PHYTOPHTHORA ROOT AND COLLAR ROT IN REHABILITATED BAUXITE MINES AND THE ADJACENT *EUCALYPTUS MARGINATA* (JARRAH) FOREST OF WESTERN AUSTRALIA

G E. St. J. Hardy

School of Biology and Biotechnology, Division of Science and Engineering, Murdoch University, Murdoch 6150, Western Australia

SUMMARY

This study gives an overview of recent and current research activities that are being conducted in Western Australia on the biology, ecology and pathology of *Phytophthora cinnamomi* in rehabilitated bauxite mines and the adjacent jarrah (*Eucalyptus marginata*) forest. The work to date indicates that the biology of this pathogen does differ between rehabilitated mines and the adjacent jarrah forest.

INTRODUCTION

Western Australia is unique in its botanical diversity. There are over 7000 species of native vascular plants of which over 3000 are endemic (Keighery 1992). *Phytophthora cinnamomi* has a major impact on this diverse flora and directly effects over 2000 species from a diverse range of plant families (Wills 1993). Its indirect effects in terms of botanical impact through the loss of vertebrate and invertabrate pollinators, and loss of canopy and litter cover has not been measured. The pathogen was probably introduced into the state with the importation of nursery plants in particular fruit trees at the turn of the 20th century. The pathogen was then spread into the natural ecosystems principally by road building, forestry and other humankind activities. This spread has had a major impact in the jarrah forest, banksia woodlands and heathlands in the lower south-west of the state. The pathogen is an excellent example of an introduced pathogen with a wide host range causing considerable damage to a diverse community made of many susceptible plant species.

In Western Australia, the majority of past and current research on this pathogen has been conducted in the jarrah forest. This has been because *P. cinnamomi* has had a considerable impact on the ecology and management of the jarrah forest (Shearer and Tippett, 1989). The research was concentrated in the jarrah forest, since jarrah is an economically important hardwood species. However, more recently, research activities have also centered on the rehabilitation of bauxite mines. The jarrah forest in Western Australia is also the location of one of the largest and most productive bauxite mines in the world (Colquhoun and Hardy, 2000). The bauxite mining and alumina refining company, Alcoa Worldwide Alumina-Australia, supplies 22% of the world's alumina. In the jarrah forest, Alcoa mines and rehabilitates approximately 500 ha of forest annually. Although *P. cinnamomi* is estimated to only impact on 14% of the forest (Davison and Shearer 1990); many of Alcoa's ore bodies encompass areas of forest free of the pathogen. Since, *P. cinnamomi* is spread in soil and water the management of this pathogen between pathogen-free and pathogen-infested pits is daunting.

P. cinnamomi and the disease it causes in the non-mined forest

The jarrah forest occupies approximately 64,000 km^2 of the south-western corner of Western Australia (Abbott and Loneragan 1986). The forest grows on an ancient plateau composed of granite rocks intruded by dolerite dykes. The weathering of this rock has produced deep regolith profiles where the soil is usually about 0.5-1 m of gravelly sandy loam topsoil. Immediately below this soil layer is a concretionary layer of bauxite up to 5 m thick. Beneath this is a layer of up to 30 m of kaolinitic clay extending to bedrock. Bauxite mining occurs in those profiles where the alumina content is sufficiently high. These tend to occur in good quality forest (Abbot and Loneragan 1986).

The climate is typically mediterranean with cool wet winters and hot dry summers. The seasonal pattern of rainfall and temperature strongly influences temporal changes in the activity of *P. cinnamomi* in the jarrah forest. In addition, the soil architecture, its morphology, hydraulic properties, temperature and fertility are important factors that influence the life cycle of *P. cinnamomi* (Shearer and Tippett 1989). Jarrah forest soils are characteristically very permeable when moist, encouraging rapid infiltration of water through the soil profile. However, soil temperatures, soil fertility and soil water matric potentials and soil water content of the jarrah forest soils can be favourable for the activity of the pathogen (survival and dissemination) in autumn through to early summer in many areas. In addition, duricrust and clay layers can impede the vertical percolation of water resulting in near-surface transient perching with lateral flow and seepage of water below the soil surface in some upland areas (Kinal 1986). The importance of subsurface perching and lateral flow of water to the life cycle of *P. cinnamomi* cannot be overstressed. For example, it has been found at depths of 3 m below the soil surface (Shea *et al.* 1983; Kinal *et al.* 1987; Shearer and Shea 1987). Passive dispersal of the fungus downwards in root channels may be very important in distributing the fungus vertically within the soil profile. Water seepage from these upslope areas keep the gravel over clay soil profiles in the zone transitional between upland laterites and the headwaters of streams in shallow valleys moist well into the summer months (Shearer and Tippett 1989). Conditions are consequentially favorable for the survival of *P. cinnamomi* in these areas for most of the year.

