Distribution, Reproduction, and Movement of *Phytophthora cinnamomi* on Sites Highly Conducive to Jarrah Dieback in South Western Australia

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ABSTRACT

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Phytophthora cinnamomi, which causes death of *Eucalyptus marginata* and other species in the jarrah forest of south Western Australia, was recovered at a high inoculum density as deep as 75 cm in soil immediately above a concreted lateritic layer. It was shown that the fungus can produce zoospores in soil at the interface of the lateritic layer and that zoospores are transmitted laterally in water flowing at the surface of the layer on sites of decline. It is proposed that the rapid mass decline and death of jarrah that has been observed is a consequence of these specific site characteristics, which allow infection and destruction of the vertical root system of jarrah by *P. cinnamomi*.

Phytophthora cinnamomi (Rands) has caused extensive mortality of jarrah (*Eucalyptus marginata* Sm.) and a large proportion of shrub and understory components of the jarrah forest in south

Western Australia over an area in excess of 200,000 ha (5). Rapid decline and death of jarrah trees occurred primarily in 1945–1965, although disease extension as measured by death of the highly susceptible *Banksia grandis* (Willd.) understory has occurred in many areas of the forest since that period.

P. cinnamomi is a soilborne or waterborne fungus believed to be of tropical or subtropical origin. It is now widely distributed in temperate, sub-tropical, and tropical regions of the world and has been recovered from 967 plant species (12). The pathogen was comparatively recently introduced to south Western Australia (5). It requires warm (21–31 C) and wet (−0.025 to −0.03 bars) soil conditions to form sporangia and requires free water for release and transmission of zoospores (12). Dry soil conditions reduce survival (8). Several studies have shown that its population density in soil decreases with depth (12).

South Western Australia has a Mediterranean climate and the jarrah forest occurs on an extensively laterized peneplain (4). In poorly drained moisture-gaining sites on valley floors, soil moisture levels remain above critical levels for survival (6) and the fungus can be detected in the soil surface horizons throughout the year (B. L. Shearer and S. R. Shea, *unpublished*). Although most of these sites within the forest have been infected, the impact of the disease is limited because a number of the shrub and understory and overstory species are

resistant to *P. cinnamomi*.

Jarrah is the dominant component of the overstory on ridges and divides, which constitute about 80% of the landscape. The soils are deeply weathered infertile laterites (Fig. 1) (4). The surface horizons of these soils are suitable for sporangial formation and release only during relatively brief periods of warm wet conditions in autumn and spring (6). Overland flow of water is insignificant unless there is disturbance; therefore, lateral transmission of zoospores in the surface horizons on these sites is negligible. During the prolonged summer dry period, water potentials of surface soil layers are less than (−10 bars) and survival of *P. cinnamomi* is negligible (8).

Although the surface soil environment on upland lateritic sites is only marginally favorable for *P. cinnamomi*, severe disease occurs (Fig. 2). Distribution of *P. cinnamomi* in time and space in surface (0−10 cm) soil horizons was monitored at a number of diseased jarrah sites for several years, but recovery rates from random soil samples rarely exceeded 10% and were usually less than 3% (8).

It was assumed that *P. cinnamomi* attacks the fine roots of susceptible *Eucalyptus* species and other hosts in the surface soil horizons (5,8,12). We were unable to explain how this pathogen can cause rapid and extensive mortality of jarrah trees by fine feeder root attrition because its population in the surface soil horizons of lateritic soils is so limited. *B. grandis*, which forms a dense understory in many areas of the forest, is highly susceptible to *P. cinnamomi* and is rapidly killed by the fungus (7,8). We have demonstrated consistent and rapid (0.5–1.4 cm per day) invasion of the xylem and phloem of the major roots and lower stems of this species (S. R. Shea and P. M. Deegan, *unpublished*). Although *P. cinnamomi* can also invade the major secondary roots and lower stems of jarrah trees (10), the absence of extensive and rapid mortality of jarrah in large areas of infected forest indicated that jarrah trees normally resist invasion of the major roots.

In a subsequent study of affected trees growing in areas where mass jarrah decline and death were occurring, it was found that *P. cinnamomi* caused death of the vertical roots (Fig. 1) where they passed through root channels in a concreted lateritic layer (9). In all sites where mass decline was observed, the concreted layer was within 1 m of the soil surface. We therefore proposed that rapid decline and death of jarrah trees resulted from destruction of the vertical root system by *P. cinnamomi* at depth in the soil profile on these specific sites. In this paper, we present preliminary results from studies of the distribution, reproduction, and movement of *P. cinnamomi* on these sites of rapid and mass decline and death of jarrah trees.

MATERIALS AND METHODS

Five plots measuring 20 × 20 m were located in an area of forest where rapid decline and death of the jarrah overstory, understory, and shrub layer were occurring. One similar plot measuring 20 × 20 m was established in adjacent healthy forest. The plots were sampled three times at about 3-wk intervals after winter rains began in May 1982. In each plot, soil from 0 to 10 cm deep surrounding the collars of 10 recently killed (leaves retained) *B. grandis* trees was sampled on each date. One soil sample was taken from each *Banksia* and processed separately. In addition, the surface (0−10 cm) horizon and the soil at the interface of the concreted lateritic layer was sampled with a heavy-gauge 40-mm-diameter pipe driven into the soil to the laterite layer at 10 random points in each plot on each sampling date. The depth of the concreted layer was recorded at each sampling point. The samples were returned to the laboratory and processed immediately.

