Pacific Science (1983), vol. 37, no. 4

Armillaria Root Rot in Eucalypt Forests: Aggravated Endemic Disease[1]

G. A. KILE[2]

ABSTRACT: Species of the woody root rot fungus *Armillaria* are indigenous in cool temperate rain forest, mixed forest, and wet and dry sclerophyll eucalypt forests in Australia. Four species have been described or identified from southeastern Australia: *A. luteobubalina* Watling and Kile, *A. fumosa* Kile and Watling, *A. hinnulea* Kile and Watling, and *A. novae-zelandiae* (Stevenson) Boesewinkel. The latter species was first described from New Zealand, and *A. hinnulea* also occurs in that country. *Armillaria novae-zelandiae* and *A. hinnulea* occur in wet forests (rain forest, mixed forest, and wet sclerophyll communities), while *A. luteobubalina* and *A. fumosa* are found mainly in dry sclerophyll forests.

Armillaria luteobubalina is so far the only species known to behave as a primary pathogen in native forests. While the fungus has an extensive geographical distribution in southeastern Australia, damage is most severe in selectively logged forests in the central highlands of Victoria, where it is estimated that approximately 3–5% of the forest area is moderately to severely affected. The fungus kills all species of eucalypts and a wide range of the understory trees and shrubs present in the forests. Most infections occur in small (0.1–1.0 ha), well-defined patches, but larger (up to 20–30 ha), more diffuse infections also occur. Evidence of primary pathogenicity includes (*a*) constant association of the fungus with disease; (*b*) the pattern of disease development within stands (the fungus spreads by root contact from infected food bases); (*c*) correlation between root infection and symptom development in large trees; (*d*) evidence of host resistance to infection; and (*e*) pathogenicity in pot and field trials. There is no evidence that climatic stress or other pests or pathogens initiate disease.

Within the forest, the fungus has a discontinuous distribution. Studies of genotypes of the fungus (identified by analyses of mating alleles, since *Armillaria* sp. are bifactorial heterothallic, or intraspecific antagonism), suggest that *A. luteobubalina* consists of a community of genetically distinct mycelia. Individual genotypes may contract, expand, or coalesce, depending on circumstance. The development and status of the community depends on the two processes of new basidiospore infection and local spread by vegetative growth through root systems. Similar patterns of genotype distribution and clonal development were evident in logged and unlogged forest. It is concluded that root rot caused by the fungus is endemic in these forests but that logging has aggravated the disease.

FOUR SPECIES OF THE WOODY root rot fungus *Armillaria* are native to cool temperate rain forest, mixed forest, and wet and dry sclerophyll eucalypt forests in southern Australia: *A. luteobubalina* Watling and Kile, *A. fumosa* Kile and Watling, *A. hinnulea* Kile and Watling, and *A. novae-zelandiae* (Stevenson) Boesewinkel (Kile and Watling 1981, 1983, Podger et al. 1978). The latter species was first described from New Zealand (as *Armillariella novae-zelandiae* Stevenson; Stevenson 1964), and *A. hinnulea* has been found in

[1] Manuscript accepted 5 October 1983.
[2] CSIRO Division of Forest Research, Stowell Avenue, Battery Point, Tasmania, 7000.

Nothofagus forest in the South Island of New Zealand (Kile and Watling 1983). *Armillaria novae-zelandiae* and *A. hinnulea* occur in wet forests (cool temperate rain forest, mixed rain forest–eucalypt communities, and wet sclerophyll eucalypt forests), while *A. luteobubalina* and *A. fumosa* are found in the drier, more open eucalypt forests (Kile and Watling 1983). Occurrence in different vegetation types is paralleled by differences in ecology and pathogenicity. *Armillaria hinnulea* and *A. novae-zelandiae* are weak primary but effective secondary pathogens (Kile 1980, Kile and Watling 1983), while *A. luteobubalina* can act as a primary pathogen in native forests. The pathogenicity of *A. fumosa*, a rare species by comparison with the other three, is unknown. *Armillaria luteobubalina* has been found in New South Wales, Victoria, Tasmania, South Australia, and Western Australia (Kile and Watling 1981, 1983, Kile et al. 1983, Warcup and Talbot 1981), and while forests in the highlands of west central Victoria are those currently most severely affected by *Armillaria* root rot, disease attributable to *A. luteobubalina* is found throughout most of its known geographical range (Kile et al. 1983). This paper considers canopy dieback caused by *A. luteobubalina* in these central highlands forests.

AFFECTED FORESTS

The association between an *Armillaria* (now identified as *A. luteobubalina*) and tree decline and death in highland forests in west central Victoria (particularly the Mt. Cole, Wombat, and Mt. Macedon State Forests) was first recorded in the early 1950s (Purnell 1959), and disease has increased in severity and extent since that time (Edgar, Kile, and Almond 1976).

