CYCLANEUSMA (NAEMACYCLUS) NEEDLE-CAST OF PINUS RADIATA IN NEW ZEALAND

2: REDUCTION IN GROWTH OF THE HOST, AND ITS ECONOMIC IMPLICATION

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ABSTRACT

Twenty pairs of 9-year-old final-crop trees were selected in a stand of **Pinus radiata** D. Don heavily infected with **Cyclaneusma minus** (Butin) **DiCosmo et al.** at Kaingaroa State Forest in 1977. Each pair consisted of one heavily diseased tree and one healthy tree. Disease severity, in terms of percentage of crown infected, was recorded annually. Relationships between disease severity and growth were studied by complete stem analysis at tree age 15 years.

Annual volume increments of the diseased trees were significantly reduced from age 7 onwards. Reduced diameter growth in the lower parts of the stem of the diseased trees caused small changes in the relative diameter distribution. The relationship between disease severity and volume loss showed a reduction of the average volume increment of approximately 60% at an average disease severity of 80%. Stand growth was projected to age 30 for various proportions of diseased trees. For each 10% increase in the proportion of diseased trees a reduction in total volume of 10–14 m^3/ha and a subsequent reduction in revenue at clearfelling of $600–$700/ha may be expected under the given stand conditions.

Nine pairs of trees were injected with a solution of carbendazim fungicide in 1980 and 1981 in an attempt to control the disease. Injection with the fungicide gave a significant growth response associated with a trend to reduced disease severity. Some of the growth response may have been attributable to a nutritional effect as it occurred in healthy and diseased trees.

INTRODUCTION

Needle-cast of *Pinus radiata* associated with *Cyclaneusma minus* has been observed in many forests for many years (Gadgil *et al.* 1977). The severity of the needle-cast has varied from year to year and periodic serious outbreaks are causing concern to forest management. It has been reported elsewhere that *Cyclaneusma minus* can cause reduced growth in young *Pinus sylvestris* L. and destroy the trees' utility as Christmas trees (Kistler & Merrill 1978). Little is known, however, about the effect of this fungus on the growth of *P. radiata* or its economic impact.

A trial was therefore set up to establish the relationship between growth loss and disease severity in a stand of *P. radiata* infected by *C. minus* in Kaingaroa State Forest.

METHODS

Site

An area of approximately 0.5 ha was selected in the centre of a stand (65 ha) of 9-year-old *P. radiata* in Kaingaroa State Forest. The stand was on a level site that was aerially seeded in 1968. The initial stocking of 4200 stems/ha had been thinned in 1971, 1974, and 1977 to respectively 2604 stems/ha, 1144 stems/ha, and 400 final-crop stems/ha. Trees in this part of the stand had not shown symptoms of Dothistroma needle blight, and thus had not been sprayed with fungicide at any time.

The study was carried out over 6 consecutive years starting in 1977 when the trees were 9 years old. Field observations indicated that approximately 50% of the trees in the stand were diseased, although the severity of infection varied. The affected trees were scattered among others that showed no visible symptoms of ill health and had normal green crowns.

In 1977, after the last thinning, 40 of the final-crop trees were selected in pairs. Each pair consisted of a healthy and a heavily diseased tree of approximately equal d.b.h., crown height, and crown width at time of selection. Paired trees, representing the full range of tree sizes, were randomly selected. The healthy trees were free from any visible infection whereas the diseased trees showed premature needle-cast of more than 65% of the crown.

Volume loss due to defoliation caused by the fungus was measured at age 15 by comparing annual growth of the healthy and diseased trees.

Treatments

In March 1980 nine randomly selected pairs of healthy and diseased trees were injected with a carbendazim solution. This fungicide has been shown to control Cyclaneusma needle-cast (Hood & Vanner 1984). The solution consisted of 3.5 g methyl benzimidazol-2-yl carbamate dissolved in 8.75 ml concentrated hydrochloric acid made up to 1 litre with water. The dosages applied were 4 litres for trees with a diameter at breast height of less than 18 cm, 5 litres for those from 18 to 25 cm d.b.h., and 6 litres for those larger than 25 cm d.b.h. The solution was gravity fed through five equidistant holes (1.2 cm diameter, 3 cm deep) drilled through the bark 30 cm above ground level. The uptake of the solution took place within 5 days for all trees. A second dosage was applied to the same trees in November 1981. Dosages were increased and ranged from 4 to 17 litres/tree depending on the size of the tree. The uptake this time was slow and some trees took much less than the applied dosage.

