

ARMILLARIA ROOT DISEASE IN NEW ZEALAND FORESTS

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ABSTRACT

The Armillaria root disease caused by *A. novae-zelandiae* (Stevenson) Herink and *A. limonea* (Stevenson) Boesewinkel continues to affect plantation forestry in New Zealand. Although the land area being converted from indigenous forest to pine plantation is declining, limited evidence demonstrates that stands already planted on such sites may harbour non-lethal, chronic infection throughout the rotation, with a consequent reduction in growth yield. There are indications that chronic infection may persist in certain second-rotation stands, although the total area of forest infected by *Armillaria* species is unknown. Effective control and management of this disease must be based on a sound knowledge of its development during the course of the rotation.

Keywords: plantation; disease control; root rot; second-rotation; stump removal; thinning; disease resistance; inoculum; spores; rhizomorphs; *Armillaria novae-zelandiae*; *Armillaria limonea*; *Pinus radiata*.

INTRODUCTION

Fungi in the genus *Armillaria* are responsible for a major root disease in plantations of exotic tree species throughout New Zealand (Fig. 1). They have a wide host range (Gilmour 1966; Dingley 1969; van der Pas *et al.* 1983) and cause an unknown, but probably significant number of deaths each year in parks and home gardens. The disease also occurs in fruit trees (Atkinson 1971), and in recent years there has been serious mortality in orchards of kiwifruit (*Actinidia deliciosa* (A. Chev.) Liange et Ferguson) in the Bay of Plenty district, centred on the stumps of newly felled shelter belt trees of *Salix matsudana* Koidz. (Horner 1985, 1987, 1988). In this article past research into the disease in exotic forests is reviewed, and critical areas are identified on which research must focus for the disease to be effectively controlled or managed.

THE PATHOGENS

Two species of *Armillaria* are responsible for most, if not all, of the deaths of trees in exotic forests in New Zealand. They are *A. novae-zelandiae* and *A. limonea* (Shaw & Calderon 1977). These fungi were first formally named by Stevenson (1964), but have been collectively known in this country since 1879 under the incorrectly determined name *A. mellea* (Vahl : Fries) Kummer (Cooke 1879; Colenso 1890; Masee 1898; Farr *et al.* 1919). Although *A. mellea* has been reported occasionally since 1964, there is no basis for assuming that this Northern Hemisphere species has been introduced into New Zealand, and all records of *A. mellea* probably refer to *A. novae-zelandiae* or *A. limonea*. Both species are readily identified during fruiting which normally occurs over

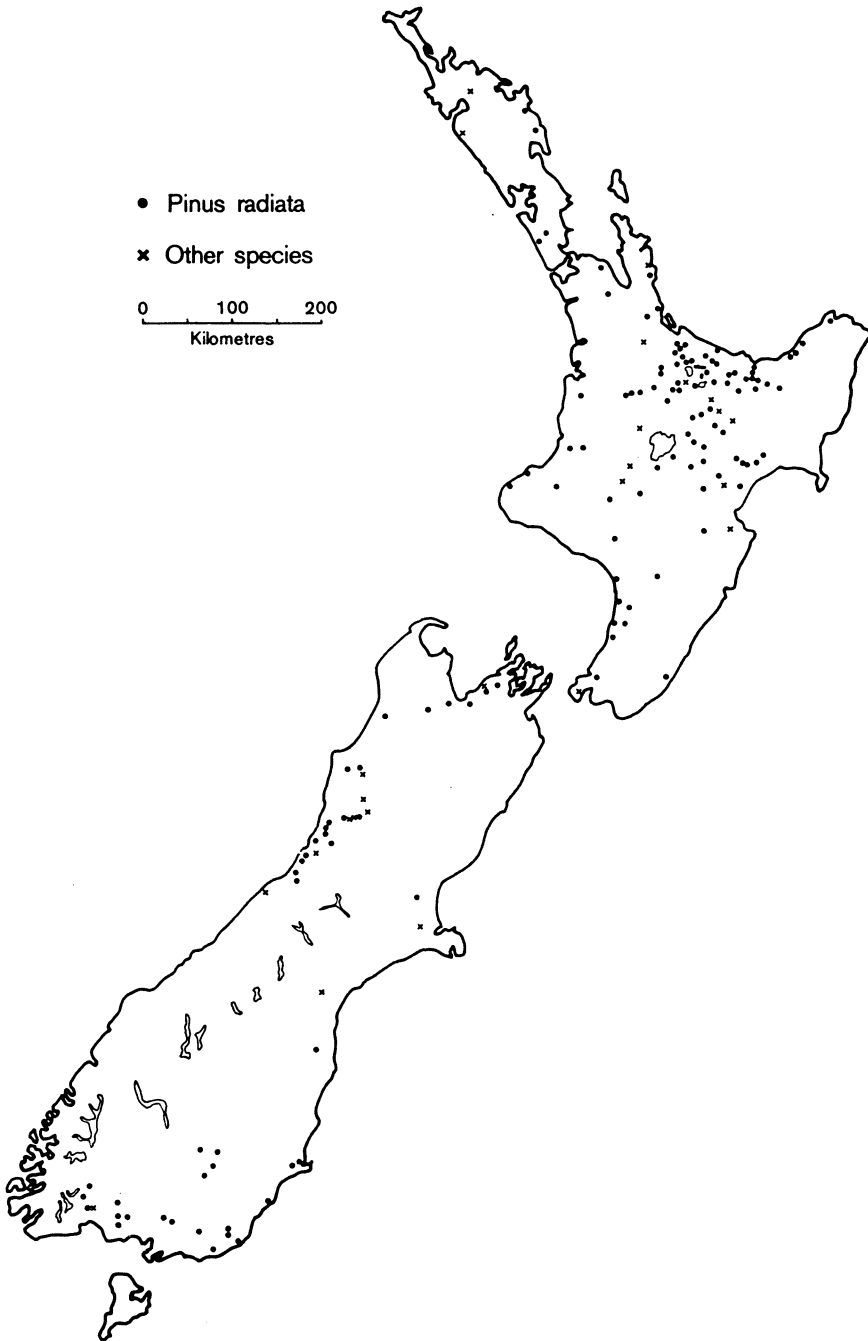


FIG. 1. — Occurrence of *Armillaria* in introduced host tree species in New Zealand. Symbols indicate locations of one or more infected or colonised seedlings or older trees present mainly in plantations or woodlots. (Compiled from miscellaneous New Zealand Forest Research Institute unpublished records since 1950).

