Incidence of Armillaria root disease in karri regrowth forest is underestimated by surveys of aboveground symptoms

Richard M. Robinson[1,2], Matthew R. Williams[3] and Robert H. Smith[1]

[1]Science Division, Department of Conservation and Land Management, Manjimup, WA 6258, Australia
[2]Email: richardr@calm.wa.gov.au
[3]Science Division, Department of Conservation and Land Management, Kensington, WA 6151, Australia

Revised manuscript received 24 February 2003

Summary

Intensive survey based on aboveground symptoms of Armillaria root disease underestimated true levels of disease by at least 20% and sometimes by up to 40% in high-quality karri regrowth stands. The results challenge the reliability of surveys based on aboveground disease symptoms. While most disease was established within the subdominant stratum, a very high proportion (30–60%) of the dominant trees were also infected. Within the study areas 15 distinct genotypes of *Armillaria luteobubalina* were identified. Individual genotypes existed as clones, with 2–3 clones per hectare. These factors need to be considered in stand management planning and yield predictions. A broader study, including lower-quality sites, is needed to determine whether these findings apply to all types of karri regrowth.

Keywords: root and butt rots; disease surveys; plant disease control; karri; *Eucalyptus diversicolor*; *Armillaria*; Western Australia

Introduction

Armillaria root disease (ARD) is a problem in regrowth forests throughout the world, causing significant economic loss due to tree mortality, windthrow and timber defect (Hood *et al.* 1991; Kile *et al.* 1991). ARD is relevant to forest management in both the effect the pathogen may have on regrowth stands and the effect intensive forest management may have on the disease.

Six species of *Armillaria* (Fr.:Fr.) Straud occur in Australia (Kile and Watling 1983, 1988). Not all of these species are pathogenic. In eucalypt forests, ARD will normally express itself in the latter stages of the disease with the presence of inverted V-shaped scars or callused lesions at the base of trees, dead and brown-stained bark extending up the stem, mycelial fans under the bark and wet stringy white-rotted wood at the root collar, and the production of basidiomes at the base of infected trees in the autumn (Marks *et al*. 1976; Kile 1981; Robinson 2003).

In the karri (*Eucalyptus diversicolor* F.Muell.) forests of Western Australia (WA), ARD is caused by the endemic species *Armillaria luteobubalina* Watling & Kile (Kile *et al*. 1983). *Armillaria luteobubalina* can act as a primary pathogen (Podger *et al.* 1978), infecting and killing apparently healthy trees. Basidiospores do not appear to be an important vector for the spread of *A. luteobubalina* (Kile 1983). Infection spreads from tree to tree via root contact and the same genotype of *A. luteobubalina* may infect large numbers of trees within a forest stand. Studies concerning the behaviour of the pathogen and its impact on karri and other regrowth eucalypts show that the spread of the disease in eucalypt forests is associated with infected stumps left following logging operations (Edgar *et al.* 1976; Pearce *et al.* 1986; Kellas *et al.* 1987). Survey results suggest that while ARD has only a scattered presence in most regrowth karri stands, it is widespread in a number of high-quality stands (M. Rayner, *pers. comm.*).

Roots may be infected for many years before symptoms are expressed in the trees (Edgar *et al*. 1976). As ground surveys can assess only the aboveground symptoms of the disease, it is very difficult to determine true levels of disease within a stand. In Western Australia, ARD surveys are conducted in regrowth karri prior to commercial thinning operations (Robinson *et al*. 1998) which take place when stands reach a top height of 30 m (CALM 1992). On suitable sites, regrowth karri reaches this height in 15–25 y (Rayner 1991). The surveys are conducted in the autumn, when *A. luteobubalina* fruits, and crews assess infection status on the presence of basal scars and *A. luteobubalina* basidiomes fruiting on infected trees. Cost, on-site conditions and non-fruiting of *A. luteobubalina* prevent such surveys detecting all occurrences of disease within a stand.

The object of the study was to determine (a) whether *Armillaria* infection is restricted to individual trees showing symptoms or is actively spreading to symptomless neighbouring trees, (b) the relationship between levels of infection in trees showing aboveground symptoms and true belowground levels on two different site types within the karri regrowth estate, and (c) the number of *A. luteobubalina* clones on each site. This information is valuable when determining management strategies for ARD.