Disease Scoring

Disease severity was visually estimated for each tree using a method similar to that used for the assessment of Dothistroma infection (Kershaw *et al.* 1982). The percentage of the normal green crown that showed visible symptoms of infection by *C. minus* was estimated in 5% steps by three trained observers whose individual scores were averaged. Scoring was carried out weekly over a 6-week period in September and October each year, the period when maximum symptom expression could be identified for each individual tree. Foliage samples were taken from each tree and isolations made of the

fungi present inside the needle tissues in June and November 1977 and 1978, May and July 1979, and September 1980 using methods outlined by Gadgil (1984).

Analyses

In July 1983 all sample trees were felled for a complete stem analysis of past growth. After felling, sections were cut at mid-internodes and diameters were measured within each annual shoot (Whyte 1974). Cumulative volumes and annual volume increments were calculated, and growth increments were plotted using program package GRANRAD (unpubl. data, available on request). Growth increments were tested by analysis of covariance with height and volume of previous years as covariates. Volume growth was calculated for healthy and diseased stands. In the latter the proportion of diseased trees in the final-crop component increased from 10% to 60% in 10% steps. Stand basal areas were extrapolated from the basal area of healthy and diseased trees measured at age 15, and basal areas of diseased stands were reduced proportionally with increased percentage of diseased trees. Basal areas were implemented in the Kaingaroa Growth Model (Elliott & Goulding 1976) for growth projection to the end of the rotation at age 30, and predictions were carried out for stockings of 400 stems/ha, 300 stems/ha, and 200 stems/ha. Program PROD (Goulding & Shirley 1978) forecast tree size distribution and program SILMOD (McGregor & Williams 1982) predicted changes in the net value of the simulated stands.

RESULTS AND DISCUSSION

The average annual disease severity of the diseased trees was significantly higher than that of the healthy trees ($p < 0.01$) from 1977 to 1982 (Table 1). Some of the initially healthy trees became infected in 1978 but the disease severity remained low.

TABLE 1—Average annual disease severity of healthy and diseased trees (percentage of green crown infected)

Category	Year						Average 1977–82
	1977	1978	1979	1980	1981	1982	
(a) Healthy	t	20	12	21	20	23	16
(b) Healthy (injected in 1980 & 1981)*	t	t	t	13	t	t	2
Average	t	10	6	17	10	11	9
(c) Diseased	74	72	64	70	67	58	67
(d) Diseased (injected in 1980 & 1981)*	77	64	48	47	37	29	50
Average	75	68	56	58	52	44	59

* Annual reduction of disease severity after injection with the fungicide was tested by analysis of covariance. Treatment differences were not significant ($p < 0.05$).
t = trace

Injection with carbendazim reduced the disease severity of both the diseased trees and healthy trees with light infection. This agrees with Hood & Vanner (1984) who found that stem injections of carbendazim gave significant control of Cyclaneusma needle-cast in a single susceptible clone of *P. radiata.* In this trial, however, improvement in crown health after carbendazim injection was not as marked and contrasts between injected and control trees could not be clearly distinguished statistically. The reason may lie in chance irregularities caused by the small sample size and imprecision of disease assessments. The reduced level of control may have been due also to the small uptake of solution by some trees during the second application. An effort was made to compensate for variation in tree size by adjusting the volume of fungicide solution injected.