For management purposes, the Mt. Cole, Wombat, and Macedon forests are subdivided into various zones. The hardwood production zone is the area in which crown dieback caused by *Armillaria* is most evident. Forests in this zone are of the tall open type (Specht, Roe, and Broughton 1974), with top heights of more than 25 m. These forests are commonly termed "mixed-species forests," because they are dominated by various eucalypt species of which the three most widely distributed are *Eucalyptus obliqua* L. Hérit., *E. radiata* Sieb. ex DC., and *E. viminalis* Labill. One or more of the species *E. globulus* Labill. subsp. *bicostata* (Maid. et al.) Kirkp., *E. baxteri* (Benth.) Maid. & Blakely, *E. cypellocarpa* L. Johnson, *E. rubida* Deane & Maid., *E. dives* Schau., and *E. ovata* Labill. also may be present, depending on site conditions. Much of the hardwood production zone carries a scrub layer dominated by wattles such as *Acacia dealbata* Link and *A. verticillata* (L. Herit.) Willd., while bush peas (*Pultanea* sp.), wire grass (*Tetrarrhena juncea* R. Br.), and bracken [*Pteridium esculentum* (Forst. f.) Nakai] are common in the lower strata. Soils in the forests range from deep sandy clays at Mt. Cole and parts of Macedon to poorly structured duplex soils derived from Ordovician sediments over much of the Wombat forest. Some forest occurs on kraznozem type soils derived from more recent basalts. Annual rainfall in the region ranges from 700 to 1200 mm, depending on elevation, with a distinct winter maximum.

Cutting in the forests commenced in the 1840s and was extremely heavy in the years following the discovery of gold in 1851. Significant areas of even-aged regeneration developed following this heavy utilization, and these stands have been selectively logged over recent decades. This pattern of management has produced a forest consisting of discrete stands of variously thinned, uniform overstory, with variable amounts of younger trees or regrowth distributed beneath.

DISEASE SYMPTOMS AND IMPACT

All eucalypt species within the forest are affected, and, in addition, a range of understory species including *Acacia dealbata*, *A. melanoxylon*, *A. verticillata*, *Cassinea aculeata* (Labill.) R. Br., *Hibbertia obtusifolia* DC., *Daviesia ulicifolia* Andr., and *Dianella* sp. may be infected and killed.

Progressive crown deterioration, including reduction in foliage density, dieback of twigs

and branches of the primary crown together with development of epicormic shoots, is a feature of *Armillaria* root rot of large eucalypts. Regrowth trees and shrubs may show little crown deterioration and often wilt suddenly. The physiological basis for these differences in disease expression is unknown. Extensive damage by *Armillaria* is present on the roots and stem bases of many trees that show moderate to severe crown decline. The symptoms of *Armillaria* attack and mode of spread in *Eucalyptus obliqua* and *E. globulus* subsp. *bicostata* have been described (Marks et al. 1976). Exudation of kino through stem and root rot bark occurs on some diseased trees (Edgar, Kile, and Almond 1976).

Many parts of the Mt. Cole, Wombat, and Macedon forests are at present only lightly affected by dieback, with scattered individuals and small groups of trees dying throughout the various stands. However, in all three forests, there are significant, defined areas of severe dieback, some of which have a long history of disease (Purnell 1959). Severely affected areas vary in extent from 0.2 to 20 ha, and from well-defined circular or oval patches to the more common case of irregularly shaped areas with diffuse boundaries. Some of these diffuse and extensive areas are coalescing patches in which cutting and burning have influenced or obscured the pattern of disease development.

In areas showing severe effects, all the eucalypts are dead or dying. Salvage logging or burning may result in eucalypt regeneration in some of these areas, but, since this is also susceptible to dieback, its longevity is questionable. In undisturbed diseased patches, the ground cover is frequently very dense and this will prevent further eucalypt regeneration.

It was estimated (Edgar, Kile, and Almond 1976) that approximately 2500 ha, or 3%, of the Mt. Cole, Wombat, and Macedon forests are moderately to severely affected by *Armillaria* root rot. Growth losses are in the range 0.3–2.0 m^3/ha/annum, depending on site and severity of dieback (Edgar, Kile, and Almond 1976, J. D. Kellas, personal communication). In addition to these losses, scattered and small patch mortality is evident in regrowth stands. The effect of *Armillaria* infection on growth of regrowth trees may vary with the species. In *Eucalyptus obliqua* with 51–75% of stem circumference infected by *A. luteobubalina*, average monthly girth increment was only 41% of that of healthy trees, while in similarly infected *E. globulus* subsp. *bicostata*, growth was not significantly reduced (Kile, Kellas, and Jarrett 1982).