Methods

Site selection

High-quality karri regrowth stands infested with *A. luteobubalina* were identified from disease survey data (CALM, Forest Management Branch, Manjimup, WA). Site index (based on projected stand dominant height at 50 y of age (Rayner 1991)) and stand composition were used to select sites. Four sites were chosen, two being pure karri with high site indices in Dombakup forest block, and two being stands of mixed karri and marri (*Corymbia calophylla* (Lindl.) K.D.Hill and L.A.S.Johnson) with

Table 1. The site index (SI) and number of trees pulled at each site

Location	Stand age (y)	SI	Number of trees pulled[1]			
			DK	SDK	SDM	Total
Dombakup 8	30	50.2	44	182	0	226
Dombakup 9	29	50.7	48	153	0	201
Warren 2	25	43.4	49	74	85	208
Warren 5	26	48.8	45	84	46	175

[1]DK = dominant karri, SDK = subdominant karri, SDM = subdominant marri

moderate to high site indices in Warren forest block (Table 1). All selected stands were aged between 25 and 30 y and were scheduled for first thinning. The stands had been regenerated either by planting or from seed trees retained following clearfelling and burning.

Site treatment

At each site, six 20 m x 20 m plots were installed. Two plots were centred on infected dominant trees, two on infected subdominant trees and two on non-symptomatic (apparently healthy) dominant trees. Within each plot, all trees were assessed for ARD on the basis of aboveground symptoms. No trees showing aboveground symptoms of ARD were present in non-symptomatic plots. The position of each tree within the plot was surveyed and numbered with a metal tag nailed on the root collar. The species, height class and diameter at 1.3 m were recorded for each tree. All trees within the plots were then pulled vertically from the soil, with as much of the root system left intact as possible, using a 28-t excavator fitted with a Unicon harvesting head. During extraction, large lateral roots usually broke off at 1.5–2 m from the root collar. Large roots that broke off near the root collar were recovered, but some smaller roots that broke off were lost. Following extraction, the stump was removed from the rest of the tree. Any remaining soil was removed by hand and roots were inspected for signs of infection by *A. luteobubalina*. The number of primary roots that were infected was recorded for each stump. Infected wood and/or bark samples were removed from all infected stumps and taken to the laboratory where isolations were undertaken to establish whether or not *A. luteobubalina* was present. At Warren 2 and Warren 5, samples were also taken from infected trees within the stand area between the plots.

Isolations were made on 2.5% malt extract agar. To determine the distribution of genetically distinct isolates (clones), the resulting *A. luteobubalina* isolates were paired in culture to determine the number of somatic incompatibility groups present (Adams 1974; Kile 1983) in each plot and at each site. Isolates from the same plots were paired in all possible combinations, with self-pairings as controls. Isolates to be tested were placed about 2–3 mm apart on the same plate and incubated in darkness at 20ºC for 4 weeks. Those pairings that grew together with no sign of intra-specific antagonism were considered to be compatible and therefore to belong to the same clone (Kile 1983). Once individual clones within plots were identified, pairings between plots were undertaken.

Statistical analysis

The amount of disease was determined at two levels: firstly by considering all the trees within each plot, then by considering the dominant and subdominant trees separately. Within each plot, the total number of infected trees, including the number of dominant and subdominant trees that were infected, was determined. These data were used to calculate means for (a) the overall incidence of disease at each site and forest block, (b) the fraction of the overall incidence contained within the dominant and subdominant trees, and (c) the actual fraction of dominant and subdominant trees infected at each forest block. The number of infected roots on each infected tree was used to determine the fraction of roots infected within the dominant and subdominant trees at each forest block.

Analysis of variance (ANOVA) was used to test for differences in disease incidence between (a) plots that were centred on infected dominant or subdominant trees, (b) sites, (c) forest types. ANOVA was also used to test for any difference in (d) the fraction of overall disease incidence contained within the dominant and subdominant strata, and (e) the fraction of dominant and subdominant trees infected at each forest block, as well as (f) differences between the fractions of roots infected within dominant and subdominant trees at each forest block. Means were compared using Student–Newman–Kuel's test at $\alpha = 0.05$. Percentage data for each plot were arcsine transformed prior to analysis, and residuals were examined using standard diagnostic tests (stem-leaf and normal-normal plots) to ensure the assumptions underlying ANOVA were met. Data were analysed using the SAS statistical package (SAS Institute Inc., Cary, NC) and graphs were generated using Microsoft® Excel.