The disease severity varied little from year to year in most trees and the annual severity levels were correlated ($r = 0.65$ to 0.91 between severity levels up to 4 years apart). This suggests that susceptibility to the disease remained relatively constant during the period of investigation. Isolations made from the foliage samples showed that the populations of *C. minus* were significantly higher in the needles of the diseased trees than in those of healthy trees (Table 2). Injection with carbendazim also reduced population levels. There was no difference between the populations of *Lophodermium* spp. which usually follow *C. minus* by about 1 month in the needle colonisation sequence (Gadgil 1984).

TABLE 2—Number of isolates of **Cyclaneusma minus** and **Lophodermium** spp. per 10 cm of needle length

Date	**Cyclaneusma minus**		**Lophodermium** spp.	
	Healthy trees	Diseased trees*	Healthy trees	Diseased trees
1977 June	2.7	3.2	1.3	2.5
November	0.5	3.0	2.7	4.5
1978 June	2.0	3.2	0.5	1.7
November	1.8	7.0	13.0	27.0
1979 May	0.3	1.3	1.3	6.5
November	1.0	3.5	4.3	4.5
1980 September	12.2 (8.3)†	25.2 (10.6)	0.5 (7.5)	9.4 (3.2)

* Numbers of isolates of **Cyclaneusma minus** from the diseased trees are significantly higher than those from healthy trees ($p < 0.05$).

† Numbers of isolates from injected trees in parentheses.

Growth Loss

Height growth was not significantly reduced by the disease (Table 3) and the average total height of the diseased trees (20.4 m) was only slightly less than that of the healthy trees (21.7 m) at age 15. This agrees with other findings that height is a

TABLE 3—Average annual volume and height increments (adjusted values) of healthy and diseased trees from 1969 to 1983

Year	Healthy trees				Diseased trees			
	Height increment* (m)	Volume increment (dm^3)†			Height increment* (m)	Volume increment (dm^3)†		
		Control	Injected	Pooled		Control	Injected	Pooled
1969	0.5	0.06	0.04	0.05	0.5	0.10	0.06	0.08
1970	1.0	0.33	0.30	0.32	1.0	0.53	0.33	0.44
1971	1.1	1.6	1.4	1.5	1.2	1.8	2.2	2.2
1972	1.3	5.5	5.4	5.4	1.5	5.1	4.8	5.0
1973	1.7	9.7	9.4	9.6	1.7	9.5	8.8	9.2
1974	1.7	18.3	16.4	17.4	1.6	15.7	15.6	15.7
1975	1.7	31	29	30	1.7	22	23	23
1976	1.7	28	29	28	1.5	24	33	28
1977	1.7	40	37	38	1.6	27	30	28
1978	1.7	58	60	59	1.5	30	37	33
1979	1.6	70	79	74	1.6	51	59	55
1980	1.6	93	103	97	1.5	63	68	65
1981	1.7	109	126	117	1.4	77	96	85
1982	1.3	106	123	113	1.3	70	93	80
1983	1.6	108	126	115	1.4	88	107	97

* Height increments not significantly different between healthy and diseased trees.
† Volume increments of injected trees significantly higher than of control trees ($p < 0.01$).
Volume increments of healthy trees significantly higher than of diseased trees ($p < 0.01$).

slow index of loss of productivity due to partial defoliation (Rook & Whyte 1976) or to defoliation by leaf pathogens such as *Dothistroma pini* Hulbary (Whyte 1976; van der Pas 1981).

Annual volume growth of the trees selected as diseased for this study had been slightly faster than that of the healthy trees during the first years after planting from 1969 to 1974 (Fig. 1). Volume growth of both categories was approximately similar in 1976 and 1977. From 1978 onwards annual volume increments of the diseased trees were increasingly reduced and this is apparent mostly in the sixth to twelfth internodes. Analysis of covariance indicated that growth differences between healthy and diseased trees were not significant from 1961 to 1974 (Table 3). Growth reduction of the diseased trees was significant from 1975 onwards. This may suggest that severe needle-cast started as early as age 7, which agrees with field observations about the onset of the disease (van der Pas *et al.* 1984). Volume reduction of both healthy and diseased trees in 1976 in the bottom internodes (eighth growth sequence of Fig. 1A and 1B) is attributable to medium pruning in the previous year. Reduced growth in both categories during the last two growing seasons (sequences 14 and 15) is probably caused by increasing competition.