several weeks between May and July on decayed wood of indigenous and, to a lesser extent, introduced host species. At other times it is difficult to determine the species unless cultures are isolated. Rhizomorphs of both fungi have dichotomous branching and appear similar (Benjamin 1983; Morrison 1989). Hood & Sandberg (1987) were unable to distinguish rhizomorphs in the field, but according to studies by Benjamin (1983) those of *A. novae-zelandiae* are slightly thicker on average and produced in greater abundance than those of *A. limonea*. Isolates can now be identified in culture with comparative ease (Shaw *et al.* 1981; Benjamin 1983; Hood & Sandberg 1987).

Armillaria novae-zelandiae and *A. limonea* occur naturally in indigenous forests, both podocarp/hardwood and *Nothofagus* spp., where they are important decay fungi of stumps, logs, and dead trees, and the cause of butt rots in living trees (Birch 1937; Gilmour 1954, 1966; Hood & Sandberg 1987; Hood *et al.* 1989; also Campbell 1962). *Armillaria novae-zelandiae* is found throughout New Zealand, having been positively identified in collections from Auckland (Hokianga) to Southland. *Armillaria limonea* is common in the central North Island. It has also been recorded near Kawhia (P.K. Buchanan, pers. comm.) and at Pukekohe (I.J. Horner, pers. comm.) and may occur elsewhere in the country, but this still requires confirmation. *Armillaria limonea* is also reported from Chile and Argentina, while *A. novae-zelandiae* is listed from Chile, Argentina, and Australia (Singer 1969; Horak 1979; Kile & Watling 1983).

Armillaria novae-zelandiae and *A. limonea* are not the only species of *Armillaria* in New Zealand. Fruitbody collections distinct from the two common species have been made from decayed wood in *Nothofagus* forests on the South Island West Coast (near Reefton, probably on *N. fusca* (Hook.f.) Oerst. — Kile & Watling 1983, PDD 36709) and in the central North Island (Tongariro National Park, on *N. solandri* var. *cliffortioides* (Hook.f.) Poole — Kile & Watling 1983; M. MacKenzie, pers. comm.; the northern Kaimanawa Range, on *N. menziesii* (Hook.f.) Oerst. or *N. fusca* — personal observation; and in southern Whirinaki Forest Park, on *N. fusca* — personal observation). The separate nature of these collections is supported by the results of cultural pairing using vegetative field isolates (North Island collections only) which proved incompatible with tester single-spore isolates of *A. novae-zelandiae* and *A. limonea* (unpubl. data). Kile & Watling (1983) identified the South Island collection as *A. hinnulea* Kile et Watling, and suggested that a collection from the North Island belonged to the same species. However, in exploratory cultural pairings single-spore isolates from a North Island collection were incompatible with *A. hinnulea* from Australia (Kile & Watling 1988), and fruitbodies appear to differ in micromorphology (R. Watling, pers. comm.). Further study is therefore needed to resolve whether more than one species additional to *A. novae-zelandiae* and *A. limonea* is present in *Nothofagus* forests, and whether *A. hinnulea* occurs in New Zealand. In addition, Kile & Watling (1983) listed a collection of *Armillaria* from the Waitakere Range, near Auckland, of uncertain species status.

Inoculation experiments have been used to demonstrate that *A. novae-zelandiae* and *A. limonea* are both pathogenic to seedlings of *Pinus radiata* D. Don (Shaw *et al.* 1981; Benjamin & Newhook 1984a). However, their relative pathogenicities have not yet been conclusively determined. Shaw *et al.* (1981) found that isolates of each species varied in their ability to infect and kill seedlings (mortality ranged up to 40%) but, although *A. novae-zelandiae* appeared to be more pathogenic than *A. limonea*, the

difference was not significant. By contrast, Benjamin (1983) found no difference in pathogenicity among three isolates of each species, but *A. novae-zelandiae* killed significantly more seedlings (83%) than *A. limonea* (60%). Despite the apparent contradictions, these studies are not necessarily in disagreement, and indicate that for conclusive results to be obtained inoculation tests must compare an adequate number of isolates of each species. In the field both species have been isolated from *Armillaria*-killed *P. radiata* seedlings, with *A. novae-zelandiae* probably being obtained more frequently than *A. limonea*. MacKenzie & Shaw (1977) found that the incidence of *Armillaria*-caused pine mortality was higher next to stumps with *A. novae-zelandiae* fruitbodies than to those with *A. limonea* fruitbodies, but this result, also, may merely reflect the relative colonising or fruiting ability of the two species. The pathogenicity to *P. radiata* of other species of *Armillaria* in New Zealand is unknown.