Results

Stand composition in plots

A total of 383 trees were extracted on the two Warren sites and 427 trees on the two Dombakup sites (Table 1). The stand composition in the Warren plots was 25% dominant karri, 41% subdominant karri and 34% subdominant marri. Only two marri trees were classed as dominant. They were left from the previous stand, showed no symptoms of ARD and were too big to extract. They were not included in the analysis. At Dombakup, 21.5% of the trees were dominant karri and 78.5% were subdominant karri.

Disease symptoms

Infected trees displayed typical above- and belowground symptoms of *A. luteobubalina* infection. Aboveground symptoms included basal scarring accompanied by a wet stringy white rot of infected sapwood, extensive killing and discolouration of the stem bark for up to 3 m above the ground, and the formation of clusters of basidiomes at the bases of infected trees in the autumn. Belowground symptoms included the ends of roots rotted away, white-rotted sapwood and white mycelial fans below dead or infected bark. Rhizomorphs were not observed in the field. *Armillaria luteobubalina* was successfully isolated from 82% of the infected trees. No isolations were attempted from one plot at

Table 2. The fraction of all trees in each plot infected with *Armillaria luteobubalina* as assessed by aboveground and belowground symptoms

Location	Plot type	Number of trees[1]	Fraction of trees infected as assessed by symptoms (%)	
			Aboveground (Mean ± se)	Belowground (Mean ± se)
Dombakup 8	Symptomatic	142	21.8 ± 4.4	42.5 ±7.8
	Non-symptomatic	84	0.0	16.6 ± 0.4
Dombakup 9	Symptomatic	121	29.9 ± 4.4	47.5 ± 6.9
	Non-symptomatic	80	0.0	18.5 ± 12.6
Dombakup total	Symptomatic	263	25.9 ± 3.3	45.0 ± 4.9
	Non-symptomatic	164	0.0	17.4 ± 5.1
Warren 2	Symptomatic	139	23.0 ± 7.4	45.5 ± 13.4
	Non-symptomatic	69	0.0	10.6 ± 1.5
Warren 5	Symptomatic	113	23.3 ± 5.8	34.4 ± 3.0
	Non-symptomatic	62	0.0	5.2 ± 5.2
Warren total	Symptomatic	252	23.1 ± 4.4	40.0 ± 6.7
	Non-symptomatic	131	0.0	7.9 ± 2.7
Overall total	Symptomatic	515	24.5 ± 2.6	42.5 ± 4.1
	Non-symptomatic	295	0.0	12.6 ± 3.2

[1]Total number of trees extracted (dominant and subdominant karri and marri) in all plots at each site.

Dombakup 9, which accounted for 7% of the infected trees. In samples that had advanced rot, yeast infection generally prevented successful isolation of *Armillaria*, but in many yeast-infected cultures, although *Armillaria* did not produce a mycelial colony, rhizomorphs extended directly from the small sample of infected wood through the agar medium and beyond the boundary of the yeast culture. Sub-cultures from the rhizomorphs produced typical mycelial colonies of *A. luteobubalina*.

