GROWTH DECLINE AND PHOSPHORUS RESPONSE BY DOUGLAS FIR ON A DEGRADED HIGH-COUNTRY YELLOW-BROWN EARTH

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ABSTRACT

An 18-year-old **Pseudotsuga menziesii** (Mirb.) Franco (Douglas fir) plantation growing on a hygrous high-country yellow-brown earth soil in Canterbury showed localised symptoms of ill-health – absence of cones, stunting and chlorosis of needles, premature needle cast, and a premature decline in shoot growth and basal area increment. Soil and foliar nutrient analyses indicated phosphorus deficiency was the principal cause of the growth disorder, and this was confirmed by growth responses to applied phosphorus in field and greenhouse trials. Soil exchangeable aluminium levels were high and aluminium toxicity may have compounded phosphorus deficiency problems. In the field trial, superphosphate increased needle nitrogen content, but no response was obtained to nitrogen applied as urea either alone or in combination with other nutrients. Urea significantly reduced phosphorus uptake at the end of the first growing season, but not the second. In contrast to the field trial, Douglas fir seedlings in the greenhouse trial responded to nitrogen applied as ammonium nitrate, provided phosphorus was also applied. It is suggested that immobilisation of urea-nitrogen in soil organic matter may have contributed to the failure of trees in the field trial to respond to nitrogen.

Keywords: growth decline; soil analysis; foliage analysis; fertilisers; phosphorus; **Pseudotsuga menziesii**.

INTRODUCTION

A zone of high productivity of conifers has been identified in the moist (annual precipitation > 1200 mm) western sector of the Canterbury high country (Ledgard & Belton 1985). Douglas fir was one of the five most common species and some stands of this species were amongst the most productive ever recorded in New Zealand or elsewhere. Most stands appeared to be free of disease and symptoms of nutrient deficiency. An exception was a young plantation located at Burnt Face (Waimakariri Valley), parts of which showed yellowing of the foliage, reduced needle retention, and a recent marked decline in height growth.

Although exotic conifers currently occupy less than 1% of the land area in the Canterbury high country, forestry provides an opportunity for a more diversified and

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productive land use, particularly in the moist western sector. Douglas fir would be one of the more favoured species for planting because of its potentially high growth rates and the ready marketability of its timber. The health disorder in Douglas fir at Burnt Face was therefore investigated.

SITE DESCRIPTION

Burnt Face (43° 03′ S, 171° 40′ E) is located on a north-facing slope of the Black Range in the Waimakariri Valley, Canterbury. Slopes in the study area range between 15° and 25°, and good drainage is provided by several shallow ephemeral streams. A regolith of about 1–4 m depth of talus or Otira tills covered in loess overlies indurated but structurally weak greywacke (Gregg 1964; J. B. J. Harrison pers. comm.). Soils are podsolised yellow-brown earths of the Bealey Hill set (New Zealand Soil Bureau 1968). A period of slope instability after destruction of the mountain beech (*Nothofagus solandri* var. *cliffortioides* (Hook. f.) Poole) forest early this century has resulted in considerable mixing of surface materials. Most of the soils examined had composite or truncated profiles.

Annual precipitation at Bealey*, 1.5 km from Burnt Face, averages 1670 mm (New Zealand Meteorological Service 1973), with 570 mm falling during the main growing period between November and March. Short summer droughts of several weeks' duration are not infrequent. Winter frosts are commonly in the range of −5 to −8°C, and frosts can also occur throughout the growing season. Large diurnal temperature fluctuations, frequent winter snowfalls, and strong, rain-bearing, north-west winds are also features of the climate.

STAND HISTORY

After removal of the beech forest, Burnt Face was grazed for about 60 years. The area was retired from grazing about 1964 because of soil erosion, and between 1965 and 1971 a plantation was established for soil protection and experimental purposes. The study area was located in an 11-ha stand of Douglas fir planted in 1965, between 750 and 950 m a.s.l. Original stocking was 2250 stems/ha and after 2 years survival was estimated as 86%, mortality being attributed to animal damage and frosting (N.Z. Forest Service unpubl. data).