DISEASE DEVELOPMENT

First-rotation Pine Stands after Indigenous Forests

Rhizomorphs of *A. novae-zelandiae* and *A. limonea* are common in indigenous forests, where they grow freely through the topsoil or adhere to the surfaces of healthy roots (Hood & Sandberg 1987; Hood *et al.* 1989). They appear unable to parasitise sound roots in podocarp/hardwood forests, and there is no evidence of *Armillaria*-caused mortality among young trees or seedlings in this type of forest. *Armillaria* species decay the roots of old-growth trees of rimu (*Dacrydium cupressinum* Lamb.) and matai (*Prumnopitys taxifolia* (D. Don) Laubenf.) in the central North Island, but it is not clear whether they invade healthy root tissue directly, or merely colonise already dead or weakened roots (Hood *et al.* 1989). Parasitic attack has been reported in *Nothofagus* forests (Birch 1937; Rawlings 1953; Forest Research Institute 1955 pp. 15–16), and both species have been isolated from specimen trees or saplings of *N. menziesii* at Rotorua (personal observation). In podocarp/hardwood forests rhizomorph production may be prolific. Hood & Sandberg (1987) reported that the average aggregate rhizomorph length, down to a depth of 22 cm beneath a forest logged of podocarps in the Bay of Plenty district, ranged from 2 to 9 m/m² of soil surface.

Inoculum levels appear to be temporarily reduced when indigenous forests are clearfelled and burned in preparation for planting in *P. radiata*. Hood & Sandberg (1989) found that the percentage of soil rhizomorphs yielding isolates of *Armillaria* fell from 41–89% down to 5–14% after clearing and burning, and suggested that this apparent reduction in rhizomorph viability was caused by heat from the fire. However, site recolonisation is rapid. Mycelial fans were observed above ground-level in a new stump of *Beilschmiedia tawa* (A. Cunn.) Kirk 1 year after felling (personal observation). Fruitbodies of *Armillaria* species become plentiful on new stumps within 2 years of burning, and rhizomorphs are common in the topsoil. MacKenzie & Shaw (1977) found that new stumps become colonised by either or both of the two common *Armillaria* species. It is not known whether stumps are colonised primarily from residual inoculum after burning, or whether some inoculum is introduced by means of airborne spores. The ability of spores to colonise billets of wood has been demonstrated experimentally (Hood & Sandberg 1987), and a role for spores is suggested by the high

density of colonies (clones or genotypes) observed in both indigenous and exotic forests. Colony numbers in a podocarp/hardwood forest were estimated at 19–93 *A. novae-zelandiae* colonies/ha and 15–56 *A. limonea* colonies/ha (0.1-ha plots, approximately — Hood & Sandberg 1987). Benjamin & Newhook (1984b; Benjamin 1983) found numerous colonies (genotypes) of *A. novae-zelandiae* (equivalent to 32 colonies/ha) attacking 4- to 6-year-old *P. radiata* trees planted in three, square, 0.25-ha plots on a native cutover site near Rotorua. Fewer colonies of *A. limonea* were present in the same plots (3 colonies/ha). In a similar study in three Southland forests (Slopedown, Longwood, and Rowallan Forests) colonies of *A. novae-zelandiae* pathogenic to 8- to 16-year-old *P. radiata* trees were densely distributed on sites previously covered in podocarp/hardwood or *Nothofagus menziesii* forests (13–80 colonies/ha in four plots of approximately 0.1 ha each — I.A. Hood, C.J. Sandberg, and R.F. Thum unpubl. data). *Armillaria novae-zelandiae* was the only species identified on these sites (culturally, and by the occurrence of fruitbodies), but the sampling method used was not designed to detect the presence of possible saprobic *Armillaria* colonies.

Mortality of pine seedlings commences 3 to 6 months after planting on cleared, indigenous cutover sites (Forest Research Institute 1973 pp. 32–3; MacKenzie & Shaw 1977), and continues at an increasing rate during the first 3–5 years, thereafter declining (Forest Research Institute 1962 p. 32, 1974 p. 49; Roth *et al.* 1979; van der Pas 1981a). Mortality is associated with the distribution of stumps and results in the formation of stand gaps (mortality centres or disease patches — MacKenzie & Shaw 1977; Roth *et al.* 1979; van der Pas 1981b). It is not known whether infection is transmitted mainly by means of rhizomorphs growing through soil from stump roots, or whether direct contact with colonised stump roots is also important. Soil rhizomorphs are plentiful on planted, indigenous cutover sites (van der Pas & Hood 1984) and must considerably increase the chances for infection to occur. In inoculation experiments it was found that infection of *P. radiata* (Benjamin 1983) and *Eucalyptus* seedlings (Benjamin & Newhook 1984a) almost always began at the root collar, and resulted from rhizomorph contacts in all trials.

MacKenzie & Shaw (1977) suggested that mortality centres would continue to expand steadily with time. However, Roth *et al.* (1979) postulated that disease patches do not enlarge during the first 3 years, but intensify as more pine seedlings become infected and die. These authors did observe a wave of peak mortality to move out within the patch area over a 3-year period, which created an illusion of expansion. It was suggested that infection of pines further from the stump centre was delayed until pine root systems had grown sufficiently to contact the smaller roots and growing rhizomorphs at the extremities of the stump root system. Van der Pas (1981b; van der Pas & Hood 1984) found no evidence of an outward movement of mortality with time, but did support the postulate of Roth *et al.* (1979). He found that mortality rates over the first 4 years conformed to van der Plank's (1963) model for primary infection (that derived directly from stump roots only), thereby excluding the possibility of pine-to-pine root spread (secondary infection) and consequent patch enlargement. However, he noted that as pine root systems increase in size and begin to overlap with each other, the possibility of pine-to-pine disease transfer may permit the spread of infection beyond the area of the original primary source. It should be noted that the data of van

der Pas (1981b) can be fitted with acceptable significance ($p < 0.01$) to both van der Plank models, so conclusions based on this method should be viewed cautiously.