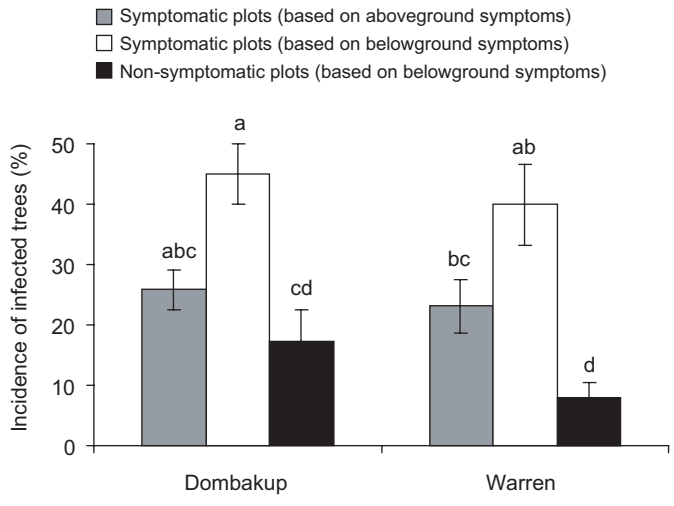

Figure 1. The average (± se) incidence of infection in plots showing aboveground symptoms or non-symptomatic for Armillaria root disease. NB. The incidence detected by aboveground symptoms in the non-symptomatic plots was zero. Significant differences ($P = 0.05$) between treatments within sites are indicated by letters above columns.

Type of plot

There was no significant difference in the incidence of infection found in symptomatic plots that were centred on either dominant or subdominant trees ($P = 0.5$ and 0.1 based on aboveground symptoms, and $P = 0.9$ and 0.6 based on actual belowground levels for Dombakup and Warren, respectively). Therefore, no further distinction was made between plots centred on either dominant or subdominant trees.

Belowground incidence of infection

Incidence of disease in the non-symptomatic plots was significantly lower ($P < 0.05$) than in the symptomatic plots at both sites (Fig. 1). The average incidence at Warren was lower (but not significantly) than at Dombakup, overall averaging 12.6% of trees (Fig. 1 and Table 2).

Within the symptomatic plots, there were no significant differences in the actual levels of infection between the two sites at both Dombakup and Warren (Fig. 1), averaging 42.5% of trees (Table 2).

Aboveground symptoms verses belowground incidence

Assessment using aboveground symptoms substantially under-estimated the level of disease (Fig. 1 and Table 2). In symptomatic plots, the mean level of infection based on belowground symptoms was 20% higher at Dombakup and 17% higher at Warren than levels based on aboveground symptoms, with 45% and 40% of the trees, respectively, being infected (Table 2). Within individual symptomatic plots, the difference between incidence based on aboveground and belowground symptoms was 0–38% at Dombakup and 0–40% at Warren. In the non-symptomatic plots, mean infection levels were 17% and 8%, respectively (Table 2),

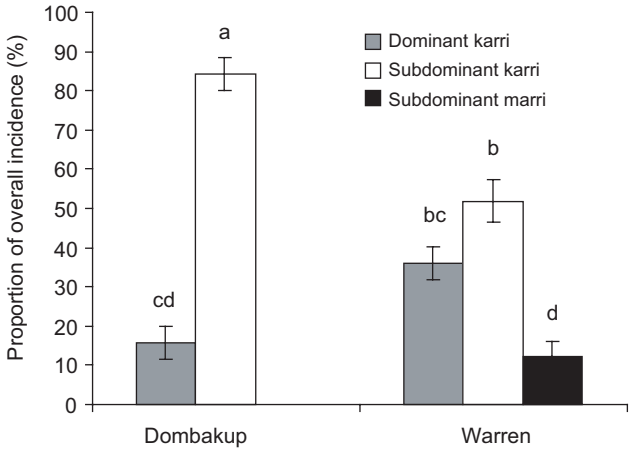

Figure 2. The fraction of the overall incidence contained in the dominant and subdominant strata. Means (± se) of plots in which trees showed aboveground symptoms of Armillaria root disease. Significant differences ($P = 0.05$) between treatments within sites are indicated by letters above columns.

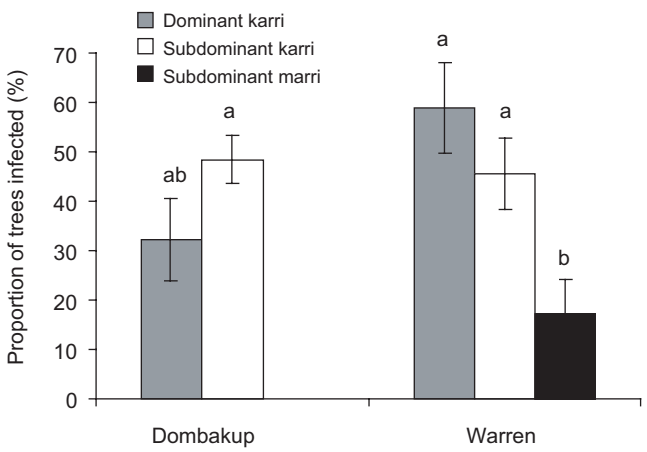

Figure 3. The fraction of trees, in each species and stand class, infected with *Armillaria luteobubalina*. Means (± se) of plots in which trees showed aboveground symptoms of Armillaria root disease. Significant differences ($P = 0.05$) between treatments within sites are indicated by letters above columns.