In autumn 1976, when mean top height was 8 m, thinning reduced stocking to about 960 stems/ha. Wide variation in tree size was recorded (N.Z. Forest Service unpubl. data). By 1980, yellowing and poor growth had been noted throughout areas of the 1965 plantings, and further thinning was postponed. By 1982 yellowing was more pronounced and the investigation reported here was initiated.

METHODS

Stand Growth and Health Disorder Symptoms

Two mensuration plots (500 m^2) were established 200 m apart in healthy and unhealthy stand areas at 780 m a.s.l. to provide information on stand growth. Height and diameter at breast height (1.4 m) of all trees were measured in each plot, and increment cores were taken from 15 trees in each plot to determine stand basal area increment (b.a.i.).

* Station now closed.

The frequency of occurrence of major symptoms of the health disorder was determined in the fertiliser trial area (see below). All trees in the trial area were scored for (1) presence or absence of cones, (2) severity of stunting of needles and branchlets, (3) needle chlorosis, and (4) needle cast. Since needle retention on healthy trees averaged 7 years, needle cast was recorded when retention was less than 4 years. Symptoms (2) to (4) were placed in three categories of severity: insignificant, moderate (< 50% of tree), or severe (> 50% of tree).

Foliage and fine roots were examined by pathologists in the field and in the laboratory for the presence of pests and diseases. Roots were also examined for the presence of mycorrhizas.

Foliage and Soil Analyses

Current-year shoots were taken in March 1982 from the tips of fourth- or fifth-year branch clusters in the upper crown from 10 unhealthy and 10 healthy trees in the area to be used for the fertiliser trial (see below). The 10 samples from each category of the tree were combined to give a single composite sample of each. Foliage was stripped from the shoots and oven-dried at 70°C for 24 hours, then analysed for nitrogen, phosphorus, potassium, calcium, magnesium, manganese, zinc, copper, iron, boron, and aluminium using the methods described by Nicholson (1984).

Soil samples were collected from beneath the 15 trees sampled for b.a.i. from both the mensuration plots. Four soil cores (0–20 cm depth, 2.5 cm diameter) were taken from north, south, east, and west positions 1.5 m out from the base of sample trees. These were bulked for each sample tree after being split into two sampling depths (0–10 and 10–20 cm). Soil samples were air-dried and passed through a 2-mm-mesh sieve before analysis for pH (H_2O and 1N KCl) and Bray-2 extractable phosphorus, using the methods described by Nicholson (1984). Exchangeable calcium, magnesium, aluminium, and manganese were determined by atomic absorption spectrophotometry after extraction with 1N KCl.

Field Trial

A trial to examine the effect of fertilisers on tree growth was established in an area where the health disorder symptoms were particularly severe. The trial design used a randomised block layout, with five treatments (control, N, P, N + P, and N + P + other nutrients) and three replicates. The fertilisers, and nutrient element application rates (kg/ha), were urea (N, 200), superphosphate (P, 100), and a mixture of other fertilisers consisting of dolomite (Ca, 226; Mg, 135) sodium borate (B, 10), and the sulphate salts of potassium (125), zinc (22), manganese (32), and copper (18). Plot size was 150 m^2, and each plot contained an average of 15 trees. All secondary vegetation, mostly manuka (*Leptospermum scoparium* J.R. et G. Forst.), in the trial plots and in buffer strips 5 m wide was slashed at ground-level.

In each plot, every tree was numbered and its dbh recorded within the internode nearest to 1.4 m above ground-level on the upslope side. The position of dbh measurement was circled with paint for future remeasurement. Current-year foliage was obtained from fourth- or fifth-whorl branches in the upper crown of the eight smallest diameter trees in each plot. From composite samples for each plot, 200 needles were

randomly selected, oven-dried for 24 hours at 70°C, and weighed. Nitrogen and phosphorus were determined using methods described by Nicholson (1984).