Several studies have attempted to determine whether mortality in planted pines is related to host stump species. MacKenzie & Shaw (1977) found that although stumps of at least seven out of 10 indigenous hardwood tree species became colonised naturally by *Armillaria* species after burning, spatial distribution of mortality appeared to be related only to the positions of *B. tawa* stumps. In addition, Shaw & Calderon (1977) reported that mortality in the first 2 years tended to be greater on sites formerly stocked with a higher component of *B. tawa*. However, van der Pas (1981b) found no statistical difference in association of pine mortality with either *B. tawa* or non-*B. tawa* hardwood stumps. Benjamin & Newhook (1984a) inoculated wood segments in order to examine the ability of different tree species to act as food bases for *Armillaria* species. *Beilschmiedia tawa* was readily colonised by both species, as was the wood of a number of other indigenous and exotic tree species. Rhizomorphs of both *Armillaria* species were produced in greater abundance on wood of *B. tawa* than on other host woods tested, but all tree species were effective as substrates for either *Armillaria* species to infect seedlings of *P. radiata*. Benjamin (1983) suggested that stump species may influence disease development not only through ability to promote infection, but also in the length of time the wood of different hosts is able to persist as a viable inoculum source in the soil. Shaw *et al.* (1976) found that *Cortaderia fulvida* (Buchanan) Zotov, a common, indigenous, graminaceous, weed species on newly planted sites, is susceptible to infection and may play a minor role in transmitting the disease. Presumably other indigenous and introduced *Cortaderia* species can act in the same way.

Mortality of infected trees continues for at least 10 years at reduced rates (Forest Research Institute 1962 p. 32; Shaw & Calderon 1977). Decline in mortality rate probably results from an increase in host resistance (greater vigour with age?) and a reduction in inoculum potential as the food base is depleted. However, infection persists throughout the rotation in a non-lethal, chronic form. In a 10-year-old *P. radiata* stand thinned to its final stocking density, 15% of crop trees were more than 65% girdled at the root collar by *Armillaria* species (Shaw & Toes 1977). Such infected trees may be uprooted or may break off near ground level (Forest Research Institute 1962 p. 32; Gilmour 1966; Shaw & Calderon 1977). MacKenzie (1987) examined the same stand as Shaw & Toes (1977) and found root collar infection still plentiful 9 years later. However, although the percentage of living trees with detectable infection present had dropped only slightly (7%) to 53% in the intervening period, root collar infections were no longer visible at age 19 years on approximately one-third of the trees infected at age 10 years. Instead, another group of trees had become newly infected at the root collar since the earlier evaluation. MacKenzie (1987) suggested that tree recovery may have occurred as a result of increased vigour after the final thinning. Similar recovery from infection by older pine trees was reported previously (Forest Research Institute 1962 p. 32) and may be only partial, in that roots hidden deeper in the soil may still be diseased. These observations do not necessarily mean that slower growing, less vigorous trees are therefore more likely to become infected than dominant or subdominant trees (MacKenzie 1987). The dynamic behaviour of infection in older stands raises the possibility that healthy trees may become diseased

after a thinning operation, if the stumps created are capable of acting as inoculum. MacKenzie (1987) observed that mortality gaps were not sharply demarcated in this older stand owing to the growth of surviving trees within disease centres and the thinning of trees elsewhere in the stand.

Second-rotation Pine Stands

To what degree does infection persist when diseased pine plantations are clearfelled and the sites replanted in *P. radiata*? Gaps in our knowledge about the development of *Armillaria* in second-rotation forests are becoming apparent as an increasing proportion of new plantings is established on land previously stocked in exotic species. Records of infection in second-rotation stands date back to the 1940s. In Whakarewarewa Forest up to 3% of 2- to 3-year-old pines were killed after regenerating naturally after the clearfelling of a stand of *P. radiata* (Lysaght 1944). A later report from the same forest stated that more than one-third of 6-year-old pines were attacked by *Armillaria* on a site previously stocked with eucalypts (Forest Research Institute 1954 p. 12). Fenton (1951) found *Armillaria* in naturally regenerated *P. radiata* after a disastrous fire that destroyed 14- to 18-year-old pine plantations north of Taupo in 1946.

The occurrence of disease in second-rotation stands is not confined to sites cleared of indigenous forest prior to the first planting. Observations indicate that infection can become established over successive rotations on previously forest-free areas (Gilmour 1966). It is not known how extensive such stands are, or whether disease development differs from that on sites originally covered in indigenous forest. Examples of the disease in stands or individual trees established on non-forested land include: *Salix* protection belts near the Ngaruroro River, Napier (Forest Research Institute 1955 p. 16), and in the lower Waimakariri Valley near Christchurch (unpubl. data); second-rotation pine stands in Waitarere and Santoft Forests established on coastal sand dunes (M.A. Stoodley unpubl. data); and stands in Kaingaroa Forest.

Armillaria root disease in Kaingaroa Forest was first studied comprehensively by Gilmour (1954), who surveyed over 40 000 ha of 20- to 28-year-old first-crop *P. radiata* stands. He concluded that parasitic attack in trees dying after infestation by the wood wasp, *Sirex noctilio* Fabricius, was virtually non-existent, but he found *Armillaria* to be widespread as a saprobe in stumps and dead trees. Gilmour assumed that inoculum had been introduced in the form of airborne spores, since the previous cover was mostly either a natural scrub composed of *Leptospermum scoparium* J.R. et G. Forst. and *Pteridium esculentum* (Forst.f.) Cockayne (better sites) or a low, shrub-grassland vegetation dominated by *Dracophyllum subulatum* Hook.f. and *Poa cita* Edgar (poorer sites). Subsequently, *Armillaria* was found colonising over one-third of 6- to 14-year-old dead or nearly dead stumps from thinnings in 50-year-old (approximately) stands of *Pseudotsuga menziesii* (Mirb.) Franco in Kaingaroa Forest (R. van Boven unpubl. data). In 1962 severe attack by *Armillaria* was found in 80% of the crop trees after the first thinning of a 7-year-old second-rotation stand of *P. radiata* (Forest Research Institute 1963 p. 48). Unexpectedly, the first-rotation *P. radiata* stumps were acting as an infection source over a prolonged period. In 1972 *Armillaria* was reported