with ranges in individual plots of 6–31% at Dombakup and 0–12% at Warren. Overall, assessment based on aboveground symptoms detected only one-half of the total number of infected trees within the symptomatic plots. Of the 810 trees pulled in the study, 125 were assessed as being infected based on aboveground symptoms but 250 were actually infected when belowground symptoms were assessed.

Tree stand class and species

At Dombakup, a significantly higher ($P < 0.05$) fraction of the overall disease incidence was established within the subdominant trees. At Warren, both dominant and subdominant karri carried a similar fraction of the overall incidence, but the amount contained in the marri component was significantly less ($P < 0.05$) (Fig. 2). A higher proportion of the dominant karri was infected at Warren than at Dombakup (Fig. 3).

Fraction of primary roots infected

When determined by the fraction of primary roots infected, the intensity of infection in the infected dominant trees on the Dombakup sites was considerably less than that in the subdominant trees at Dombakup and in both the dominant and subdominant karri at Warren. On the Warren sites, the intensity of infection was similar in infected dominant and subdominant karri and infected subdominant marri. Data concerning marri stump coppice were not included (see discussion). Infected subdominant karri had a similar fraction of their roots infected at both the Dombakup and Warren sites (Fig. 4).

The number of genotypes and size of clones

Three genotypes were found to be present at each of the two Warren sites. At Warren 2, one genotype was present in four separate plots, and another in two plots, but only one plot had two separate genotypes present. At Warren 5, each of the three genotypes was present in two separate plots, but only one plot contained two separate genotypes. One plot was free of infection. By isolating from infected trees between the plots, the largest clone was found to occupy about 0.4 ha within the study area. The remainder occupied areas ranging from 0.01 ha to 0.2 ha. One small clone (0.02 ha) was identified at Warren 2 which was not present in any plots but had infected a number of trees in an area of forest between the plots.

There were four genotypes within the plots at each of the two Dombakup sites. At Dombakup 8, one genotype occupied three plots and spread over a distance of 100 m. At Dombakup 9, only one genotype spanned at least two plots, which were separated by 50 m.

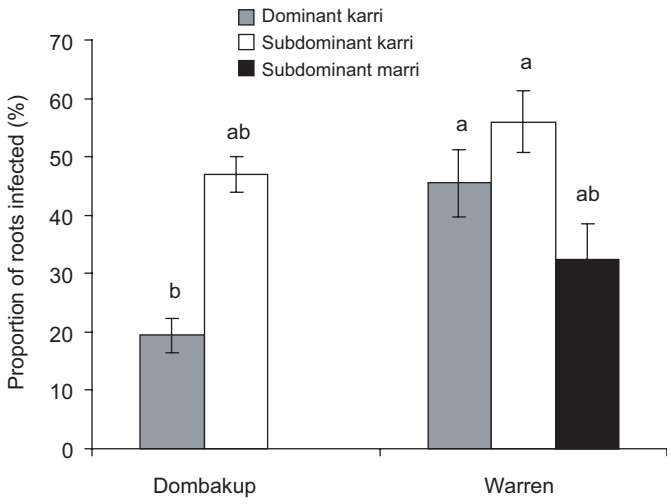

Figure 4. The fraction of primary roots infected, in each species and stand class, on all infected trees extracted from each site. Significant differences ($P = 0.05$) between treatments within sites are indicated by letters above columns.