Foliage sampling and dbh measurements were done immediately before the establishment of the fertiliser trial (October 1982) and then in April 1983 and 1984. Shoot extension over the trial period was measured on branches taken in April 1984 from the upper crown of the eight trees sampled for foliage in each plot.

Analysis of covariance was used to examine responses in needle weight, needle nitrogen and phosphorus content, and shoot and basal area growth over the trial period.

Greenhouse Trials

The field fertiliser trial was established in the largest uniform area of unthrifty trees on Burnt Face, but the area available was not large enough to test the treatments used in all possible combinations. Also, as phosphorus and sulphur were applied together (as superphosphate) it was impossible to distinguish the effects of these two nutrients.

A greenhouse nutrition trial was set up to overcome these problems. Although nutrient factors tested (nitrogen, phosphorus, other nutrients) were similar to those in the field trial, they were compared in all possible combinations. Other differences were (i) the rate of nutrient elements applied, (2) the form of nitrogen applied (ammonium nitrate instead of urea), and (3) the source of phosphorus (sodium dihydrogen orthophosphate was used to allow phosphorus to be tested independently of sulphur). To determine whether soil pathogens were involved in the health disorder, another greenhouse trial examined the effects of soil sterilisation on the growth of Douglas fir seedlings.

Soil for both trials was collected from beneath unhealthy trees on Burnt Face to a depth of 20 cm. The soil was passed through a 6-mm sieve and then split into two portions, one for the nutrition trial, the other for the sterilisation trial.

Nutrition trial

A randomised block design (2^3) with five replicates was used to examine the effects of presence or absence of nitrogen, phosphorus, and a combination of other nutrients on the growth of Douglas fir seedlings. Nitrogen and phosphorus were applied at the rate of 50 kg/ha as NH_4NO_3 and $NaH_2PO_4.2H_2O$ respectively. The other nutrient treatment contained 50 kg K/ha, 20 kg Mg/ha, 1 kg Cu/ha, 2 kg Zn/ha, and 2 kg Mn/ha, all as sulphates (total sulphur 48.5 kg/ha); and 5 kg Fe/ha as FeNa EDTA, 50 kg Ca/ha as $CaCl_2.2H_2O$, and 1 kg B/ha as $Na_2B_4O_7.10H_2O$. All nutrients were applied in solution to the soil surface after the seedlings had been planted. The pots (14 cm top diameter, 13 cm deep) contained 1.3 kg of soil. Seedlings were germinated on water-washed sand and were transplanted (five per pot) in December 1982. The pots were supplied with drainage holes and watered as necessary to maintain the soil in a moist condition.

Sterilisation trial

Soil for the sterilisation trial was divided into two equal quantities and one batch was steam sterilised at 80°C for 30 min. After sterilisation, duff (collected from

beneath a healthy stand of Douglas fir) was mixed with both soils (200 ml duff to 1.3 kg soil) to inoculate seedlings with mycorrhizas. The effect of sterilisation was examined in the presence or absence of the complete fertiliser treatment used in the fertiliser trial. Other conditions were the same as for the fertiliser trial, except that the seedlings were transplanted 1 month later (January 1983).

In both trials treatments with nitrogen received a repeat application (50 kg N/ha as NH_4NO_3) in October 1983. The trials were harvested in December 1983 by cutting stems at the soil surface. Harvested material was weighed after oven-drying.

RESULTS

Stand Growth and Health Disorder Symptoms

Trees in the mensuration plot in the healthy stand at 780 m had a mean top height of 14.5 m, mean dbh of 21.5 cm, and mean volume increment of 10.4 m^3/ha/yr. The canopy had closed since thinning in 1976 and all ground vegetation was suppressed. In contrast, trees in the unthrifty plot 200 m along the contour had a mean top height of 10.0 m, mean dbh of 10.9 cm, and mean volume increment of 1.9 m^3/ha/yr. In this area, the stand structure was open and an understorey of manuka was developing.