There are approximately 600 species of vascular plants in the jarrah forest (Bell and Heddle, 1989), with an average of 58 species per 400 m^2 quadrat in the upland forest (Koch, J. pers. comm.). In the forest, *P. cinnamomi* has a patchy distribution with most of the valley floors being infested. In the western, high rainfall region of the forest the disease is present in many midslope and some upslope positions. The pathogen has been consistently associated with the death of understorey and overstorey species (Podger, 1972; McDougall, 1998).

P. cinnamomi and the disease it causes in rehabilitated bauxite mines

The mining process involves a defined sequence of events that results finally in a landscaped minesite seeded with a range of plant species similar to those of the adjacent forest . The process starts off with the removal of timber for wood products and firewood. The remaining timber is burnt. The top 5-10 cm of upper organic rich soil or topsoil is removed and immediately spread on to a rehabilitated minepit. This ensures minimal disturbance to seed bank and microbial biomass. The low organic gravel overburden is then removed to stockpiles adjacent to the future minepit or to a nearby minepit that has been mined. The duricrust is then shattered by explosives or bulldozer. This shattered ore is then excavated and transported to a primary crusher where the ore is broken into small

pieces and then transported to the alumina refinery. Once mined, the pit has had 3-5 m of ore removed, it is landscaped to fit the topography of the adjacent non-mined forest. The overburden from stockpiles is returned and then the topsoil. The rehabilitated minepit is then ripped with a winged tine to a depth of 1.2 m on the contour at 2 m spacing in a pattern that ensures no surface water from the pit enters adjacent forest. Ripping encourages the vertical infiltration of rainfall, decreases the risks of surface water ponding, minimizes surface water flow and provides vertical and horizontal cracks in the surface profile to enable roots to penetrate into the soil profile. Once ripped the pit is seeded with seed collected from adjacent forest. The process of mining and rehabilitation has been described in more detail by Colquhoun and Hardy (2000).

The mining process has therefore resulted in a substantial change in the forest. It has removed the duricrust layer and caprock. There has been a major disturbance of the organic rich topsoil and the age of plants is uniform. The disease triangle (Figure 1) outlines how the mining process has changed the host and environment relationships with *P. cinnamomi* and the diseases it causes.

In order to understand how *P. cinnamomi* was killing jarrah in pits, it was decided to sample young trees showing very early symptoms of stress. Symptoms included slight yellowing and purpling of the foliage. Unexpectedly, these trees has lesioned collar tissue at, or just above the soil line. At no time were roots necrotic in the absence of necrotic collars or lignotubers (Hardy *et al.* 1996). These results were in contrast to those from the jarrah forest which clearly show that it is the roots of jarrah that become infested (Shearer and Tippett 1989). It is for this reason we now consider *P. cinnamomi* as a root and collar rot pathogen (Colquhoun and Hardy, 2000). In the forest, it is believed that the pathogen most likely enters through the roots of primary structure or wounds and gradually moves up to lateral roots and the collars (Shearer and Tippett 1989).