Discussion

In the past, anecdotal evidence suggested that trees on wetter, higher quality sites appeared to be more prone to infection. The results of this study, however, suggest that dominant trees in mixed karri–marri stands are more susceptible to ARD than those in pure stands on higher quality sites. In the course of this study, however, it was observed that disease centres with little or no regeneration were common on both of the Dombakup sites, while unstocked openings due to disease were not observed on the two Warren sites. This indicates that the disease was further advanced on the Dombakup sites, the more susceptible trees having already been eliminated from the stands, supporting the argument that high quality sites are more susceptible to infection. The susceptibility of trees on different sites needs to be further investigated.

In all the plots centred on subdominant trees, a neighbouring dominant tree was also found to be infected. This may account for there being no difference in the amount of infection in the plot types based on the status of the central tree.

Most marri trees sampled were multi-stemmed coppice that had sprouted from stumps following the previous harvest. Data concerning infected marri stump coppice were used only to determine infection levels in plots and at sites; they were not used to determine the intensity of infection in marri trees. Infected marri saplings (with single stems) generally showed no aboveground symptoms of infection. When marri stump coppice was infected, it was generally restricted to the original stump. Mycelial sheets formed between the old stump and the heel of the coppice, but infection did not extend into the roots or collar of the new growth. In jarrah (*Eucalyptus marginata* Donn ex. Sm.) forests, marri is regarded as a susceptible species, with extensive colonisation of root and stem sapwood occurring in infected trees (Shearer and Tippett 1988). In karri forest, however, the observed lack of colonisation of marri coppice on infected stumps and the lower incidence of infected roots on regenerated trees (Fig. 4) suggest it may be more resistant than karri. This difference may be related to a drier environment creating stress-related susceptibility of marri trees in jarrah forest.

In this study, detailed aboveground examination detected only 50% of the total number of trees that were actually infected. This led to true levels of infection being underestimated by at least 20% (and up to 40% in some plots). In apparently disease-free portions of the stands (i.e. non-symptomatic plots) up to 12% of the trees were infected at Warren and up to 31% at Dombakup. Similar results have been recorded in Canada. In the southern interior of British Columbia, work in mixed conifer stands infested with *A. ostoyae* (Romagn.) Herink showed that an average of 51%, 28% and 23% of non-symptomatic trees in plots centred around infected dead trees in dry, moist and wet climatic regions, respectively, had infected root systems. In addition, the actual disease level in non-symptomatic plots in dry, moist and wet climatic regions was 6%, 24% and 15% respectively (Morrison *et al*. 2000). In conifer plantations in Ontario, an average of 58% of symptomless trees surrounding trees infected with *A. obscura* (Pers.) Herink (= *A. ostoyae*) were reported to be infected (Whitney *et al*. 1989). Similarly, in 25–60-y-old Douglas-fir stands on Vancouver Island, about one-half of the trees infected with *Phellinis weirii* (Murr.) Gilb. were detected by using aboveground indicators (Wallis and Bloomberg 1981).

Applying disease status to yield prediction models may be important for demonstrating sustainability. Using the Prognosis model (Stage *et al*. 1990) adapted for the dry climatic region of southern interior British Columbia, Morrison *et al*. (2000) determined a reduction in yield of 10% from that of a disease-free prediction when using aboveground incidence of ARD to initialise the model. When actual disease incidence was used, the predicted yield was reduced by 35%.

These results challenge the reliability of surveys based on aboveground disease symptoms to determine actual disease levels and management options and thus to predict future mortality, growth and yield in infested stands. All four sites in this study were in stands scheduled for first thinning. In karri regrowth, first thinning is normally carried out commercially. Thinnings are either chipped for export, or utilised in the local small-sawlog market, depending on the size and quality of logs. Conventional thinning exacerbates disease incidence in infested stands (Robinson 2003).

At present, disease management options require nominal 25-m buffer zones to be placed around all infection sites identified by ground surveys, to indicate possible exclusion zones for thinning (Robinson *et al*. 1998). In heavily infested stands, overlapping buffers may encompass the whole stand, but in low to moderately infested stands or in stands where disease is present but not expressed, ground surveys may fail to detect sufficient infection sites in order to successfully buffer the entire area of infestation. Managers, however, must critically evaluate disease impact and management objectives to ensure that the level of loss justifies any control measures taken (Wargo and Shaw 1985) and/or the cost of more intensive survey (Wallis and Bloomberg 1981). Once it has been established that ARD is present, Robinson (2003) has recommended that whole-tree pulling be utilised during the first thinning on high quality karri regrowth sites. This negates the need for disease buffers and more intensive survey, and allows scheduled thinning to go ahead.