Stand basal area increment increased in the healthy stand after the 1976 thinning (Fig. 1), but declined after the summer of 1980. Stand basal area at 19 years was 37.9 m^2/ha, and the mean increment of 2.0 m^2/ha/yr is low compared to a range of 3.6 to 4.0 m^2/ha/yr for 20-year-old stands elsewhere in New Zealand (Beekhuis 1978). The basal area of the unhealthy stand at 19 years was only 9.1 m^2/ha and basal area increment had declined steadily since thinning.

Many trees in the unhealthy sample had main stem internodes of about 70 cm length in the early 1970s, but were averaging less than 10 cm apical shoot growth by 1982. Height growth declined by the mid 1970s in severely affected trees and more recently for less severely affected trees. Reduction in shoot growth and needle length

FIG. 1—Annual basal area increment since thinning for healthy (————) and unhealthy (— — — — —) stands. Data are from increment cores taken from trees in the mensuration plot (n = 15).

gave foliage of unhealthy trees a characteristic stunted appearance. Stunting of branchlets and foliage was evident over the entire canopy in severely affected trees, but was restricted to the lower half of the canopy in less affected trees. The most severely affected trees carried only 1 or 2 years' needles compared to 7 years for healthy trees. Absence of cones was the most widespread symptom, and it appeared to signal the onset of deterioration in health.

In the fertiliser trial area, most trees showed some symptoms of the disorder. Only 13% of trees had cones. Most trees (70%) had varying degrees of needle and branchlet stunting and chlorosis and some 60% of trees were affected by premature needle cast. Combinations of symptoms varied for individual trees. The root grafting characteristic of Douglas fir is often shown by stump callousing after thinning but calloused stumps were rare in the thinned area on Burnt Face and occurred only within groupings of healthy trees.

Foliage and fine roots examined by pathologists showed no sign of pathogens (P. D. Gadgil pers. comm.; I. A. Hood unpubl. data). Swiss needle-cast fungus (*Phaeocryptopus gaeumannii* (Rohde) Petrak) and needle damage by *Lepidoptera* spp. larvae, which have been implicated in defoliation and growth decline of Douglas fir elsewhere in New Zealand (Cameron *et al.* 1978), were not detected. Larvae of grass grub chafer beetles were frequently found in soil samples, and the possibility that their feeding causes significant damage to fine roots cannot be discounted.

In the field, few mycorrhizas were found on roots of unhealthy trees and they were not abundant on healthy trees (I. A. Hood unpubl. data). However, many sporocarps of *Boletus* sp. were seen during the autumn trial assessments. The density of sporocarps near healthy trees was approximately five times higher than that near unhealthy trees. Laboratory investigations revealed that fine root samples of both unhealthy and healthy trees had mycorrhizas of the type formed by *Rhizopogon* and *Boletus* spp. (M. Chu-Chou pers. comm.). *Rhizopogon parksii* A. H. Smith, which is considered to be a good symbiont for Douglas fir, was cultured from root samples of unhealthy trees (M. Chu-Chou pers. comm.).

Foliage and Soil Analyses

Levels of all nutrients determined (except magnesium) were lower in the unhealthy trees than in the healthy trees (Table 1). Critical levels of macro-nutrients in current-year foliage from the upper crown of Douglas fir in California sampled late in the growing season have been given by Powers (1983) as N 1.20%, P 0.15%, K 0.60%, Ca 0.12%, and Mg 0.06%. Binns *et al.* (1980) gave tentative values for deficient levels in foliage from the uppermost whorl of branches of Douglas fir trees (top height less than about 5 m) in Britain which are similar (N 1.2%, P 0.18%, K 0.6%, Mg 0.04%). The healthy trees on Burnt Face had phosphorus levels below the critical levels, and the unhealthy trees were deficient in both nitrogen and phosphorus. Levels of other macro-nutrients seemed adequate.

