ARTICLES

Cyclaneusma needle-cast and Dothistroma needle blight in NZ pine plantations

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Abstract

Cyclaneusma minus (Butin) DiCosmo *et al.* and *Dothistroma pini* Hulbary both cause severe defoliation of pine trees on some sites. *Cyclaneusma* affects one-year-old foliage, is most severe after a mild wet winter, and is most common on 11- to 20-year-old trees; whereas *Dothistroma* affects current foliage, is most severe after a wet summer, and trees are usually resistant after about 15 years. Trees with average *Cyclaneusma* disease levels of 80% showed a reduction in volume increment of 60%; and if 50% of final-crop trees are affected by the disease, losses of 71m³/ha and $3200/ha in net revenue were predicted at the end of the rotation. A *Dothistroma* infection level of 50% over several seasons would result in a 50% reduction in volume increment. *Cyclaneusma* may have a more significant effect on stand growth than *Dothistroma* because it affects trees from six to 20 years old, whereas *Dothistroma* affects trees from planting to 15 years old, thereby creating the opportunity to cull highly susceptible, stunted trees during the first and second thinning operations.

At high-risk sites, *Cyclaneusma* can be controlled by selecting susceptible trees and culling them during the first and second thinning operations. Aerial applications of copper fungicide to control *Dothistroma* were effective in unpruned, highly stocked stands; growth response to spraying was difficult to repeat in stands which had been pruned and thinned regularly.

Introduction

The pine forests of New Zealand suffer from two significant defoliating diseases caused by *Cyclaneusma minus* and *Dothistroma pini*. Both diseases are present throughout New Zealand, although their prevalence varies from region to region. *Cyclaneusma* is associated with premature casting of one-year and older foliage, and usually affects trees aged between six and 20 years (Bulman 1988). *Dothistroma* results in the loss of the current year's foliage and affects trees from planting, but trees normally become resistant by the age of 15 years (Bassett 1972). Both diseases cause growth loss through defoliation (Gilmour *et al.* 1973; van der Pas 1981, van der Pas *et al.* 1984 a,b), and *Dothistroma* has been recorded to cause tree death in extreme cases (Gilmour 1967).

The disease development, distribution, extent, and effect on tree growth of both fungi are discussed, along with options for control.

Biology

Spore release of *Cyclaneusma* occurs during periods of rain, and peaks in autumn and winter, which leads to infection of foliage older than six months. Infected needles turn mottled yellow-green, then brown in spring and most are cast by summer when they are about one year old. Infection is usually most apparent in the mid-crown of affected trees. Some infected needles may linger on the tree and are cast the following autumn when they are about 20 months old.

Infection can take place over a wide range of temperatures from 10 to 25°C (Gadgil 1984), but the weather in spring and summer when foliage is resistant to infection and spore levels are low cannot affect the severity of the disease. Outbreaks can be expected during a spring following a mild and wet autumn and winter.

Infection by *Dothistroma* depends on three main factors – duration of needle-wetness period, temperature, and the amount of spores present. Spores of *Dothistroma*, like *Cyclaneusma*, are liberated in a film of water. Infection usually starts at the base of the tree and progresses upwards. Brick-red bands appear on green needles and infected needles eventually die and are prematurely cast. Defoliation is most apparent between September and October but begins in summer on current foliage. Severe infection takes place when temperatures are mild (16-20°C), needles are moist for over 10 hours, and inoculum levels are high.

Distribution

Cyclaneusma is present throughout New Zealand, although the disease is more severe in some regions than others. An aerial survey carried out over three consecutive years from 1983 to 1985 showed the disease was most severe in East Cape forests, and severe outbreaks were recorded in Otago, Northland, and the central North Island (Bulman 1988). Levels were low in Westland

Cyclaneusma.

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and Nelson over all three years. The disease is not evenly distributed throughout stands; rather, infected trees susceptible to the disease are scattered among healthy, resistant ones. The same trees are affected from year to year (van der Pas *et al.* 1984b), the degree of infection depending on the climate prevailing during the previous winter.

The aerial surveys showed the disease is most severe in trees aged between six and 20 years: approximately 10% of the trees in this age-class had disease levels greater than 40%. Stands younger than six years had little disease, and stands older than 25 years were commonly affected but only at trace levels.