Injection with the fungicide solution resulted in significantly faster growth in both healthy and diseased trees than in the control ($p < 0.01$) from 1981 to 1983 (Table 3). The growth sequences of these years were plotted for both injected and control trees (Fig. 2). Extra volume growth of the injected healthy trees was apparent in most of the bole except in the upper two internodes. Growth recovery of the injected diseased trees occurred in each internode from apex to base. The increment recovery in the top of the bole could indicate some increased needle retention in the upper crown. The response in the healthy trees suggests that carbendazim may supplement the nitrogen requirements of the trees. The active ingredient consists of 22% by weight of this element (Hood & van der Pas 1979) and it is estimated that the injected trees received a total of between 9 and 12 g nitrogen each (average 10 g; approximately equivalent to 2 kg N/ha/year, over 2 years). Although this is not a great quantity when compared to fertiliser applications, it is still a contribution that may account for some of the growth response.

The growth sequences of the diseased trees suggest that growth reduction due to Cyclaneusma may cause changes in the stem form. This was supported by relative diameter series of the 11 trees in each group that were not injected. The diseased trees show diameter growth in the bottom part of the stem reduced by about 3% relative to healthy trees. This is in agreement with earlier findings of the effect on stem form of (a) defoliation caused by *D. pini* (Whyte 1976; van der Pas 1981) and (b) partial defoliation (Rook & Whyte 1976). It seems reasonable to expect, however, that a nominal effect on stem taper will not seriously restrict the use of the Growth Model for increment predictions.

Disease/Loss Relationships

A stepwise regression analysis of the annual volume increments on initial tree size and annual disease severity of current and previous years was performed for each year