EVIDENCE OF PRIMARY PATHOGENICITY OF *Armillaria luteobubalina*

Armillaria luteobubalina is only one of a number of potential causes of crown dieback and mortality of the eucalypts in the mixed-species forests. Accordingly, detailed investigations may be required to establish the cause(s) of dieback in any particular locality (Edgar, Kile, and Almond 1976). The detailed evidence for the primary pathogenicity of *A. luteobubalina* has been discussed (Kile 1981) and includes the constant association of the fungus with the disease syndrome described, a pattern of contagion consistent with that for an organism dependent on a woody food base, a correlation between infection and symptom development in large trees, evidence of host resistance to infection via compartmentalization reactions, and pathogenicity of the fungus in pot and field inoculations. Pathogenicity trials indicate variation in the pathogenicity of genotypes which could explain some variations in disease expression.

CLONAL DEVELOPMENT OF *Armillaria luteobubalina* IN EUCALYPT FOREST

Within the forest the fungus has a discontinuous distribution often occurring in relatively well-defined disease patches or centers, and significant areas of the forest are apparently free of the fungus (Kile 1981). Therefore, it seems probable that dispersal of the fungus has been the result of two processes: first, the initiation of new infections by basidiospores, and second, subsequent

vegetative growth of the fungal mycelium through host root systems leading to a wider local distribution. These hypotheses were tested by assessing methods for identifying genotypes (genetically distinct mycelia) of the fungus within forest stands and then applying these methods to determine the origin, size, and distribution of clones (defined as multiple isolates of the same genotype from different hosts in a particular area; Kile 1983).

Genotypes were differentiated by pairings of single basidiospore isolates to determine the identity and distribution of mating alleles (*Armillaria luteobubalina* is bifactorial heterothallic) or by utilizing intraspecific antagonism between genetically distinct forest isolates (Kile 1983). Both methods gave similar results although neither could distinguish between closely related genotypes.

Forty-nine patches of *Armillaria luteobubalina* infection with areas from approximately 24 m^2 to 3.5 ha were mapped, and isolates were obtained from hosts within the patches. Seventy-one percent of infected areas were occupied by a single genotype, and the maximum number found in any one patch was three. In multiple genotype patches the different genotypes occupied separate sectors. Clones of some genotypes had a locally discontinuous distribution, while others were restricted to a single location. Within areas occupied by a single genotype all hosts were infected by that genotype. Clonal development was similar in unlogged and logged stands.

The bifactorial heterothallism of *Armillaria luteobubalina* and the occurrence of mycelia with distinctive combinations of mating alleles is strong evidence that basidiospores are involved in establishing new infections, although experimental attempts to reproduce basidiospore infection have been unsuccessful. The sole occupancy of areas by single genotypes, their occurrence on all hosts within those areas, and evidence that disease transmission occurs via root contacts (Kile 1981) supports the hypothesis that local spread is by vegetative growth through host root systems.

The discontinuous distribution of some genotypes (some clones of the same genotype were located up to 600 m apart) most probably arose from irregular spread from initial sources of infection or the breakup of large clones, rather than from the development of sibling clones (Kile 1983). The great range of clone size emphasizes the irregular age of infections, many no doubt predating the present forest stands, and combined with a knowledge of pathogen survival in inoculum and rate of spread leads to an appreciation of the dynamic nature of the fungus within these eucalypt forests (Kile 1983).

Armillaria ROOT ROT IN VIRGIN FORESTS

The greatest incidence and severity of *Armillaria* root rot in recent years has been associated with increased or more frequent forest harvesting (Edgar, Kile, and Almond 1976, Kile 1981). No quantitative comparison of the occurrence and impact of *Armillaria* root rot in virgin and logged forests is possible because of the lack of any significant areas of the former. However, there is evidence that the fungus caused disease in virgin forests.

In a near-virgin community in the Wallaby Creek catchment of the Melbourne Metropolitan Board of Works adjacent to the Mt. Disappointment State Forest in central Victoria (which is comparable in species composition and site quality to areas affected by *Armillaria* in the Mt. Cole, Wombat, and Macedon forests), nine *Armillaria* root rot patches were located within a 50-ha sector of forest. No logging had ever occurred within or adjacent to at least five of the patches, although occasional stumps (average 12/ha) were found in other parts of the area, apparently from trees cut during the 1880s prior to the declaration of the area as a water catchment in 1896. The disease centers were relatively open, some dead or declining trees were present, and trees around the margin of patches often showed compartmentalized root and stem infections. The overall impression was that the disease centers were relatively stable or perhaps expanding slowly. It seems probable that the more susceptible individuals had been killed while the more resistant trees, which could compartmen-

talize infections, remained alive. Most patches contained only one *A. luteobubalina* genotype, which suggests clonal development similar to that in more disturbed stands. The value of these patches for long-term observations was reduced when the area was burnt by wildfire in late 1982.