Critical levels have not been established for micro-nutrients in Douglas fir. Oldencamp & Smilde (1966) found that copper-deficient trees usually contain < 2.6 ppm in current-season's (1-year-old) needles sampled in autumn, but Binns *et al.* (1980) found that trees with 1.5 ppm Cu in autumn-sampled foliage showed no signs of copper

TABLE 1—Nutrient concentrations in current-year needles of healthy and unhealthy trees from the fertiliser trial area (data are from composite samples from 10 trees)

	Healthy trees	Unhealthy trees
N (%)	1.5	0.9
P (%)	0.10	0.08
K (%)	0.91	0.75
Ca (%)	0.44	0.30
Mg (%)	0.09	0.09
Al (μg/g)	374	288
Mn (μg/g)	525	160
Fe (μg/g)	40	30
Zn (μg/g)	14	13
Cu (μg/g)	4	2
B (μg/g)	9	6

deficiency. Boron levels in unhealthy trees (6 ppm) were similar to those measured by Carter *et al.* (1984) in a 30-year-old Douglas fir plantation in British Columbia which was affected by severe dieback and distorted growth. However, none of the major symptoms the authors attributed to boron deficiency were observed at Burnt Face.

There was no significant difference in pH between soils under healthy and unhealthy trees (Table 2). Bray-2 phosphorus values were low (Ballard 1970) in both healthy and unhealthy stands, but the healthy stand had twice the Bray-2 phosphorus level of the unhealthy stand ($p < 0.01$ for the 0–10 cm samples). Exchangeable calcium and magnesium levels were low (Miller 1968), and the values beneath healthy trees were

TABLE 2—Some chemical characteristics of soils from beneath healthy and unhealthy Douglas fir stands (n = 15; SEM in parentheses)

	0–10 cm		0–10 cm	
	Healthy	Unhealthy	Healthy	Unhealthy
pH (KCl)	3.8(0.03)	3.8(0.03)ns	3.7(0.03)	3.9(0.03)ns
pH (H_2O)	4.8(0.04)	4.9(0.04)ns	4.7(0.05)	5.0(0.03)ns
Bray-2 P (μg/g)	5.50(0.64)	2.71(0.27)**	2.86(0.43)	1.54(0.14)*
Al (me. %)	5.85(0.57)	5.85(0.29)ns	5.90(0.53)	7.91(0.66)ns
Ca (me. %)	1.64(0.16)	2.53(0.14)*	0.86(0.15)	1.42(0.15)ns
Mg (me. %)	0.32(0.03)	0.40(0.01)*	0.17(0.02)	0.15(0.01)ns
Mn (me. %)	0.026(0.003)	0.040(0.003)*	0.004(0.001)	0.010(0.001)*

t-test comparison between healthy and unhealthy stands:
ns not significant
* significant at $p < 0.05$
** significant at $p < 0.01$

mostly lower than those beneath unhealthy trees. Exchangeable manganese levels were higher beneath unhealthy trees than healthy trees at both sampling depths ($p < 0.05$), while the aluminium levels were marginally higher beneath unhealthy trees in the 10–20 cm samples ($p < 0.12$). Soils in both stands contained higher levels of exchangeable calcium, magnesium, manganese, and Bray-2 phosphorus in the 0–10 cm than in the 10–20 cm horizon.

Field Trial

The addition of superphosphate alone increased needle phosphorus content significantly ($p < 0.05$) at the end of the first growing season after fertiliser application (Fig. 2). Smaller (non-significant) increases in needle phosphorus content were also recorded in the treatments which combined superphosphate with urea and other nutrients. The other measured parameters (needle weight, needle nitrogen content, shoot and basal area growth) showed no response to any treatment by the end of the first growing season.