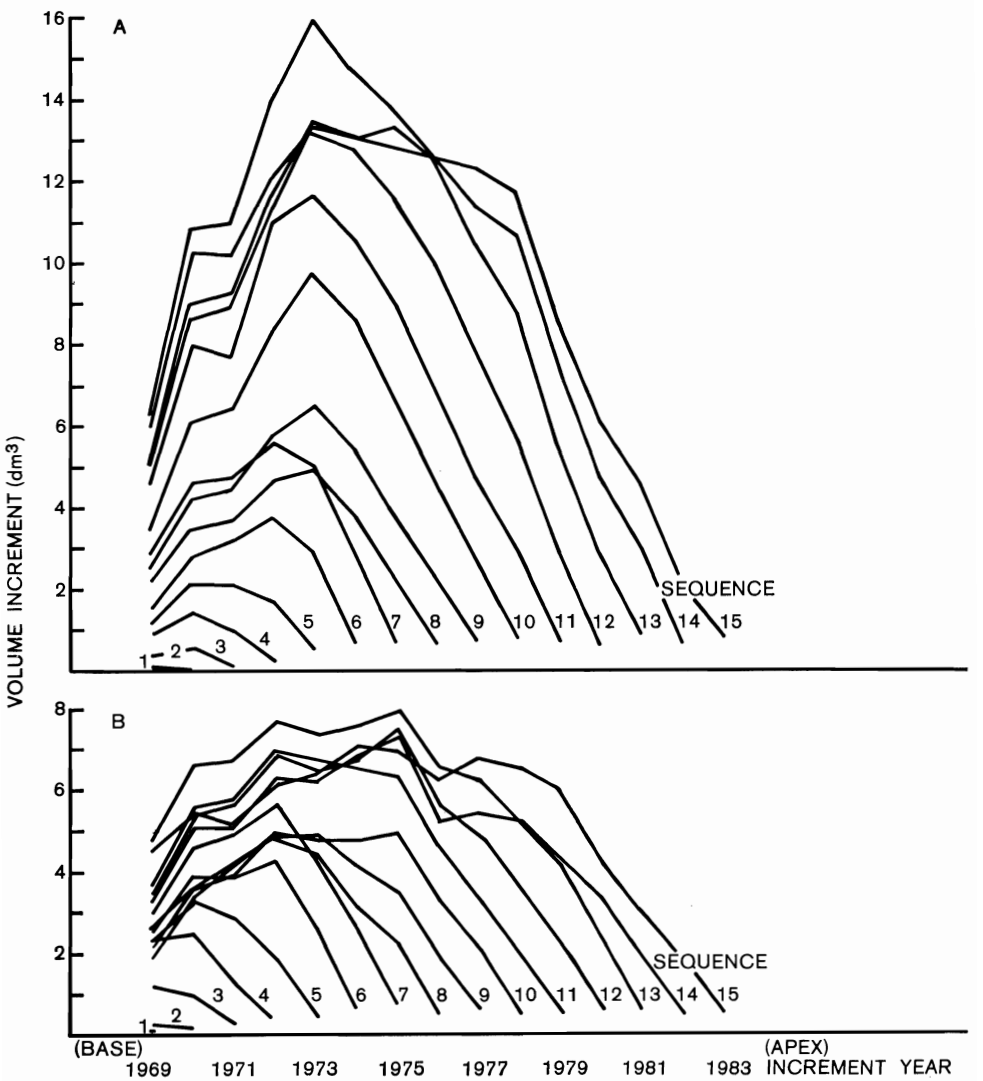

FIG. 1—Family of sequences of the average volume increments of the pooled healthy trees (A) and diseased trees (B). Each sequence shows the volume increment at each successive internode (apex to base) for 1 year's growth, e.g., 15 shows the sequence for the latest year's growth and 1 for the earliest year's growth.

from 1977 to 1983 (Table 4). The average disease severity of previous years was successively added to the regression equation until the added variable failed to make a significant contribution at a 5% level. The best-fit models are given by adding only the current annual disease severity to the volume of the penultimate year. The disease severity of the previous years removed no significant variation when added to the