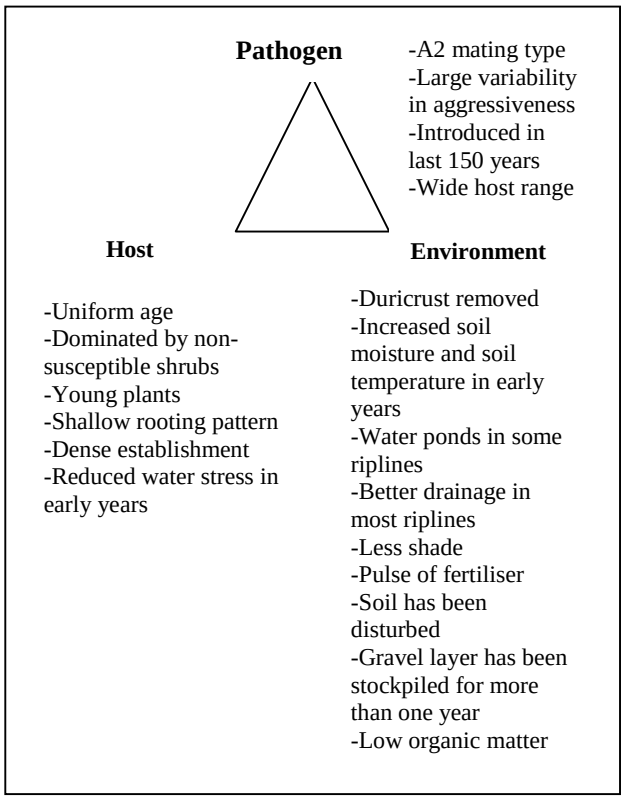

Figure 1. Disease triangle of *Phytophthora cinnamomi* in rehabilitated bauxite mines

Therefore, we (Hardy and Colquhoun, unpublished data) decided to test the null hypothesis that 'in rehabilitated bauxite mines, roots become infected and plants die as a result of the pathogen moving up the roots into the collars and girdle the stem'. A large trial was established in an uninfested minepit. In all, three hundred and sixty large bags which allowed water movement, but prevented large root growth through the bags were filled with minesite soil and buried in the minepit. Two six-month old jarrah seedlings were planted into the bags. At the time of planting three 19 X 600 mm hollow plastic tubes were placed at even spacing vertically into the bags. After 6 months, one seedling was removed from each bag. Approximately, 12 and 18 months after planting (the following spring and autumn) wooden plugs (20 mm x 12-18 mm dia.) colonised with *P. cinnamomi* were placed at different depths in the bags by removing the tubes and inserting the plugs. This ensured that minimal soil disturbance and root damage occurred. Once inoculated the bags were harvested 2, 4, and 8 weeks after inoculation or when symptoms were observed. At harvest, the bags were gently removed from the surrounding soil with a mechanical excavator and the soil around the trees was removed with care by hand to expose roots and plugs, respectively. The types of roots (fine, lateral or tap) which were in contact with the plugs, the presence and absence of lesions and the recovery of *P. cinnamomi* from the roots and plugs were recorded. At no time over 2 years were disease symptoms or the presence of lesions recorded. *P. cinnamomi* was never recovered from any plant material including those roots which were in contact with the inoculum plugs. This was despite *P. cinnamomi* being recovered in all cases from the inoculum plugs for the duration of the trial. Therefore, these observations confirmed our initial survey which indicated that in rehabilitated minepits roots do not readily become infected.

We therefore, instigated a study to determine if zoospores were able to infect and colonise jarrah through the periderm of non-wounded stems under conditions of temporary waterlogging. Trials were conducted in the glasshouse and field which mimicked temporary inundation or ponding of lower stems. This involved placing inoculum receptacles around the stems of trees into which motile zoospore suspensions of *P. cinnamomi* could be were placed. These studies (O'Gara 1999; O'Gara et al. 1996) clearly indicated that 100% of trees inoculated in this fashion became infected and that wounding was not required for the infection process to occur. These studies also showed that only short periods of ponding (2 days) was required for infection to occur. Another study showed that in the field these ponds were never hypoxic (Burgess *et al.*1999a).

In the 1980's a program was established by Alcoa, Murdoch University and CALM to select jarrah trees surviving in 'graveyard' sites. Seeds from these resistant clones were collected, propagated and screened for resistance against isolates of *P. cinnamomi* (Stukely and Crane, 1994). The most resistant seedlings were clonally propagated through tissue culture (McComb et al. 1990; 1992). Ninety-eight clones of jarrah that demonstrate resistance to *P. cinnamomi* in the glasshouse have been tissue cultured. Field trials have been established to check the field resistance of these clones. This involves planting clonal trees on a range of sites and inoculating the soil surrounding each plant with wooded plugs colonised with 4 isolates of *P. cinnamomi.* Since resistance to *P. cinnamomi* is a highly heritable trait (Stukely and Crane 1994), seed orchards have been established to produce plants that are cheaper than the average cost of US$4 a clonal plant.