Dothistroma occurs in all parts of New Zealand, but is most severe on the west coast of the South Island and in the central North Island. Levels are very low in drier areas such as Canterbury, Central Otago, and the east coast of the North Island. Disease levels are usually uniform throughout stands, although some trees display more resistance than others.

Effect on Tree Growth

In a trial at Kaingaroa Forest, 20 pairs of trees (each pair consisting of a healthy tree and a heavily diseased tree) were selected to examine if defoliation by Cyclaneusma needle-cast would lead to significant growth loss. Average disease levels over a six-year period were 9% for the healthy trees and 59% for the diseased trees. Volume growth of the diseased trees was initially slightly faster than that of the healthy trees to age six but from age eight to when the sample trees were felled growth reduction of the diseased trees was significant, resulting in a difference between diseased and healthy trees of almost 100 m^3/ha at age 15. An average disease level of 80% led to a reduction in volume increment of 60%. Losses of 71 m^3/ha were predicted at the end of rotation, assuming 50% of the final-crop trees were affected by the disease. Such losses were estimated to reduce net revenue by $3200/ha (van der Pas *et al.* 1984b).

Tree growth from establishment to eight years in a plantation at Kaingaroa Forest was related to infection levels of *Dothistroma*. Volume loss on an individual tree basis was approximately proportional to disease level, where an average disease level of 50% over eight years resulted in a volume loss of almost 50% (van der Pas 1981). Diameter-at-breast-height growth was reduced to a lesser extent, where the percentage loss was about half the average disease level, i.e., 40% disease level would lead to a 20% reduction in diameter growth.

It is clear that both diseases can cause significant growth loss to individual trees. It is more difficult to estimate growth loss on a stand basis. Compensation of growth by healthy trees taking advantage of reduced competition from diseased trees (Zadoks & Schein 1979) takes place in densely stocked stands, but is not important prior to crown closure, or in thinned stands. Because *Dothistroma* affects trees from planting to about age 15, growth loss on a stand basis may be significant prior to the first thinning at age six. After the first thinning usually the smaller, highly susceptible trees are culled, eliminating much of the growth loss. A similar effect occurs during subsequent thinnings. After successful disease control in two aerial spray trials stand growth was significantly greater in the sprayed treatments than the unsprayed controls, but differences were eliminated after the second thinning (van der Pas 1984a).

Cyclaneusma is most severe in stands aged between six and 20 years old. Stands usually receive the first thinning prior to the first appearance of the disease, and the thinning to final-crop stocking at about age 10. The disease, if not reduced by culling susceptible trees during the final thinning, has the opportunity to affect final-crop trees from 10 to 20 years old, during the period of greatest periodic annual increment.

Disease Control

There are only two practical ways to control Cyclaneusma needle-cast – selection of resistant trees through the tree breeding programme and selection of resistant trees during thin-to-waste operations. Aerial applications of fungicides have proved unsuccessful and expensive (Vanner 1986), and although some success has been achieved by injecting trees with systemic fungicides (Hood *et al.* 1984) this control method is not feasible for large-scale forestry operations.

Because some trees show marked susceptibility to Cyclaneusma needle-cast it is possible to identify and cull susceptible trees during thinning operations. At two trial sites in Kaingaroa Forest, trees received their first thinning at ages ranging from five to nine years with thinning selection based on disease severity where the most severely diseased trees were removed. All treatments received a final thinning at age 10, using the same selection criterion. At one site, where disease levels were high, the plots that were thinned at four and five years when it was too early to select for disease susceptibility had average disease levels of 23% (25% of the trees had disease levels of 40% or greater) at age 11. The plots thinned at eight and nine years, when disease symptoms were apparent and selection could take place, had disease levels of 11% (1% of the trees had disease levels of 40% or greater). At age 11 the trees in the plots thinned early were smaller than those in the later-thinned plots, and this growth difference is expected to widen because disease levels are expected to remain high in the susceptible trees until about age 20.

At the other site, where disease levels were low, thinning age made no difference to disease in the final crop, and trees in the later-thinned plots were smaller than those in the early-thinned plots. Delayed thinning reduced tree growth because of suppression effects. At locations where high disease levels are predicted, such as East Cape, Otago, Northland, and parts of the central North Island, selective thinning should result in improved growth of final-crop trees, even after allowing for initial increment reduction due to suppression.