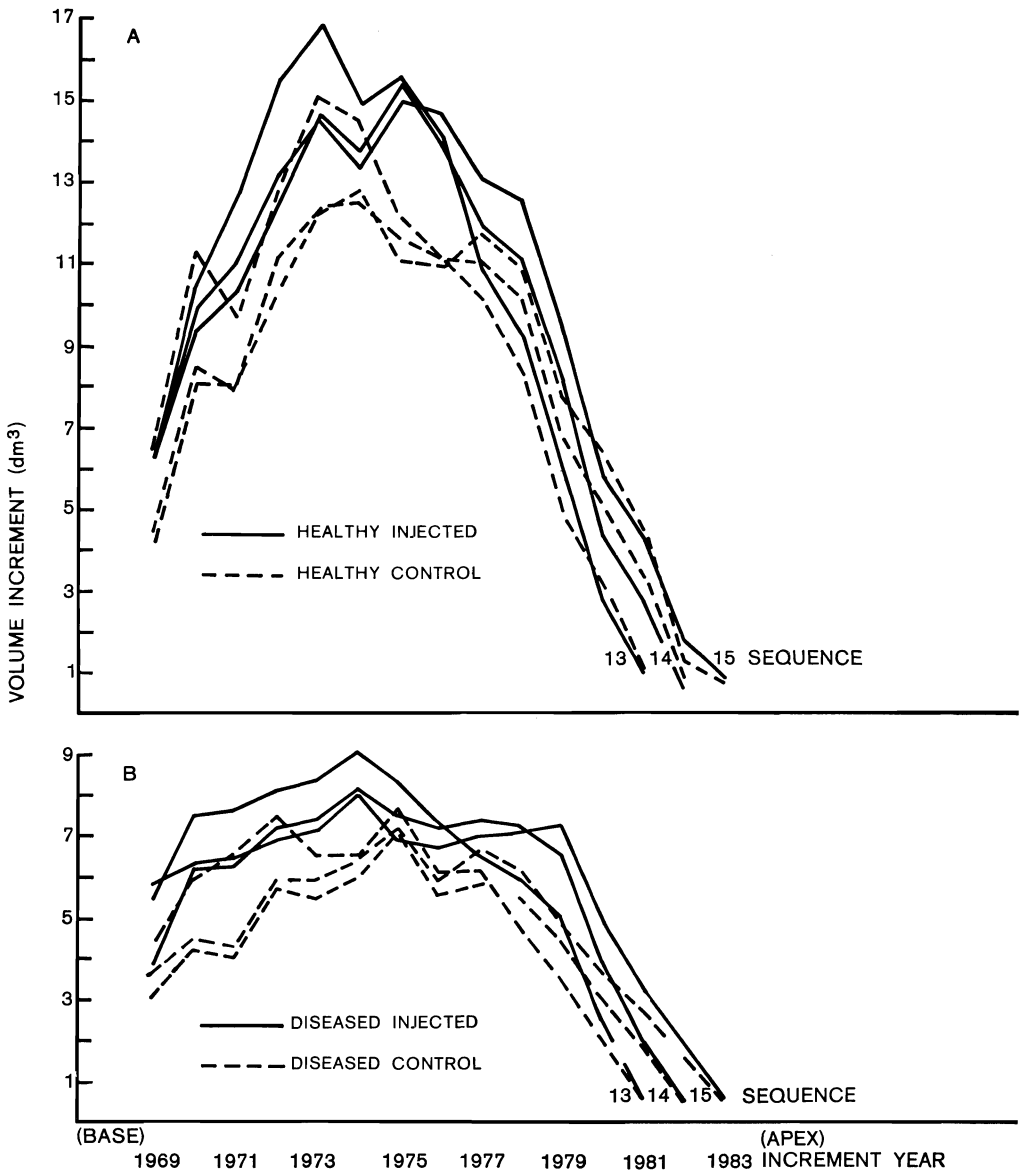

FIG. 2—Family of sequences of the average volume increments of injected and control trees - (A) healthy and (B) diseased. Sequences of the successive volume increments are given for the period from 1981 to 1983.

equation. This may not be surprising because the peak period of needle-cast occurs in spring 3–6 months after the infection of the current needles which are, at that time, about 8–9 months old (Gadgil 1984), and so may directly affect the current year's wood production. The effect of previous years' defoliation, however, may be concealed since

TABLE 4—Summary of regression analysis of annual volume increment on initial size and disease severity

Year	b_0	b_1	b_2	r^2
1978	10.0	0.274***	-0.232***	0.93
1979	28.2	0.289***	-0.279***	0.81
1980	35.0	0.270***	-0.525***	0.84
1981	41.9	0.263***	-0.585***	0.88
1982	48.3	0.164***	-0.572***	0.83
1983	37.1	0.168***	-0.570***	0.77

Regression coefficients computed from $Y = b_0 + b_1X_1 + b_2X_2$.

Where Y = annual volume increment
X_1 = the corresponding tree volume of the penultimate year
X_2 = the current annual disease severity

*** Significant ($p < 0.001$).

annual disease severity levels were highly correlated and growth response may extend over more than 1 year. Therefore, the average disease severity was taken for the 6-year period from 1978 to 1983 and related to the volume increments of that period. This gave a plausible linear relationship (Fig. 3). The scattergram exhibits large variation which is attributable mainly to inaccurate disease assessments and competition effects, but it shows that growth during the 6-year period was severely reduced by the disease. Volume increment reduction at an average disease level of 80% was approximately 60% of that of the increment of healthy trees. This is in agreement with findings by Rook & Whyte (1976) who reported a reduction of 50% in volume increment after removing 1- and 2-year-old needles. The slightly higher growth reduction in this experiment may be attributed to the compounded effect of defoliation for 6 consecutive years.