However, despite the success of the clonal jarrah program we still had concerns about the long-term survival of these clones in PRCP sites. These concerns were raised from our observations that there is a large variation in the pathogenicity of isolates of *P. cinnamomi* with some isolates being significantly more pathogenic that the isolates which were originally used to screen for resistance. Our concerns were also increased by the observation that temporary ponding in certain rehabilitated pits in the presence of the pathogen resulted in stem not root infection. We also felt that temporary waterlogging and/or hypoxia might predispose the resistant clones to PRCR. We also wanted to determine whether older plants (6-7 years old) remained resistant to the pathogen, since all the earlier screenings had been conducted on approximately one-year-old plants. Finally, we wanted to confirm earlier observations that there was still a good

correlation between resistance in plants inoculated in the stems compared to root inoculation. Original selection of resistant clones had been based on underbark inoculation of stems in seedling jarrah that had been shown to compare favourably with root inoculation.

With our early field trial to determine how jarrah were becoming infected in minepits we had collected a large range of *P. cinnamomi* isolates from diseased and dying jarrah and marri trees (Hardy et al. 1996). Initial morphological studies indicated that isolates from marri and jarrah were slightly different from each other in oogonial and sporangial characteristics. However, this was not found to be the case although we did find that *P. cinnamomi* was able to produce multiple paragynous antheridia in addition to the amphigynous antheridium (Hüberli et al. 1997a; 1997b).

The pathogenicity of 84 isolates form marri and jarrah was then assessed in a number of trials. (i) The 84 isolates were screened on one year-old excised marri and jarrah stems over 3 days under controlled laboratory conditions. The results indicated that the isolates were equally pathogenic on marri or jarrah, regardless of their original host (Hüberli , Tommerup and Hardy, unpublished). However, the isolates did vary significantly in pathogenicity. (ii) All 84 isolates were then screened for pathogenicity in an evaporatively cooled glasshouse inoculation trial of a resistant jarrah clone (6 replicate plants per isolate). (iii) Ten isolates ranging in pathogenicity (4, 4 and 2 isolates highly, moderately and weakly pathogenic, respectively) were then screened on 2 resistant and 1 susceptible clone in climatically controlled phytotrons under a range of temperature regimes (15, 20, 25 and 32 °C) with plants either underbark inoculated or inoculated with zoospores. (iv) In the field, a range of 1 and 6 year old clonal plants of varying resistance (4 RR and 2 SS clones and seedlings) where underbark inoculated with one highly pathogenic and one weakly pathogenic isolate of *P. cinnamomi* selected from the previous trial. (v) Finally, we examined *in planta* the relationship between lesion development on roots and stems of resistant and susceptible clonal trees after underbark inoculation (Hüberli , unpublished).

Plants were either underbark inoculated or inoculated with zoospores using the methodology of O'Gara et al. (1996). Briefly, plants were underbark inoculated with *P. cinnamomi* colonised Miracloth (Calobiochem, USA) discs (5 mm^2 in glasshouse studies or 5 cm^2 in field studies), whilst motile zoospores were placed into plastic receptacles on the collars of plants. There was a large variability in the pathogenicity of the 84 isolates when excised stems of jarrah were inoculated and harvested 3-4 days later. This observation was repeated when the single clonal line of 18 month-old jarrah was underbark inoculated in stems *in situ*. However, there was not a good correlation in pathogenicity between isolates inoculated into excised stems and those inoculated *in planta* with zoospores or colonised Miracloth discs. There was, however, a good correlation between zoospore inoculation and underbark inoculation. In the glasshouse inoculation trial, the most pathogenic isolate killed all trees within 5 weeks. In contrast, the least pathogenic isolate failed to kill any plants during the 26 week trial period. In the temperature controlled phytotron trial the two resistant clones died as rapidly as the susceptible clone at 28 °C, whilst at 20 °C the susceptible clone died significantly faster than the resistant clones (Hüberli et al. 1998). In the 1 and 6 year-old field trials, one resistant clone was more susceptible than the susceptible clones, whilst seedling trees were more susceptible than the *P. cinnamomi* susceptible clones. However, no differences were observed in pathogenicity between the two isolates. To date there does not appear to be a difference in susceptibility between the 1 year old and 6 year old clonal plants. Finally, a good relationship was found between lesion development in the stems and roots of the jarrah plants. This was heartening as it continues to justify the use of stems to screen for resistant jarrah plants.