Resistance to Cyclaneusma needle-cast had been proved heritable, and trees with good needle retention have been selected for the tree breeding programme. Results so far are promising, and growers planting high-risk sites should consider using seedlings with high needle retention ratings.

Stands affected by Dothistroma needle blight have been sprayed with aerial applications of copper fungicide from 1966 (van der Pas *et al.* 1984a). Excellent control was achieved in the late 1960s by both single and double aerial applications of copper oxychloride in unthinned, highly diseased stands (Gilmour & Noorderhaven 1971, 1973), and Whyte (1976) demonstrated growth response to spraying at these sites. Van der Pas *et al.* (1984a) could not replicate those results during the early 1980s at four sites throughout New Zealand where pruning and thinning were carried out to prescription. Basal area was increased by spraying only after one or more years of successful disease control, but the response was of no practical value as differences between sprayed and unsprayed stands were eliminated by the final thin-to-waste.

In 1985, five spray trials were established throughout New Zealand to study disease progress in unsprayed blocks to end of rotation. Plots were also established in neighbouring blocks which were sprayed to management criteria. At four sites, disease levels have not increased significantly despite absence of spraying, and no difference in growth between sprayed and unsprayed sites could be demonstrated. The remaining site in the central North Island was the only one to show differences in growth and *Dothistroma* levels. This site was unpruned and stocked to about 900 s/ha at age nine. Disease levels in the unsprayed stand were 42% in 1989, and rose to 59% and 63% in 1990 and 1991, respectively. Disease levels in the sprayed

Dothistroma.

stand for those years were 38%, 29%, and 13%. In 1992, levels dropped in both treatments to 15% and 8% for the unsprayed and sprayed stands, respectively. Both treatments were thinned-to-waste to 300 s/ha during 1991/1992. The thinning and a drier-than-usual summer was responsible for the drop in disease levels. Diameter increments from 1989 to 1992 were 59 mm and 69 mm for the unsprayed and sprayed stands, and it is expected that a carry-over effect of the high levels in 1990 and 1991 will result in an increased growth difference between treatments in 1993.

It appears that significant growth responses to spraying may only be expected in highly stocked stands, or sites where the microclimate is favourable to the disease, e.g. unpruned stands, gullies, and sheltered humid sites. At drier sites, where the trees are pruned and thinning takes place before crown closure, spraying may only be necessary after extreme conditions, i.e. an exceptionally wet summer. It has also been recommended that spraying be delayed if a stand is due for pruning that same year (Kershaw *et al.* 1988).

References

Bassett, C. 1972: The *Dothistroma* situation. Farm Forestry 14: 47-52.

Bulman, L.S. 1988: Incidence and Severity of Cyclaneusma needle-cast in fifteen *Pinus radiata* plantations in New Zealand. New Zealand Journal of Forestry Science 18: 92-100.

Gadgil, P.D. 1984: Cyclaneusma (Naemacyclus) needle-cast of *Pinus radiata* in New Zealand. 1: Biology of *Cyclaneusma minus*. New Zealand Journal of Forestry Science 14: 179-96.

Gilmour, J.W. 1967: Distribution and significance of the needle blight of pines caused by *Dothistroma pini* in New Zealand. Plant Disease Reporter 51: 727-300.

Gilmour, J.W., G.J. Leggat, J. Fitzpatrick, 1973: Operational control of Dothistroma needle blight in radiata pine plantations in New Zealand 1966-73. Proceedings 26th New Zealand Weed and Pest Control Conference: 130-138. (N.Z. Forest Service Reprint No. 680).