Economic Impact

There are no data on the final yield of a diseased stand which can be used for economic evaluation of growth losses due to the disease. The growth data of this trial were therefore projected to the end of the rotation at age 30 using the Kaingaroa Growth Model. Volume reductions were predicted for stands with increasing proportions of diseased trees in 10% steps to 60%, assuming no further impact by the disease after age 15 (Table 5). Future disease progress and subsequent impact are impossible to predict and the assumption has to be accepted to accommodate further analysis. It seems reasonable to expect continuation of infection after age 15 at least for some years. Infected stands can show severe disease symptoms at ages up to 25 years (van der Pas *et al.* 1984). On these grounds, growth reduction in diseased stands at the end of the rotation may be under-estimated. Conversely, growth of healthy stands and of stands with less than 50% of the trees infected may be over-estimated since competition effects were not taken into account. This in turn may lead to an over-estimation of the growth

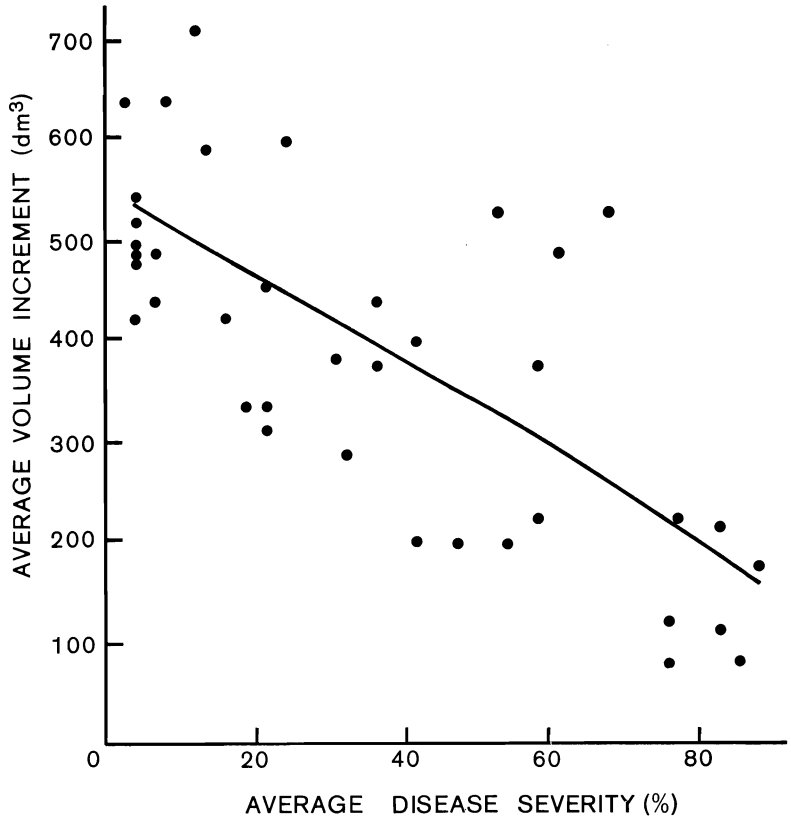

FIG. 3—Average volume increment as related to the average disease severity for the period from 1978 to 1983. The regression line is of the form $Y = b_1X$ where Y = volume increment (dm^3), b_1 = regression coefficient, and X = disease severity (%). Y = 537 - 4.29 X (N = 40, r^2 = 0.54).

reduction in diseased stands. It may be expected, however, that the opposite effects are of similar order of magnitude and cancel out.

Growth projections are based on volume loss caused by the observed disease severities in this trial (*see* Table 1). For the current regime of 400 stems/ha a total volume loss of 71 m^3/ha was predicted at age 30 with 50% of the final-crop trees diseased. When silvicultural regimes of 300 stems/ha and 200 stems/ha were similarly modelled with increasing proportion of diseased trees, total stand volume losses due to the disease decreased only slightly with decreasing number of stems per hectare. By running the projected yield data through program PROD, the contribution by log size changed noticeably, i.e., 25% fewer logs over 40 cm s.e.d. and an over-all 8–10% reduction in total harvested volume with increasing proportion of diseased trees. The financial impact of reduced yields on the net value of the stand was predicted by implementing the yield data in program SILMOD to show the approximate revenue losses that could

TABLE 5—Prediction of volume at age 30, top height 40 m, of stands* with increasing percentage of diseased trees (using the Kaingaroa Growth Model)