Our work has also shown that there is a large variation in the pathogenicity between isolates of *P. cinnamomi* (O'Gara *et al.* 1997; Hüberli *et al.* 2000). It appears that host-environment interactions influence the pathogen as to whether it will express itself as a biotroph or a necrotroph. We have also shown that measuring lesion lengths as a means of determining pathogenicity is inappropriate. Often an isolate under a given set of conditions (for example, temperature and host water status) can colonise the host extensively with only a very small lesion apparent. Another isolate under the same set of conditions can produce large lesions and little colonisation of tissue beyond the lesion. The former isolate in the past might have been considered less pathogenic than the latter. Yet under field conditions be the more aggressive of the two. This observation has management implications. For example, the screening process used to select resistant jarrah for clonal production has only used a few isolates. These isolates are intermediate in their pathogenicity, as a consequence the selection process has not been rigorous enough. At present we are trying to elucidate how the host and the environment interact with a range of isolates of *P. cinnamomi* to influence the expression of the pathogen as a necrotroph or biotroph. It is apparent that isolates of *P. cinnamomi* are extremely 'plastic' in their ability to express themselves as pathogens. This plasticity varies considerably with changes in the host and the environment, and it is this characteristic that probably helps account for the pathogen having such a wide host range across the lower south-west of Western Australia and elsewhere. Essentially, conditions under which one isolate is pathogenic another might not be pathogenic, however, as conditions change their ability to act as pathogens might change. Tommerup et al (2000) discuss the genetics and inheritance of these traits and their implications.

An aeroponics system was developed which allowed the roots of clonal or seedling plants to be readily subjected to hypoxia (2 mg O_2 l^{-1}) or anoxia. The system allows for the roots and stems to be easily accessible for inoculation with zoospores, non-destructive monitoring and harvest for enzyme and histological studies (Burgess and Hardy, 1996; Burgess et al. 1997; 1998). When plants were inoculated in the stems using the inoculum receptacle technique of O'Gara et al. (1996), colonisation by the pathogen extended further up the stems in a resistant clone than in a susceptible clone subjected to hypoxia rather than normal oxygen levels (Burgess et al. 1999b). This result was surprising as a much higher concentration of soluble phenolics leached into the inoculum receptacles from the resistant compared to the susceptible clones. Neither the duration of root hypoxia nor the timing of root hypoxia in relation to the ponding affected the final extent of stem colonisation by *P. cinnamomi*. If root hypoxia was imposed before inoculation the plants were stessed and the pathogen entered the stem rapidly; if hypoxia was imposed after ponding and inoculation, then the pathogen, which was present on the stem, was able to take advantage of the induced stress and penetrate rapidly

The activity of enzymes involved in the phenylpropanoid pathway (PAL, 4-Cl, CAD) were monitored in plants that were subjected to hypoxia or normal oxygen conditions in the absence or presence of the pathogen (Burgess et al. 1999c). Root hypoxia induced activity of these enzymes and their products in stems of the plants. Colonisation by *P. cinnamomi* further increased the activity of these enzymes. Actual levels of enzyme activity were higher in plants exposed to hypoxia, however, the relative increase in enzyme activity in response to the pathogen was greater in control plants grown under normal conditions. Peroxidase induction appeared to reflect tissue damage rather that plant defence. Overall, plants subjected to hypoxia were less able to switch on rapid defence responses against the pathogen.

Another study that also used the aeroponics system, examined the effects of varying periods of hypoxia (0, 2, 5, 11 or 29 days) on the roots of a resistant *E. marginata* clone before being inoculated with zoospores of *P. cinnamomi.* A similar set of roots was inoculated 3 days after the hypoxia treatments. All hypoxia treatments reduced root extension. However, 6 days after the hypoxia treatments, root extension had returned to normal for roots that had been exposed to 5 days of hypoxia, while for roots exposed to 11 or 29 days, extension was half the normal rate. Exposure to hypoxia for 5, 11 or 29 days was shown to reduce cell division, but not cell expansion. In the case of roots exposed to 3 days of hypoxia, the apical meristem appeared