Gilmour, J.W.; A. Noorderhaven, 1971: Influence of time of application

of cuprous oxide on control of Dothistroma needle blight. New Zealand Journal of Forestry Science 1: 160-66.
Gilmour, J.W.; A. Noorderhaven, 1973: Control of Dothistroma needle blight by low volume aerial application of copper fungicides. New Zealand Journal of Forestry Science 3: 120-36.
Hood, I.A.; A.L. Vanner, 1984: Cyclaneusma (Naemacyclus) needle-cast of *Pinus radiata* in New Zealand. 4: Chemical control research. New Zealand Journal of Forestry Science 14: 215-22.
Kershaw, D.J.; P.D. Gadgil, J.W. Ray, J.B. Van der Pas, R.G. Blair, 1988: Assessment and Control of Dothistroma needle blight. Second revised edition. New Zealand Forest Service, FRI Bulletin No. 18.
Van der Pas, J.B. 1981: Reduced early growth of *Pinus radiata* caused by *Dothistroma pini*. New Zealand Journal of Forestry Science 11:210-20.
Van der Pas, J.B., L. Bulman, G.P. Horgan, 1984: Estimation and cost benefits of spraying *Dothistroma pini* in tended stands of *Pinus radiata* in New Zealand. New Zealand Journal of Forestry Science 14: 23-40.
Van der Pas, J.B., J.D. Slater-Hayes, P.D. Gadgil, L. Bulman, 1984: Cyclaneusma (Naemacyclus) needle-cast of *Pinus radiata* in New Zealand. 2: Reduction in growth of the host, and its economic implication. New Zealand Journal of Forestry Science 14: 197-209.
Vanner, A.L. 1986: An aerial spray trial to control Cyclaneusma needle-cast on radiata pine. Proceedings of the 39th New Zealand Weed and Pest Control Conference: 106-7.
Whyte, A.D.G. 1976: Spraying pine plantations with fungicides – The manager's dilemma. Forest ecology and Management 1: 7-19.
Zadocks, J.C., R.D. Schein, 1979: "Epidemiology and Plant Disease Management". Oxford University Press, New York.

Upper Mid-Crown Yellowing (UMCY) in *Pinus radiata* forests

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Abstract

Upper Mid-Crown Yellowing is a condition of radiata pine in which needles in the sub-apical zone of the upper crown become yellow tipped with age, needle retention is low, and crown dieback occurs.

Many factors have been suggested as possible causes of UMCY; however, ecological, physiological, and chemical evidence suggests that a nutritional imbalance involving magnesium and potassium is the most likely cause. Magnesium deficiency in young stands and UMCY in older stands occur because the effective supply of magnesium is too low to meet the needs of radiata pine. Expected changes in the relative supplies of soil Mg and K suggest that the incidence and severity of UMCY is likely to increase in the future; however monitoring of the severity of UMCY on a national scale has been inadequate to confirm this.

The available evidence suggests that deficiency symptoms appear more severely in genotypes with a predisposition to accumulate low amounts of magnesium and high amounts of potassium in their foliage. Radiata pine has an inherently low capacity to accumulate magnesium in its foliage in comparison with other species, and variation within radiata pine is also large. Foliar chemistry data from seedling trees, clones, and radiata families show that within-stand variation in foliar Mg arises from genetic differences in tree nutritional characteristics. Based on the evidence linking UMCY to nutritional traits, the heritability of UMCY is likely to be high but family differences in UMCY (narrow sense heritability) need to be determined.

Research is under way to survey the incidence and severity of UMCY, and its association with site and management factors. The effects of UMCY on individual tree and stand growth and yield are being determined, to assess the cost of UMCY. Trials are being established to determine if UMCY can be economically treated by fertilisation with magnesium. The heritability of UMCY, and the extent breeding for tolerance to UMCY impacts on growth and form are being investigated. Soil tests to predict Mg deficiency, and plant tests to screen for tolerance to UMCY are being examined.

Introduction

Typified by yellow needles in the upper crown followed by needle loss and various degrees of crown dieback (Beets *et al.* 1991), UMCY is only easily visible from above the canopy of New Zealand's older *Pinus radiata* D. Don stands. UMCY is difficult to observe from the ground because the affected zone is easily masked by healthy branches lower in the crown, but UMCY has been increasingly recorded by Forest Health Observers following adoption of aerial surveillance methods in 1982 (P. Gadgil, pers. comm.). Concern about UMCY subsequently increased, and a major effort is being initiated to identify the causes of UMCY and to find solutions.

Reports by forest health officers indicate that UMCY is widespread throughout New Zealand, with the possible exception of Northland. Many reasons have been suggested, singly or in combination, to explain UMCY, among these being physical damage

An example of severe Upper Mid-Crown Yellowing in *Pinus radiata*, New Zealand.

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