In this study 15 genotypes of *A. luteobubalina* were isolated from about 7 ha of forest. At the Warren 2 site at least 50 trees were infected by the same genotype. In mixed-species eucalypt forests in Victoria, Kile (1983) reported 36 genotypes of *A. luteobubalina* from a total forest area of 24 ha, and estimated that new disease centres, arising from basidiospore infection, had occurred at the rate of less than one per year. Thus disease spread by airborne spore dispersal does not need to be considered when formulating management options in eucalypt forests infested with *A. luteobubalina*.

This contrasts dramatically with the situation in New Zealand where up to 93 genets of *A. novae-zelandiae* (G. Stev.) Herink and 56 genets of *A. limonea* (G. Stev.) Boesewinkel per hectare have been observed in the soil following clearing of native forests (Hood and Sandberg 1987) and airborne spread of disease must be considered because basidiospores have the ability to infect freshly cut stumps and wood in the *Pinus radiata* D. Don plantations that are established on such sites (Hood and Sandberg 1987; Hood *et al*. 2002a,b).

The results of belowground survey in this study support the suggestion that whole-tree pulling can be used to thin high-quality karri regrowth stands infested with ARD (Robinson 2003). This eliminates the possibility that infested portions of stands may not be buffered despite disease survey. The fact that aboveground survey underestimates true levels of disease needs to be taken into account when modelling future yields. A broader study, including lower quality sites, is needed to determine if these findings are consistent for all types of karri regrowth.

Acknowledgements

We thank John Neal, Ros Pilbeam and Jeanette Knudsen for their technical contributions to the project, Tim Wardlaw, Janet Farr and Martin Rayner for informative discussion, and an anonymous referee for useful comments on the draft. Technical assistance in the project was partially funded by the State Forest Resources Business Unit (now the Forest Products Commission).

References

Adams, D.H. (1974) Identification of clones of *Armillaria* in young-growth ponderosa pine. *Northwest Science* **48,** 21–28.

CALM (1992) *Silviculture Specification 1/92: Karri Thinning*. Department of Conservation and Land Management, Western Australia.

Edgar, J.G., Kile, G.A. and Almond, C.A. (1976) Tree decline and mortality in selectively logged eucalypt forests in central Victoria. *Australian Forestry* **39,** 288–303.

Hood, I.A. and Sandberg, C.J. (1987) Occurrence of *Armillaria* rhizomorph populations in the soil beneath indigenous forests in the Bay of Plenty, New Zealand. *New Zealand Journal of Forestry* **17,** 83–99.

Hood, I.A., Redfern, D.B. and Kile, G.A. (1991) *Armillaria* in planted hosts. In: Shaw, C.G.III and Kile, G.A. (eds) *Armillaria Root Disease*. Agriculture Handbook No. 169. USDA Forest Service.

Hood, I.A., Horner, I.J., Gardener, J.F. and Sandberg, C.J. (2002a) Armillaria root disease in *Pinus radiata* in New Zealand. 1: Basidiospore dispersal. *New Zealand Journal of Forestry Science* **32,** 94–102.

Hood, I.A., Kimberley, M.O., Gardener, J.F. and Sandberg, C.J. (2002b) Armillaria root disease in *Pinus radiata* in New Zealand. 3: Influence of thinning and pruning. *New Zealand Journal of Forestry Science* **31,** 116–132.

Kellas, J.D., Kile, G.A., Jerrett, R.G. and Morgan, B.J.T. (1987) The occurrence and effects of *Armillaria luteobubalina* following partial cutting in mixed eucalypt stands in the Wombat Forest, Victoria. *Australian Forest Research* **17,** 263–276.

Kile, G.A. (1981) *Armillaria luteobubalina*: A primary cause of decline and death of trees in mixed species eucalypt forests in central Victoria. *Australian Forest Research* **11,** 63–77.