normal at the end of the treatment, but 3 days after the return to normal oxygen conditions many of the apical meristems had died. Thus, *E. marginata* roots have an acclimatization period to hypoxia of between 2 and 5 days, after which they can tolerate hypoxia for extended periods. (Burgess et al. 1999c). Root extension ceased when a lesion developed. A hypoxic pretreatment effected lesion development posthypoxia by reducing the number of roots that became infected and any lesions that developed were significantly ($P<0.05$) smaller than those that developed under normal oxygen conditions. Six days after the resumption of normal oxygen conditions, lesion development in roots exposed to 5 days of hypoxia had returned to normal, while for roots that had been exposed to 2, 11 or 29 days of hypoxia, the lesions that developed were still significantly smaller. Therefore, root tips of jarrah exposed to hypoxia and then returned to normal oxygen conditions were more resistant to invasion by *P. cinnamomi* than roots grown under normal oxygen conditions. The study suggested that *P. cinnamomi* was only able to enter roots and infect through actively growing apices.

Current Research Activities

At present, we have a number of other studies in place. One study is determining the effects of drought on disease development after infection. Previous studies have shown that once bark moisture drops, *E. marginata* is able to contain lesion development (Tippett and Hill 1983; Tippet et al. 1987 ; Bunny et al. 1995). There is some concern that if the south-west of Western Australia starts to experience summer rainfall events, a predicted result of the greenhouse effect, then we will experience more deaths of jarrah. The study involves subjecting 2 year old clonal jarrah resistant or susceptible to *P. cinnamomi* to continuous watering, withholding water over summer (drought), or mimicking a summer rainfall event after inoculation with *P. cinnamomi* in spring. Plants are being harvested monthly over 2 years and rated for disease status. If continuous watering or summer rainfall events do increase deaths in jarrah on rehabilitated mines, it will be necessary to develop strategies to reduce this impact.

Another study is examining the long-term survival of *P. cinnamomi* in rehabilitated mines and adjacent jarrah forest. It is looking at saprophytic ability, potential exogenous and endogenous dormancy of chlamydospores and the effects of environmental factors on survival. Outcomes of this trial will be helpful in reducing the spread of *P. cinnamomi* in stockpiled topsoil or during the movement of soil and vehicles during the mining process.

Monitoring of rehabilitated bauxite mines for plant deaths caused by *P. cinnamomi* has indicated that deaths are less prevalent in areas where *Acacia* species are abundant. In the jarrah forest it has been observed that understorey dominated by *Acacia* species significantly changes the soil environment to one with a greater suppressive effect on the population levels of the pathogen (Shearer and Tippett, 1987). Therefore, we are examining the impact of a range of *Acacia* species on disease impact, sporangial production and zoospore release and survival of *P. cinnamomi* in rehabilitated minesite soils in the pits and in glasshouse studies.

The impact of *P. cinnamomi* is a major concern to the management of natural plant communities and rehabilitated communities during and after mining in Western Australia. This concern arises from the fact that this introduced pathogen has a wide host range which includes jarrah, the dominant tree species of the jarrah forest and many other understory species that make up key structural components of the forest. In order to effectively manage all the structural components of the vegetation in rehabilitated mines it is important to understand the dynamics of the pathogen and ascertain how this differs from the adjacent forest. This knowledge will assist managers to effectively reduce the impacts of *P. cinnamomi* in natural plant communities.

ACKNOWLEDGEMENTS

I would like to thank Emer O'Gara, Keith McDougall, Daniel Hüberli, Sarah Collins and Anne Lucus whose work has been mentioned in this paper, and Ian Colquhoun, Inez Tommerup and Bryan Shearer for their support and input over the years.

REFERENCES

Abbott, I., and Loneragan, O. W. (1986). Ecology of jarrah (*Eucalyptus marginata*) in the northern jarrah forest of Western Australia. Department of Conservation and Land Management, Perth, Australia. Bulletin No. 1.

Bell, D. T., and Heddle, E. M. (1989). Floristics, morphology and vegetation diversity. Pages 53-66 in: The Jarrah Forest. A Complex Mediterranean ecosystem. B. Dell, J. J. Havel and N. Malajczuk eds., Kluwer Academic Publishers, The Netherlands.

Bunny, F. J., Crombie, D. S. and Williams, M. R. (1995). Growth of lesions of *Phytophthora cinnamomi* in stems and roots of jarrah (*Eucalyptus marginata*) in relation to rainfall and stand density in mediterranean forest of Western Australia. *Canadian Journal of Forest Research* **25,** 961-969.