at a site in northern Kaingaroa Forest killing 50-year-old *P. ponderosa* P. et C. Lawson trees debilitated by repeated *Dothistroma pini* Hulbary defoliation (unpubl. data). Attack appeared to develop from colonised stumps from thinnings. Mortality from *Armillaria* in the next rotation (*P. radiata*) was not high on this site (5% at age 4 years — Shaw & Calderon 1977). However, on an adjacent site, also previously stocked with *P. ponderosa*, mortality was ongoing in a 6-year-old replacement stand of *P. radiata* (van der Pas 1981a). Deaths in standing trees then declined (as in plantations after indigenous forest cover), but windthrow associated with chronic infection was still occurring in the second-rotation stand after age 10 years.

Mortality was also severe in young *P. radiata* stands at Karioi Forest planted on sites previously stocked in *P. ponderosa*, *P. nigra* ssp. *laricio* (Poir.) Maire, or *P. contorta* Douglas ex Loudon (van der Pas 1981a). This time, although the original inoculum may have been derived from former podocarp/hardwood forest, the disease developed late in the first rotation in a similar way to that in Kaingaroa, by colonising trees in stands stressed by tight stocking and wind damage, or treated by poison thinning about 20 years before clearfelling. Roots of living, first-crop trees became infected without showing crown symptoms, and stumps of these trees acted as inoculum sources after clearfelling and replanting. Extensive mortality developed in young second-rotation *P. radiata* stands on these sites, but the death rate in standing trees slowly decreased after the first 3 years, as is typical of stands planted on native cutover sites. However, windthrow associated with chronic infection appears to be common in old second-rotation stands in this forest (M. MacKenzie, pers. comm.).

More recently (since 1984) there have been reports of low to moderate levels of mortality in *P. radiata* stands planted on sites previously stocked with *P. ponderosa*, *P. nigra*, or *P. contorta* elsewhere in Kaingaroa Forest, and in Tairua, Erua, and Taurewa Forests in the North Island (J.D. Hayes, C.A. Scott, M.A. Stoodley unpubl. data). However, although early second-rotation *Armillaria*-caused mortality may be locally serious, these examples appear to be special cases associated with particular previous-crop *Pinus* species on certain sites (such mortality has not always occurred in young pine stands replacing these particular species). Since 1984 there have been several other reports of mortality in younger (under 5 years) *P. radiata* stands following *P. radiata* (Kaingaroa Forest), *Ps. menziesii* (Kaingaroa Forest), and *Larix decidua* Mill. (Pomahaka Forest, Tapanui, Southland — J.D. Hayes, R.F. Thum unpubl. data) but, in general, standing deaths in most young second-rotation plantings appear to be scattered and of minor significance. Even so, the discovery of apparently extensive chronic infection in second-rotation stands in Kaingaroa Forest has been disturbing. In a recent study, M. MacKenzie (unpubl. data) found variable levels of infection in 27 out of 29 stands of 5- to 11-year-old *P. radiata* trees on sites previously stocked in *P. radiata*, *P. ponderosa*, *P. contorta*, *P. nigra*, or *Ps. menziesii*. Infected trees appeared to be clustered, but it is not known how the disease is developing in these stands. Chronic *Armillaria* infection was previously reported in 7-year-old natural regeneration of *P. radiata* in Whakarewarewa Forest where 23% of living trees were found to be infected at the root collar (Forest Research Institute 1955 p. 16), but it is unclear how widespread such infection is in New Zealand forests, and how it will develop in subsequent rotations.

DISEASE IMPACT

The establishment of *P. radiata* forests in New Zealand occurred in two planting waves, from 1922 to 1937 and from about 1970 virtually to the present. Interest in *Armillaria* root disease began during the first period and intensified during the second. Mortality was first observed in 1930 in young (4-year-old) pine plantations established on sites cleared of podocarp/hardwood forest (New Zealand State Forest Service 1933; Birch 1937). Occurrence was then only sporadic and Birch suggested that *Armillaria* would probably have minimal effect on the establishment of exotic forests if care was taken with site selection and planting technique. Subsequent reports, however, indicated that the disease was more significant than first realised (Hocking & Mayfield 1939; Jolliffe 1941; Fenton 1951; Forest Research Institute 1952–57, 1961–63; Newhook 1964). It was estimated that a level of 20% infection was present in *Pinus elliotii* Engelm. planted on indigenous cutover sites in Tairua Forest (Forest Research Institute 1956 p. 18). Significant mortality was reported among young plantings of other exotic species, including *P. radiata*, on similar sites on the South Island West Coast (Forest Research Institute 1957 pp. 15–16) and at Pureora (Forest Research Institute 1961 p. 31). According to the Forest Research Institute (1963 p. 48) "... reports of severe infection by this root rot have again focused attention on this ever present pathogen and highlighted our lack of knowledge about it...". A summary of knowledge up to the mid 1960s was provided by Gilmour (1966).