By the end of the second growing season all five measured parameters showed significant responses to superphosphate (Table 3). These responses were unaffected by the presence or absence of nitrogen or other nutrients. Superphosphate alone more than doubled needle phosphorus content and basal area growth, while needle weight,

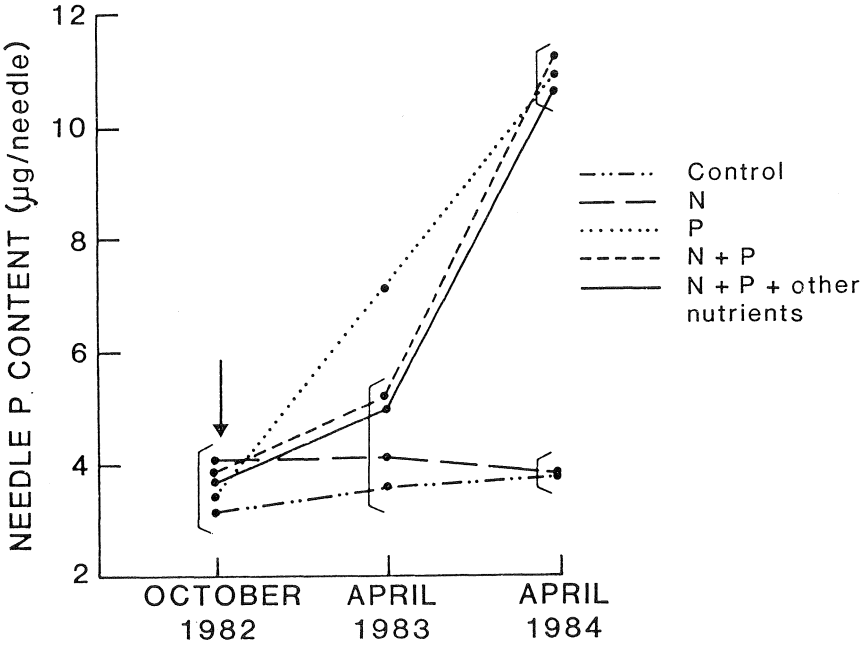

FIG. 2—Effect of fertilisers on needle phosphorus content in the field trial. Values linked by square brackets are not significantly different ($p < 0.05$) by Duncan's Multiple Range Test. The arrow indicates time of fertiliser application. The 1984 values differ slightly from those in Table 3 which were adjusted by covariance analysis.

TABLE 3—Effect of fertiliser treatments on needle weight, needle nitrogen and phosphorus contents, and shoot and basal area growth. Data are from measurements and samples taken in April 1984, two growing seasons after trial establishment. Means are adjusted by co-variance analysis. Means with the same letter do not differ significantly ($p < 0.05$) by Duncan's Multiple Range Test. SEM is given in parentheses

	Control	N	P	N + P	N + P + other nutrients
Needle weight (mg/needle)	5.06 a (0.15)	4.57 a (0.24)	7.95 b (0.33)	7.37 b (0.82)	7.10 b (0.45)
Needle nitrogen (μg/needle)	75 a (2.9)	66 a (2.3)	101 b (4.7)	112 b (8.9)	106 b (8.0)
Needle phosphorus (μg/needle)	3.9 a (0.43)	3.7 a (0.12)	10.9 b (0.75)	11.1 b (1.26)	10.6 b (0.70)
Shoot growth (cm/100 cm)	19.7 a (1.59)	20.6 a (1.17)	27.3 b (1.48)	24.1 ab (1.40)	27.8 b (1.99)
Basal area growth (cm^2/100 cm^2)	7.2 a (0.60)	7.3 a (0.47)	15.8 b (0.85)	14.2 b (0.84)	14.1 b (1.07)

needle nitrogen content, and shoot growth showed increases of 57%, 35%, and 38% respectively. Superphosphate alone marginally, but not significantly, depressed needle nitrogen concentration. Urea had no effect on needle nitrogen content, nor on any of the other parameters. Similarly, there was no response to the other fertilisers applied in combination with the urea and superphosphate treatments.