Burgess, T., and Hardy, G. E. St. J. (1996). Effect of low oxygen on peroxidase activity in roots of clonal *Eucalyptus marginata* infected with *Phytophthora cinnamomi*. Phytopathology 86:S102 (Abstract).

Burgess, T., McComb, J. A., and Hardy, G. St. J. (1997). Responses of *Eucalyptus marginata* to simulated waterlogging and *Phytophthora cinnamomi* infection. Acta Hortic. **447**, 401-406.

Burgess, T., McComb, J., Hardy, G. (1998). The use of aeroponic chambers to study the influence of low oxygen levels on root and lesion development of *Eucalyptus marginata* Donn. Ex Smith infected with *Phytophthora cinnamomi* Rands. Plant Dis. 82:368-373.

Burgess, T., Collins, S., Hardy, G. E. St. J., Colquhoun, I. J., and McComb, J. A. (1999a). A survey of the frequency, duration and oxygen content of surface and sub-surface water in rehabilitated mined areas at Huntly mine. Alcoa World Alumina Australia Research Bulletin No. 29, Applecross, Australia.

Burgess, T., Hardy, G.E.St.J., McComb, J.A., and Colquhoun, I. (1999b). Effects of hypoxia on root morphology and lesion development in *Eucalyptus marginata* infected with *Phytophthora cinnamomi*. *Plant Pathology* **48,** 786-796.

Burgess, T., McComb, J.A., Colquhoun, I. and Hardy, G.E.St.J. (1999c). Increased susceptibility of *Eucalyptus marginata* to stem infection by *Phytophthora cinnamomi* resulting from root hypoxia. *Plant Pathology* **48,** 797-806.

Colquhoun, I. J. and Hardy, G. E. St J. (2000). Managing the risks of *Phytophthora* root and collar rot during bauxite mining in the *Eucalyptus marginata* (jarrah) forest of Western Australia *Plant Disease* **84,**

Davison, E. M., and Shearer B. L. (1990). *Phytophthora* spp. in indigenous forests in Australia and New Zealand. N.Z. J. Forest Science **19**; 277-289.

Hardy, G. E. St. J., Colquhoun, I. J., and Nielsen, P. (1996). The early development of dieback disease in *Eucalyptus marginata* and *Eucalyptus calophylla* growing in rehabilitated bauxite mined areas. Plant Path. **45**; 944-955.

Hardy, G. E. St J., Colquhoun, I. J. and Shearer, B. L. (2000). This publication

Hüberli, D., Hardy, G. E. St J. and Tommerup, I. C. (1997a). Morphological and physiological characteristics of two *Phytophthora cinnamomi* populations *Eleventh Biennial Australasian Plant Pathology Society Conference* Perth. p. 307.

Hüberli, D., Tommerup, I. C. and Hardy, G. E. St J. (1997b). The role of paragynous and amphigynous antheridia in sexual reproduction of *Phytophthora cinnamomi*. *Mycological Research* **101,** 1383-1388.

Hüberli, D., Tommerup, I. C., Hardy, G. E. St J. and Colquhoun, I. J. (1998). Temperature changes resistance of *Eucalyptus marginata* to *Phytophthora cinnamomi*. *Seventh International Conference of Plant Pathology*, Edinburgh, Scotland. p. 2.5.4.

Hüberli, D., Tommerup, I. C. and Hardy, G. E. St J. (2000). False-negative isolations or absence of lesions may cause misdiagnosis of *Phytophthora cinnamomi* diseased plants *Australasian Plant Pathology* (in press)

Keighery, G. (1992). The impact of *Phytophthora* species on rare plants. Pages 29-36 in: Dieback – What is the future? M.J. Freeman, R. Hart, and M. Ryall, eds. Northern Sandplain Dieback Working Party, Perth, Australia.

Kinal, J. (1986). Perching and throughflow in a lateritic profile in relation to the impact of *Phytophthora cinnamomi* in the northern jarrah forest. Honours thesis, Murdoch University, Western Australia.

Kinal, J., Fairman, R. G. and Shearer, B. L. (1987). Perching and the throughflow in relation to the impact of *Phytophthora cinnamomi* in the jarrah forest. Abstract 20. Proceedings of the Australasian Plant Pathology Society Sixth National Conference. University of Adelaide, South Australia.