Kile, G.A. (1983) Identification of genotypes and the clonal development of *Armillaria luteobubalina* Watling & Kile in eucalypt forests. *Australian Journal of Botany* **31,** 657–671.

Kile, G.A. and Watling, R. (1983) *Armillaria* species from south-eastern Australia. *Transactions of the British Mycological Society* **81,** 129–140.

Kile, G.A. and Watling, R. (1988) Identification and occurrence of Australian *Armillaria* species, including *A. pallidula* sp. nov. and comparative studies between them and non-Australian tropical and Indian *Armillaria*. *Transactions of the British Mycological Society* **91,** 305–315.

Kile, G.A., Watling, R., Malajczuk, N. and Shearer, B.L. (1983) Occurrence of *Armillaria luteobubalina* Watling & Kile in Western Australia. *Australian Plant Pathology* **12,** 18–20.

Kile, G.A., McDonald, G.I. and Byler, J.W. (1991) Ecology of disease in natural forests. In: Shaw, C.G.III and Kile, G.A. (eds) *Armillaria Root Disease*. Agriculture Handbook No. 169. USDA Forest Service.

Marks, G.C., Almond, C.A., Edgar, J.C. and Kile, G.A. (1976) Spread of *Armillaria* spp. in the bark of *Eucalyptus obliqua* and *E. bicostata*. *Australian Forest Research* **7,** 115–119.

Morrison, D.J., Pellow, K.W., Norris, D.J. and Nemec, A.F.L. (2000) Visual versus actual incidence of Armillaria root disease in juvenile coniferous stands in the southern interior of British Columbia. *Canadian Journal of Forest Research* **30,** 405–414.

Pearce, M.H., Malajczuk, N. and Kile, G.A. (1986) The occurrence and effects of *Armillaria luteobubalina* in the karri (*Eucalyptus diversicolor* F.Muell.) forests of Western Australia. *Australian Forest Research* **16,** 243–259.

Podger, F.D., Kile, G.A., Watling, R. and Fryer, J. (1978) Spread and effects of *Armillaria luteobubalina* sp. nov. in an Australian *Eucalyptus regnans* plantation. *Transactions of the British Mycological Society* **71,** 77–87.

Rayner, M.E. (1991) Site index and dominant height growth curves for regrowth karri (*Eucalyptus diversicolor* F.Muell.) in south-western Australia. *Forest Ecology and Management* **44,** 261–283.

Robinson, R.M. (2003) Short-term impact of thinning and fertilizer application on Armillaria root disease in regrowth karri (*Eucalyptus diversicolor* F.Muell.) in Western Australia. *Forest Ecology and Management* **176,** 417–426.

Robinson, R.M., Farr, J.D. and Wallace, J.M. (1998) Ground surveys to assess forest health: sampling strategies in regrowth forests. Paper to Forest Health Surveillance Workshop, Research Working Groups 7 and 8, 2 November 1998, Brisbane.

Shearer, B.L. and Tippett, J.T. (1988) Distribution and impact of *Armillaria luteobubalina* in the *Eucalyptus marginata* forest of south-western Australia. *Australian Journal of Botany* **36,** 433–445.

Stage, A.R., Shaw, C.G., Marsden, M.A., Byler, J.W., Renner, D.L., Eav, B.B., McNamee, P.J., Sutherland, G.D. and Webb, T.W. (1990) *User's Manual for Western Root Disease Model*. USDA Forest Service General Technical Report No. INT-267.

Wallis, G.W. and Bloomberg, W.J. (1981) Estimating the total extent of *Phellinus weirii* root rot centres using above- and below-ground disease indicators. *Canadian Journal of Forest Research* **11,** 827–830.

Wargo, P.M. and Shaw, C.G.III (1985) *Armillaria* root rot: the problem is being solved. *Plant Disease* **69,** 826–832.

Whitney, R.D., Ip, D.W. and Irwin, R.N. (1989) *Armillaria* infection in symptomless white spruce, black spruce and red pine saplings in Ontario plantations. In: Morrison, D.J. (ed.) *Proceedings of the 7th International Conference on Root and Butt Rots.* Vernon and Victoria, British Columbia, Canada, August 9–16 1988. Forestry Canada, Victoria, BC, pp. 546–549.