Greenhouse Trials

In the nutrition trial, seedlings showed a strong response to phosphorus (Table 4), confirming the results of the field trial. However, in contrast to the field trial, there was also a significant response to nitrogen in the presence of phosphorus, which resulted in a significant N × P interaction. There was no response to other nutrients, either alone or in combination with nitrogen and phosphorus.

There was a strong response to fertiliser in the sterilisation trial (Table 5), but no response to sterilisation, indicating that soil pathogens were not involved in the health disorder of Douglas fir at Burnt Face.

DISCUSSION

Fertiliser trials indicated that phosphorus deficiency is the principal cause of the decline in growth of Douglas fir at Burnt Face. Supporting evidence for phosphorus deficiency is provided by the critically low phosphorus concentrations in current-year

TABLE 4 — Effect of nitrogen, phosphorus, and other nutrients on the growth of Douglas fir seedlings in the greenhouse trial. Results are oven-dry weights (g/shoot)

	Nitrogen (kg/ha)	Phosphorus (kg/ha)	
		0	50
Without other nutrients (–)	0	0.13	0.30
	50	0.11	0.38
	Mean	0.12	0.30
With other nutrients (+)	0	0.14	0.25
	50	0.11	0.47
	Mean	0.13	0.36

F value and significance from analysis of variance

N	7.38*	NP	15.15 ***
P	98.77 ***	N+	2.29 ns
+	0.24 ns	P+	0.02 ns
		NP+	2.69 ns

SEM = 0.016

TABLE 5—Effect of sterilisation in the presence and absence of complete fertiliser on the growth of Douglas fir seedlings. Results are oven-dry weights (g/shoot)

	Unsterilised	Sterilised
Without fertiliser	0.18	0.25
With fertiliser	0.43	0.37
Mean	0.30	0.31

F value and significance from analysis of variance

Sterilisation	0.01 ns
Fertiliser	26.03 ***
S × F	2.76 ns

SEM = 0.026

foliage of unhealthy trees and, less directly, by the low levels of available soil phosphorus measured by Bray-2 extractions. A gradual decline in growth, evident in the basal area increment histories (Fig. 1) as well as in the shoot extension measurements, is characteristic of phosphorus deficiency (Binns *et al.* 1980). The low foliar and soil phosphorus levels and the premature decline in basal area increment recorded in apparently healthy stands indicate that phosphorus deficiency is not restricted to areas showing the more obvious visual symptoms.

A check in the growth of Douglas fir, with similar symptoms, also attributed to phosphorus deficiency, has been observed in a 16-year-old stand at Longwood State Forest (Southland). The Longwood yellow-brown earth topsoils associated with deficiency symptoms had Bray-2 phosphorus levels of 1.9 μg/g, while soils beneath healthy trees had marginally higher levels of 2.5 μg/g (P. J. Knight pers. comm.). These values are about half of those for trees of corresponding health at Burnt Face, suggesting that some additional factor may have been limiting phosphorus uptake and tree growth there.