By the 1970s the extent of the disease was such that it became necessary to initiate a full-scale research programme. It was estimated that somewhere between 50 000 and 60 000 ha of land formerly covered in native forest was affected or potentially affected by this disease (Shaw & Calderon 1977; van der Pas 1981a). Evaluation of disease levels on these sites revealed severe mortality losses in the first few years after planting. A.E. Beveridge (Forest Research Institute 1974 p. 52) found mortality levels ranging from 20 to 30% after 5 years in a *P. radiata* plantation established on an indigenous forest cutover site on the Mamaku Plateau. In another Mamaku study (Shaw & Calderon 1977), *P. radiata* mortality from *Armillaria* ranged from 11 to 27% after 2 years on sites previously stocked in residual podocarp/hardwood forest of varying composition. At Pureora, levels of *P. radiata* mortality on several former podocarp/hardwood sites were variously recorded as at least 36% after 6 years, 33% after 2 years (Shaw & Calderon 1977), and over 50% after 6 years (van der Pas 1981a). Van der Pas (1981a) estimated that the over-all mortality on former indigenous forest sites was about 5% (presumably after 5 years), whereas on former non-wooded sites, where infection was scattered, mortality was considered to be less than 0.1%. All reports quoted observed that mortality was spatially uneven in distribution. It was suggested that stand mortality gaps led to a reduction in stem quality because of excessive branch growth in clearings (Shaw & Calderon 1977). Open areas also became filled with shrub hardwood weed growth that hindered tending operations.

Armillaria caused severe mortality in young plantations of *P. radiata* in Westland (Forest Research Institute 1957 pp. 15–16) and Southland established on land previously covered in podocarp/hardwood or *Nothofagus* forest (Fig. 1). On a former *Nothofagus* site in Westland, Shaw & Calderon (1977) found 5% mortality in a stand of *P. radiata* after 2 years, which they felt to be unexpectedly low. The initial period of *Armillaria*-caused mortality appears to be prolonged in some Southland stands (e.g., in

Rowallan Forest), and deaths of standing final-crop trees (stocked at less than 250 stems/ha) have been reported to be ongoing beyond the age of 10 years (R.F. Thum pers. comm.). Climate or soil conditions may be less favourable to the host species in these areas than elsewhere.

It is now known that significant growth loss occurs on trees with chronic infection later in the rotation period. Incremental measurements were made by Shaw & Toes (1977) in a 10-year-old stand of *P. radiata* trees just thinned to final stocking intensity. They found that trees with more than 65% of the root collar girdled by *Armillaria* infection (15% of final-crop trees) had increased their dbh after 1 year by 14–24% less than had uninfected trees of equivalent dbh, height, and crown depth at the start of the measurement period. Trees in another stand heavily infected by both *Armillaria* and *D. pini* showed a reduction in growth rate greater than the sum of the losses attributed to heavy infection by each fungus alone.

There have been two attempts to quantify growth loss due to *Armillaria* in first-rotation pine stands established on podocarp/hardwood forest cutover sites. Shaw & Calderon (1977) estimated volume losses ranging from 29 to 32% for 15- to 21-year pulpwood rotations, and a 26-year sawlog regime. These were calculated assuming an estimated 25–30% loss of land area occupied by mortality patches, and a diameter growth reduction of 14% on 15% of crop trees older than 10 years (based on the study described in the previous paragraph). MacKenzie (1987) considered that this value was over-estimated. He found evidence for increased growth rates on trees recovering from chronic infection later in the rotation. In addition, he felt that the losses in productive land area early in the rotation were given undue weight by Shaw & Calderon (1977), and that early mortality really amounted to a biological thinning, being compensated for by increased growth on released residual trees. Losses in infected crop trees due to windthrow were considered to be of greater significance, because residual tree spacings late in the rotation were such that increased increment growth could no longer compensate for loss. He estimated a loss of 6–13% of potential volume for a 28-year sawlog regime due to *Armillaria*-caused windthrow and growth reduction (a total loss of 32–72 m³/ha from a projected 571 m³/ha final volume without disease). Since canopy gaps occupied by indigenous hardwood shrubs do still occur in many older first-rotation pine stands established on former native forest sites (e.g., at Pureora, Mamaku Plateau —F.H. Crockett, pers. comm.), it seems likely that actual stand losses lie somewhere between the estimates of Shaw & Calderon on the one hand, and MacKenzie, on the other. No attempts have been made to quantify growth loss on a rotation basis for infected stands in Southland and Westland.

Second-rotation stands of *P. radiata* with severe mortality early in the rotation are estimated to occupy about 1000 ha, mainly in Karioi and Kaingaroa Forests (van der Pas 1981a). At Karioi Forest, mortality reached 11% in 5- to 6-year-old stands, but over-all second-rotation mortality was estimated at only 5%. However, MacKenzie (pers. comm.) has found that chronic infection in Karioi second-rotation stands can be 60–70% of the final stocking. MacKenzie also found widespread chronic infection in second-rotation *P. radiata* stands in Kaingaroa Forest. This suggests that *Armillaria* may be more serious than was hitherto accepted. Many first-rotation plantations on former native forest sites, and some second-rotation stands, may be suffering growth loss as a result of *Armillaria* infection, even though crowns appear healthy. Evidence

of *Armillaria* on the roots and root collars of living trees standing or uprooted in recent storms has been recorded in several central North Island forests (F.H. Crockett, J.D. Hayes, A. Zandvoort unpubl. data). However, the actual extent of chronic *Armillaria* infection in New Zealand pine forests is unknown.

PROSPECTS FOR CONTROL AND MANAGEMENT

Research into the control of *Armillaria* root disease in New Zealand forests began in the 1970s, and has continued to the present day (Forest Research Institute 1976, 1988 p. 14). The only proven eradicated control so far demonstrated has been that of removing the stumps after clearfelling and burning the native forest, before planting the new crop (Forest Research Institute 1974 p. 49). In a trial at Pureora Forest, *Armillaria*-caused mortality of pine seedlings was 33% after 2 years and 52% after 5 years on a conventionally treated site set aside as the experimental control (Shaw & Calderon 1977; van der Pas 1981a). However, on adjacent sites in which most stumps had been pushed out of the ground using a bulldozer blade (some sites also being windrowed, or windrowed and ground-cultivated by discing or root ripping) mortality levels were 12–21% after 5 years. Windrowing and ground cultivation appeared to offer no additional disease control over that already achieved by stump removal (J.B. van der Pas unpubl. data). In another trial in the eastern Bay of Plenty, thorough removal of stumps and clearing of debris into windrows reduced mortality to only 2% after 4 years, compared with 23% in conventionally treated plots (van der Pas & Hood 1984). This treatment also significantly reduced the abundance and frequency of soil rhizomorphs. MacKenzie (unpubl. data) has had similar success in reducing mortality among young, second-rotation pine stands at Karioi Forest by removing stumps of infected first-crop pine stands before replanting.