CONCLUSIONS

The wide distribution of *Armillaria luteobubalina* in southern Australia and its intimate association with native forest communities indicate that it is a native species and that the fungus probably caused low-level endemic disease in virgin forest communities. It is believed that forest harvesting in areas where the fungus occurs, especially relatively frequent partial cutting, creates additional food base for the fungus and increases the intensity and extent of disease development by this mildly aggressive pathogen, i.e., aggravated endemic disease. *Armillaria* root rot is a further illustration of the diversity of agents that may cause canopy dieback in native forests.

The development of *Armillaria* root rot in residual stands following selective cutting has been observed in a number of conifer forest types (Geschwind 1920, Koenigs 1969, Shaw 1975). Damage by *A. luteobubalina* in mixed-species eucalypt forest is the first such report of damage in a major natural hardwood forest. The ability of *Armillaria* species to act as primary pathogens in native forest communities has been less well recognized in the past than their role as disease in plantations established in cutover native forests and as secondary pathogens in many dieback and decline diseases.

LITERATURE CITED

EDGAR, J. G., G. A. KILE and C. A. ALMOND. 1976. Tree decline and mortality in selectively logged eucalypt forests in central Victoria. Austral. For. 39:288–303.

GESCHWIND, A. 1920. Das Vorkommen des Hallimasch (*Agaricus melleus* Quel.) in den bosnisch-herzegowinischen Wäldern. Naturwiss. Z. Forst. Landwirtsch. 18:182–186 (CSIRO translation).

KILE, G. A. 1980. Behaviour of an *Armillaria* in some *Eucalyptus obliqua–Eucalyptus regnans* forests in southern Tasmania and its role in their decline. Eur. J. For. Path. 10:278–296.

———. 1981. *Armillaria luteobubalina:* A primary cause of decline and death of trees in mixed species eucalypt forests in central Victoria. Austral. For. Res. 11:63–77.

———. 1983. Identification of genotypes and clonal development of *Armillaria luteobubalina* Watling and Kile in eucalypt forests. Austral. J. Bot. 31:657–671.

KILE, G. A., and R. WATLING. 1981. An expanded concept of *Armillaria luteobubalina*. Trans. Br. Mycol. Soc. 77:75–83.

———. 1983. *Armillaria* species from south-eastern Australia. Trans. Br. Mycol. Soc. 81:129–140.

KILE, G. A., J. D. KELLAS, and R. G. JARRETT. 1982. Electrical resistance in relation to crown dieback symptoms, *Armillaria* infection and growth in *Eucalyptus obliqua* and *E. globulus* subsp. *bicostata*. Austral. For. Res. 12:139–149.

KILE, G. A., R. WATLING, N. MALAJCZUK, and B. L. SHEARER. 1983. Occurrence of *Armillaria luteobubalina* Watling and Kile in Western Australia. Austral. Plant. Path. 12:18–20.

KOENIGS, J. W. 1969. Root rot and chlorosis of release thinned western red cedar. J. For. 67:312–315.

MARKS, G. C., C. A. ALMOND, J. G. EDGAR, and G. A. KILE. 1976. Spread of *Armillaria* spp. in the bark of *Eucalyptus obliqua* and *bicostata*. Austral. For. Res. 7:115–119.

PODGER, F. D., G. A. KILE, R. WATLING, and J. FRYER. 1978. Spread and effects of *Armillaria luteobubalina* sp. nov. in an Australian *Eucalyptus regnans* plantation. Trans. Br. Mycol. Soc. 71:77–87.

PURNELL, H. 1959. "Collar rot" or "dead eye" of *Eucalyptus* species. For. Comm. Vic. Techn. Pap. No. 2, pp. 12–15.

SHAW, C. G. 1975. Epidemiological insights into *Armillaria mellea* root rot in managed

ponderosa pine forest. Ph.D. Thesis. Oregon State University, Corvallis.

SPECHT, R. L., E. M. ROE, and V. H. BROUGHTON. 1974. Conservation of major plant communities in Australia and Papua New Guinea. Austral. J. Bot. Suppl. Ser. No. 7.

STEVENSON, G. 1964. The Agaricales of New Zealand. V. Tricholomataceae. Kew Bull. 19:1–59.

WARCUP, J. H., and P. TALBOT. 1981. Host-pathogen index of plant diseases in South Australia. Waite Agriculture Research Institute, University of Adelaide.