McDougall, K. L. (1996). Vegetation patterns in the northern jarrah forest of Western Australia in relation to dieback history and the current distribution of *Phytophthora cinnamomi*. Murdoch University, **1996 McComb, J.A., Bennett, I.J., Cahill, D., Stukely, M., and Crane, C.** (1992). Selection, propagation, field and laboratory testing of jarrah (*Eucalyptus marginata*) resistant to dieback (*Phytophthora cinnamomi*). IUFRO/AFOCEL conference on 'Mass production technology for genetically improved fast growing tree species'. Bordeaux, France. **2;** 451-452.

McComb, J. A, Bennett, I., Stukely, M and Crane, C. E. (1990). Selection and propagation of jarrah for dieback resistance. *Combined Proceedings of the International Plant Propagators' Society* **40,** 86-90.

O'Gara, E. (1999). Infection and disease of *Eucalyptus marginata* (jarrah), caused by *Phytophthora cinnamomi* in rehabilitated bauxite mines in the south-west of Western Australia. PhD thesis, Murdoch University, Murdoch, WA, Australia.

O'Gara, E., Hardy, G. E. ST. J., and McComb, J. A. (1996). The ability of *Phytophthora cinnamomi* to infect through unwounded and wounded periderm tissue of *Eucalyptus marginata*. Plant Path. **45**; 955-963.

O'Gara, E., McComb, J. A., Colquhoun, I. J. and Hardy, G. E. St. J. (1997). The infection of non-wounde and wounded periderm tissue at the lower stem of *Eucalyptus marginata* by zoospores of *Phytophthora cinnamomi*, in a rehabilitated bauxite mine. *Australasian Plant Pathology* **26,** 135-141.

Podger, F. D. (1972). *Phytophthora cinnamomi*, a cause of lethal disease in indigenous plant communities in Western Australia. *Phytopathology* **62,** 972-981.

Shea, S. R., Shearer, B. L., Tippett, J. T. and Deegan, P. M. (1983). Distribution, reproduction and movement of *Phytophthora cinnamomi* on sites highly conducive to jarrah dieback in south western Australia. *Plant Disease* **67,** 970-973.

Shearer, B. L., and Shea, S. R. (1987). Variation in seasonal population fluctuations of *Phytophthora cinnamomi* within and between infected *Eucalyptus marginata* sites of south western Aust. For. Ecol. Man. **21**; 209-230.

Shearer, B. L., and Tippett J. T. (1989). Jarrah Dieback: The Dynamics and Management of *Phytophthora cinnamomi* in the jarrah (*Eucalyptus marginata*) Forests of South-Western Australia. Department of Conservation and Land Management, Perth, Australia. Research Bulletin No 3.

Shearer, B. L. (1990). Dieback of native plant communities caused by *Phytophthora* species - a major factor affecting land use in south-western Australia. *Land and Water Research News***,** 15-26.

Stukely, M. and Crane, C. E. (1994). Genetically based resistance of *Eucalyptus marginata* to *Phytophthora cinnamomi*. *Phytopathology* **84,** 650-656.

Tippett, J. T., Crombie, D. S. and Hill, T. C. (1987). Effect of phloem water relations on the growth of *Phytophthora cinnamomi* in *Eucalyptus marginata. Phytopathology* **77,** 246-250 .

Tippett, J. T. and Hill, T. C. (1983). The relationship between bark moisture and invasion of *Eucalyptus marginata* by *Phytophthora cinnamomi*. *Australasian Plant Pathology* **12,** 40-41.

Tommerup, I. C., Dobrowolski, M.P., Hüberli, D., O'Brien, P.A., Hardy, G. E. St. J. and Shearer, B.L. (2000). Risks and threats due to genetic variation in *Phytophthora cinnamomi* for disease management in natural vegetation ecosystems. This proceedings.

Wills, R. T. (1993). The ecological impact of *Phytophthora cinnamomi* in the Stirling Range National Park, Western Australia. *Australian Journal of Ecology* **18**, 145-159.

Zentmyer, G. A. (1980). *Phytophthora cinnamomi* and the diseases it causes. *American Phytopathological Society Monograph* **No. 10**, St. Paul MN, 96 pp.