Diseased trees (%)	Basal area (m^2/ha)	Mean d.b.h. (cm)	Volume (m^3/ha)
400 stems/ha			
0	61.8	45.6	812
10	61.0	45.2	793
20	60.0	44.8	780
30	58.9	44.4	766
40	58.0	44.1	753
50	57.0	43.7	741
60	56.0	43.3	728
300 stems/ha			
0	60.4	51.2	797
10	59.4	50.7	784
20	58.5	50.3	772
30	57.5	49.9	758
40	56.5	49.4	745
50	55.6	49.0	733
60	54.5	48.6	719
200 stems/ha			
0	55.9	59.7	723
10	55.1	59.2	713
20	54.3	58.8	702
30	53.4	58.3	691
40	52.6	57.9	680
50	51.6	57.4	668
60	50.8	56.9	657

* Clearwood regime; site index 30; pruned at ages 5, 7, and 9; thinned at ages 5 and 9.

be expected in stands with similar conditions. It must be cautioned that the values and costs used in SILMOD vary considerably depending on stand conditions, market assumptions, and many other factors (the selected options are given in Table 6). The reduction of revenue at clearfelling increased by approximately \$600–\$700/ha for each 10% increase in the proportion of diseased trees (Table 7). When 50% of the final-crop trees were diseased the reduction in revenue that could be expected ranged from \$3200/ha to \$3600/ha. Differences in revenue between the silvicultural regimes were relatively small.

The predicted reductions of revenue indicate that in heavily diseased stands substantial financial losses may be expected and that control of the disease may be warranted. The options for action, however, are limited. Chemical control by spraying or injecting does not seem economical on a large scale mainly because of the high cost of the fungicides (Hood & Vanner 1984). The only option may be to select families genetically improved against needle loss associated with *C. minus*. This approach has already been adopted in the New Zealand *P. radiata* breeding programme. It is also possible that good control could be achieved by removing the most susceptible trees in a delayed first thinning at age 7, by which time disease symptoms should be visible.

TABLE 6—Selected variables for program SILMOD

Price for 100 × 50-mm No. 1 framing grade	$147.42/m³
Price for delivered pulpwood	$25.29/m³
Price for chip residue	$15.17/m³
Minimum s.e.d.	200 mm
Conversion factor	52.6%
Conversion standard	Average 2 mm overcut, 2.4 m min. length
Grading standard	Visual to maximise value
Mill	Waipa State Sawmill
Consumer price index	1369
Discount rate	10%
Harvesting method	Skidder
Topography	Flat
Distance from forest to mill	50 km
Thinning cost - first	$165/ha
Thinning cost - second	$62/ha
Pruning cost - first	$244/ha
Pruning cost - second	$185/ha
Pruning cost - third	$178/ha

TABLE 7—Changes in stand worth resulting from changes in harvestable volume and log-size distribution in stands with increasing percentage of diseased trees (using SILMOD)

Diseased trees (%)	Net decrease in total volume (m³/ha)	Net decrease in harvestable volume (m³/ha)	Total cost ($000/ha)	Total revenue ($000/ha)	Net decrease due to disease ($000/ha)
400 stems/ha					
0			56.8	56.1	
10	19	16	55.5	54.1	0.7
20	32	27	54.9	52.9	1.3
30	46	38	54.3	51.7	2.0
40	59	47	54.3	51.0	2.6
50	71	58	53.8	49.9	3.2
60	84	67	53.4	48.9	3.8
300 stems/ha					
0			53.0	56.6	
10	13	11	52.6	55.5	0.7
20	25	20	52.2	54.5	1.4
30	39	30	51.7	53.3	2.1
40	52	41	51.8	52.5	2.9
50	64	49	51.4	51.5	3.5
60	78	61	50.9	50.2	4.3
200 stems/ha					
0			45.7	54.3	
10	10	8	45.4	53.4	0.7
20	21	16	45.2	52.4	1.4
30	32	25	44.9	51.4	2.1
40	43	33	44.6	50.5	2.8
50	55	43	44.3	49.2	3.6
60	66	50	44.0	48.4	4.2

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