Despite its proven effectiveness, stumping is not widely practised as a control method. It is an expensive operation which cannot be conducted in forests on steeper slopes. It is also not known how changes to the soil quality (removal of topsoil, ground compaction) influence growth later in the rotation (van der Pas & Hood 1984). Some forest owners employ ground cultivation, such as "ripping" or "V-blading", which disturbs roots without shifting the larger stumps. These operations improve planting efficiency, reduce early frost losses, and probably enhance establishment and growth rates at the start of the rotation period. They may also give some measure of *Armillaria* control, of mortality at least, but this remains untested. Shaw & Calderon (1977) attempted to calculate the permissible cost limits for pre-planting ground preparation by stumping, up to which the anticipated improved yields due to *Armillaria* control would still return a net profit at the end of the rotation. As they noted, however, these calculations are hampered because the actual effectiveness of ground treatment in increasing yields over a full rotation period is unknown. In part, it depends on a precise knowledge of when in the rotation losses from standing mortality or windthrow are no longer compensated for by increased growth of the released residual trees, many of which are growing at reduced rates because of chronic infection (MacKenzie 1987). MacKenzie assumed this cross-over point to occur somewhere between ages 10 and 18 years in a 28-year rotation, whereas Shaw & Calderon (1977) believed it to occur earlier (in which case the anticipated greater returns from control of *Armillaria* were more likely to justify the treatment cost). From his observations of older infected

stands, MacKenzie felt justified in assuming that the area of productive ground lost to *Armillaria* was much less than the 20–30% assumed by Shaw & Calderon, presumably because surviving trees must continue to grow with age and re-occupy the early disease patch areas.

From the studies described earlier there is no basis for assuming that removing stumps of selected indigenous tree species, only, would give equivalent control in first-rotation stands to that achieved by complete stumping of the whole area. On the other hand, it may be possible to restrict ground preparation treatment to sites of higher disease potential identified by the composition of the indigenous forest cover (Shaw & Calderon 1977). In second-rotation stands ground treatment prior to planting may prove to be more cost effective because of the smaller size of previous crop stumps than is found after clearing indigenous forest. On level ground where severe *Armillaria* infection in the next crop was anticipated, short rotations followed by an alternative form of mechanical root system extraction might be a serious option (e.g., tipping over whole trees at harvest, or vibration stump pulling, possibly also using the stump wood fibre in pulping for extra financial returns — Roth *et al.* 1979; Arnold 1981).

Stump removal would not be necessary at all if colonisation by *Armillaria* of the newly created stumps could be prevented. There has been one attempt in New Zealand forests at using chemicals to poison new stumps and so render them toxic to invading mycelia of *Armillaria*. Van der Pas poured a commercial hydrocarbon mixture containing methyl isothiocyanate into holes drilled into indigenous stumps shortly after burning (van der Pas & Hood 1984). Mortality of young pines 4 years later showed a significant reduction (9% as compared with 23% in untreated plots), although the mechanism of apparent control is not clear since frequencies and quantities of adjacent soil rhizomorphs were not reduced significantly. Chemicals have also been applied to the soil without achieving any effective control. Shaw *et al.* (1980) added sodium pentachlorophenate and/or pentachlorophenol to container-grown *P. radiata* seedlings, either by mixing the chemicals with the soil during potting, or by applying them to the soil surface. These chemicals gave no protection against artificially applied inocula of *A. novae-zelandiae* or *A. limonea*. Heavy liming of the soil surface appeared to cause some reduction in natural field mortality of 4-year-old *P. radiata*, but rhizomorph frequencies and amounts were not reduced, and soil pH values were not altered, making it unclear how this treatment was acting (van der Pas & Hood 1984).

An alternative stump treatment may be to use a biological control method. Inoculating stumps with a saprobic decay fungus immediately after felling may prevent colonisation by *Armillaria* species. Use of a chemical such as ammonium sulphamate in conjunction with a biological agent may also be beneficial (Rishbeth 1976, 1979). The chosen fungus may act by rapidly depleting food reserves, making them unavailable to its competitor, or by preventing the entry of the pathogen, or else restricting it to small segments of the stump where it can no longer be active beyond a limited time period (Rishbeth 1976, 1979). Saprobian *Armillaria* isolates might be possible contenders among a number of naturally occurring decay fungi for use as potential biological agents. Chemical or biological control methods may be more effective in second-rotation stands, where treatment of first-crop clearfelled and second-rotation thinning

stumps may achieve more complete penetration. It may be important, however, to determine whether stumps are colonised primarily from soil or by means of airborne inoculum.

Other options, besides eradicating the inoculum or depriving it of its food base, involve managing the stand so that *Armillaria*, although present, has minimal impact. In the past, planting at a greater density was not encouraged. It was considered that *Armillaria* would eliminate the majority of trees in the mortality centres, while uninfected areas would carry undesirably high stocking densities. However, MacKenzie (pers. comm.) considered that at Karioi Forest high density operational planting (2400 stems/ha was an effective management method because soil inoculum was widespread and uniform in distribution. Enough trees survived to an age where they became more tolerant of infection, giving rise to stands of acceptable stocking intensities over the whole site. Beveridge *et al.* (1973) recommended a short-rotation sawlog regime combined with grazing on badly infected sites previously covered in podocarp/hardwood forest. Returns from grazing in mortality gaps would help recoup losses from *Armillaria* attack, and at the same time weed growth would be controlled, facilitating stand tending operations. Because these methods do not remove *Armillaria* from the site, inoculum may be transferred to the next rotation, perpetuating the problem. Fallowing the land after clearing a forest, by grazing until stump food reserves are well decayed, has been suggested as another management option. In plantations established on farmland originally in native forest, *Armillaria*-caused mortality appears to be absent, or only scattered and insignificant. However, it is not known how long land must be kept clear of trees before inoculum is exhausted.

