86	0.31
40	74	75	0.03
50	63	62	0.01
60	53	53	0.01
70	42	41	0.24
80	30	28	0.02
90	15	15	0.01

[1] Relative diameters are given as percentages of the diameter at 20% of the total height
** Significant at the 1% level

TABLE 5—Percentage loss of the final-crop trees in 1980 for increasing disease levels

Average disease level 1973–80 (%)	Loss (%)[1]		
	Volume	Diameter	Height
10	9.7 (2.8)	4.7 (3.2)	2.9 (1.4)
20	19.4 (5.6)	9.4 (6.4)	5.8 (2.8)
30	29.1 (8.4)	14.1 (9.6)	8.7 (4.2)
40	38.8 (11.2)	18.8 (12.8)	11.6 (5.6)
50	48.5 (14.0)	23.5 (16.0)	14.5 (7.0)

Estimates of variances were obtained of the ratio b/a in:
loss (%) = −100 (b/a) DL, where DL = average disease level (1973–80) and var(b/a) nearly equal to $\text{var}(b)/a^2 + b^2\text{var}(a)/a^2 + 2b\text{cov}(a,b)/a^3$. The variances and covariances of a and b were found from the original regressions.

[1] Confidence limits at the 5% level are given between parentheses

the normal amount should be known and this can only be assessed with any degree of accuracy in stands before crown closure. This may be the reason why very high residual variation was found by regressing increments to disease levels in a study of unthinned stands by Whyte (1976).

The assessment method employed in this experiment may be questionable on physiological grounds as *D. pini* epidemics progress through various stages of crown development. Since the diseased part of the crown has a proportional rather than an absolute value, the visual assessment method can be useful only for empirical studies. For an understanding of causal relationships, knowledge of the disease/physiology complex is desirable. It may be, for instance, that any percentage of green foliage of the diseased tree is not necessarily equivalent to the same percentage of green foliage of a healthy tree in terms of photosynthetic capacity. Photosynthesis can be regulated by the utilisation of its products, e.g., if the demand is increased by some means, the photosynthesis of the remaining healthy needles will also be increased. Conversely, if sink demands decrease, e.g., when crown closure takes place, there may be a feedback inhibition. Site quality may interact with these processes since the supply of nutrients affects photosynthetic activities (Assmann 1970). Therefore accurate quantification of infected needle area and nutritional status of the tree are needed in fundamental models that explain disease/loss relationships.

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