Marked acidification can occur in soils beneath coniferous plantations (Nilsson *et al.* 1982). At Burnt Face the soil pH (H_2O) at sampling depths of 0–10 and 10–20 cm beneath grassland within 10–15 m of the plantation edge was 5.3 ($n = 13$). Soil pH in the mensuration plots varied between 4.7 and 5.0 (Table 2), indicating a pH decline of between 0.3 and 0.6 units beneath the developing plantation. In acid soils, aluminium toxicity can impair phosphorus uptake and use, and cause restricted, or abnormal, root growth in many crop and pasture species (Foy 1974). Less is known of the effect of aluminium toxicity on the growth of coniferous species. However, James *et al.* (1978) found a strong negative association between growth and foliar aluminium concentration in Sitka spruce (*Picea sitchensis* (Bong.) Carr.) in Scotland, and Ulrich *et al.* (1980) suggested that aluminium toxicity may be associated with dieback in European forests subject to acid deposition. Results of recent studies support this suggestion (for reviews *see* Hüttermann & Ulrich 1984; Hüttermann 1985). At Burnt Face, soil exchangeable aluminium levels were high, and were marginally higher beneath unhealthy trees than healthy trees in the 10–20 cm zone ($p < 0.12$). Under the influence of the developing plantation, acidification in the rhizosphere may have promoted critical levels of available aluminium, thereby reducing root growth and phosphorus uptake and compounding the problems of phosphorus deficiency. Because of lower soil phosphorus levels these effects would have been greater beneath unhealthy trees than healthy trees. However, the above suggestion is not supported by the fact that foliar aluminium levels were higher, and soil pH levels were slightly lower, in healthy than in unhealthy stands (Tables 1 and 2).

The failure of trees in unhealthy stand areas to respond to nutrient release after thinning and pruning in 1976 (Fig. 1) and the patchy occurrence of symptoms of the disorder at Burnt Face suggested that disease or perhaps mycorrhizal failure may have contributed to the growth decline. Field and laboratory observations of foliage and fine roots showed no sign of disease, and the soil sterilisation trial confirmed the absence of soil pathogens. Absence of pathological factors is consistent with the findings of a recent survey of exotic forest stands in the Canterbury high country which showed little evidence of pathogens in Douglas fir (Ledgard & Belton 1985). Suitable species of root mycorrhizas were present, but were more abundant beneath healthy than unhealthy trees. It is uncertain whether the poorer development of mycorrhizas beneath unhealthy trees contributed to the growth decline, or was a result of reduced vigour and root growth of affected trees.

Soil profile morphology varied locally in the study area as a result of soil disturbance after removal of the original beech forest. Differences in soil physical properties such as bulk density and texture may therefore have contributed to the variable growth.

While no detailed study of soil physical properties was made, an examination of profiles in healthy and unhealthy stand areas did not reveal any obvious relationship between profile morphology and stand health.

The absence of a growth response to nitrogen applied as urea in the field trial was surprising, considering that a significant response was obtained to ammonium nitrate applied in conjunction with phosphorus in the greenhouse trial. The difference in response may have been due to the different forms of nitrogen applied. In Scandinavia a number of studies have shown that Scots pine (*Pinus sylvestris* L.) responds more to ammonium nitrate than to urea on infertile, mineral soils (for review *see* Ballard 1984). Some studies with Douglas fir in Canada (Dangerfield & Brix 1979; Brix 1981) and the United States Northwest (Harrington & Miller 1979) support these findings. The superior performance of ammonium nitrate compared to urea has been attributed to greater volatilisation losses of ammonia-nitrogen after hydrolysis of urea, and/or higher immobilisation of urea-nitrogen in soil organic matter (Ballard 1984). In the present study it is unlikely that major losses of nitrogen through volatilisation occurred as 130 mm of rain was recorded at Craigieburn, 20 km south-east of Burnt Face, in the month after fertiliser application. Immobilisation of urea-nitrogen in soil organic matter therefore seems the more likely explanation for the failure of trees to respond to urea. Mineralisation of the immobilised nitrogen may have been limited by phosphorus deficiency, as is suggested by the fact that nitrogen uptake was increased significantly by superphosphate (Table 3).

Gill & Lavender (1983) have shown that urea may damage mycorrhizas of western hemlock (*Tsuga heterophylla* (Raf.) Sarg.) roots close to the soil surface for periods of up to 9 months after fertiliser application. It is possible that similar damage occurred in the present study as needle phosphorus contents at the end of the first growing season were significantly lower in treatments combining urea with superphosphate than in treatments with superphosphate alone (Fig. 2), indicating that phosphorus uptake was reduced by urea. Such damage would also have contributed to the failure of trees to respond to urea.

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