Use of a resistant tree species is not a realistic option in forest plantations although it should be considered when planting farm or orchard shelter belts or specimen trees on infected sites. It would need a drastic upheaval in the forest industry to replace *P. radiata* as the dominant tree in New Zealand plantations, susceptible as it is to *Armillaria*. Early references to species variation in resistance to natural *Armillaria* infection were made in reports of the Forest Research Institute (1953 p. 20, 1955 p. 16, 1961 p. 31). On sites previously in indigenous forest, the species most susceptible to infection and mortality were *P. radiata*, species of *Larix*, and *Chamaecyparis lawsoniana* (A. Murr.) Parl. (Hocking & Mayfield 1939), while the least susceptible were *Cryptomeria japonica* (Linn. f.) D. Don and *Thuja plicata* D. Don. However, *Armillaria* has recently been found causing butt rot in older *C. japonica* (G. Steward, pers. comm.). *Pseudotsuga menziesii* was found to be as susceptible as *Pinus radiata* in these early field surveys, but showed lower mortality rates. Although species of *Eucalyptus* proved to be susceptible to infection, losses did not appear to be significant (Newhook 1964). Information on species susceptibilities was summarised by Weston (1957) and Gilmour (1966). Current observations in plantations confirm that *Ps. menziesii* and eucalypts are less prone to mortality from *Armillaria* than is *P. radiata*. There appear to be differences in susceptibility among *Eucalyptus* species but field observations are not straightforward. In inoculation studies, Benjamin & Newhook (1984a) found variation among host species in susceptibility to the two common species of *Armillaria*. More seedlings of *P. radiata* became infected than did those of several *Eucalyptus* species and fewer infected *Eucalyptus* seedlings died, possibly because *Eucalyptus* seedlings were more vigorous in growth than were seedlings of *P.*

radiata. *Armillaria novae-zelandiae* appeared to be more virulent to eucalypt seedlings than *A. limonea*, but eucalypts demonstrated evidence of species variation when inoculated with either *Armillaria* species. Using a *B. tawa* inoculum substrate, more seedlings of *E. delegatensis* R.T. Baker and, to a lesser extent, of *E. regnans* F. Muell., were killed by *Armillaria* than were those of *E. fastigata* Deane & Maid. and *E. saligna* Smith. In fact, seedlings of *E. saligna* were almost immune.

Within-species variation in resistance to disease has not been investigated in *P. radiata*. An ability to select for resistant or tolerant seedlots might lead to significantly reduced mortality among young pines and less chronic infection later in the rotation. There appears to be no advantage in using cuttings rather than seedlings on high-risk sites. Klomp & Hong (1985) found significantly higher mortality from *Armillaria* (23%) among rooted cuttings from 7-year-old trees 6 years after planting on a clearfelled indigenous forest site, than among 6-year-old seedlings (14%) which had better developed root systems.

Significant gaps which still exist in our knowledge of the *Armillaria* root disease throughout the rotation hamper the application of effective control measures. A number of questions remain. How does inoculum enter and develop in a stand? Does chronic infection originate predominantly from previous crop inoculum, from new inoculum introduced at the beginning of the rotation period, or from inoculum entering through thinning stumps? The occurrence of infected second-rotation stands in Kaingaroa Forest, some of which do not have a history of extensive early mortality, suggests that some inoculum may enter later in the rotation. How important is pine-to-pine spread of infection? How intensively, and when, should a severely infected stand be thinned to avoid losing final-crop trees? Does thinning increase the level of chronic disease by producing new inoculum through spore infection, and by furnishing existing inoculum with an additional food substrate? If so, is stump size, and therefore timing of thinning important? Or does thinning ensure greater vigour and perhaps improved resistance for final-crop trees (not forgetting that more vigorous root systems may enhance the chances of chronic infection through increased root contact with inoculum — Shaw *et al.* 1988)? Van der Pas (1981b) found that “thinning” (i.e., removing roots as well) between stand ages 2 and 3 years led to a significant reduction in mortality rates (this effect was partly artificial, in that trees were removed before they were recorded as dead). However, once trees are of sufficient size to permit pine-to-pine root contact, the situation may be different. What is the effect of pruning? Recent observations indicate that pruning of trees already stressed by *Armillaria* infection can induce mortality (C.W. Barr, A. Zandvoort pers. comm.). When should infected stands be harvested? Should heavily infected stands with green crowns be continued through to the end of the projected rotation period or clearfelled early? If volume loss through windthrow exceeds the net incremental growth gain over the same period, there is little point in maintaining the stand. The work of Shaw & Toes (1977) indicates that good control of *D. pini* is to be recommended in severely infected stands, if they are not clearfelled early. What is the effect of current management on the level of *Armillaria* in future stands? Will the disease continue to build up on new sites, with more extensive early mortality? How widespread are stands with chronic infection, and how evenly distributed is within-stand infection? How does the forest

manager identify stands with severe chronic infection? When in the rotation is the best time to apply control or management measures?

Although the nature of plantation forests in New Zealand is changing as first-crop stands mature and are replaced, there are indications that *Armillaria* root disease will continue to influence forest management. Only when the development of this disease is fully understood will it be clear how best to approach the problem, and when the appropriate treatment should be applied.

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