From each sample of about 100 cm^3, 5 g of soil that passed through a 2-mm sieve was placed in a glass petri dish to determine soil moisture content expressed as a percent of oven-dry weight. Of the remaining sieved soil, 25 g was mixed with 50 ml of distilled water and poured on a 15-cm-diameter petri dish containing selective (11) half-strength potato-dextrose agar. The plates with soil were incubated for 2 days in darkness at 24 C. After incubation, soil was washed from the plates and the colonies of *P. cinnamomi* were counted. The plates were further incubated for 1 day and the colonies recounted. The number of colonies per gram oven-dry weight of soil was determined by calculating the oven-dry weight of soil applied per plate from moisture-content determination.

Six weeks after autumn rainfall began, a plot measuring 12 × 8 m was established in the severely diseased area. Soil samples were taken at 10-cm depth intervals to the laterite layer at each intersection of a 1-m-square grid. After this systematic sampling, the surface layer of the soil was removed and the soil in the root channels sampled at about 1-m intervals along the channels. There were 50 samples, each containing about 100 cm^3 of soil. Soil
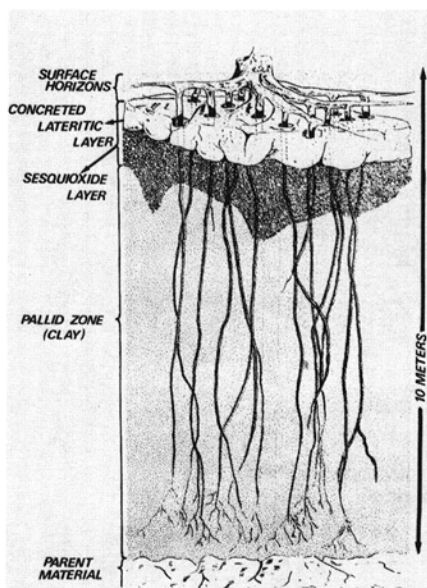

Fig. 1. Diagrammatic representation of a jarrah tree root system. Blackened areas of roots at the surface of the concreted layer show infection by *Phytophthora cinnamomi*. Roots below the concreted lateritic layer were not sampled.

SURFACE HORIZONS

CONCRETED LATERITIC LAYER

SESQUIOXIDE LAYER

PALLID ZONE (CLAY)

10 METERS

PARENT MATERIAL

Fig. 2. Jarrah forest showing mass rapid decline and death caused by *Phytophthora cinnamomi*.

samples were assayed for *P. cinnamomi* as described before, then the soil was washed from the root channels.

Sporangial formation in the surface soil (1–3 cm) and at the surface of the concreted lateritic layer (2.5–60 cm) was determined by counting sporangia formed on inoculated *B. grandis* leaf disks that had been buried in the surface soil and at the interface with the concreted lateritic layer. For this procedure, disks 4 mm in diameter were punched from new-season *B. grandis* leaves and autoclaved in distilled water for 20 min at 1.05 kg/cm². Fifty disks were suspended in 10 ml of sterile V-8 juice (3), inoculated with 2 ml of a comminuted mycelial suspension, and incubated at 24 C in darkness for 2 days. After incubation, disks with peripheral mycelium were rinsed three times in distilled water and placed between 1-cm² pieces of fine gauze, three disks per square. The squares and disks were placed into gauze envelopes that were inserted in the soil. Five series of disks (about 200 envelopes per series) were inserted during the 2 mo after autumn rains began in May. The disks were removed 7–14 days after insertion and stained and mounted, then the number of sporangia formed per disk was counted.

The capacity for lateral transmission of zoospores was determined by sampling soil at the interface with the lateritic layer 75 cm downslope of trenches measuring 30 × 30 cm that had been dug to the depth of the lateritic layer and had each received 2 L of water containing about 10⁷ zoospores. Zoospores were produced by growing mycelium in sterile pea broth and transferring this to 20% soil extract for several days, then chilling for 30 min. Five trials were carried out on sites with concreted laterite present and the procedure was repeated on adjacent sites where the lateritic layer was not present. Each point of zoospore introduction was matched with a plot that contained no inoculum. The soil downslope of the introduction point at the interface with

the lateritic layer was sampled 24 hr after inoculation and the presence of *P. cinnamomi* was determined by the direct-plating procedure described earlier.

RESULTS

P. cinnamomi was detected four to eight times more frequently in soil at the interface of the concreted lateritic layer than in the surface layer (Table 1). The detection rate around the collar of recently killed *B. grandis* trees was lower (6%) than that in random surface soil samples. Among samples containing *P. cinnamomi*, there was no significant difference between the density of propagules in surface soils and at greater depths. *P. cinnamomi* was not recovered from the control plot. The depth distribution of *P. cinnamomi* in the plot sampled on a systematic grid was similar to that described earlier (Figs. 3 and 4). The detection rate in soil samples taken from the root channels, however, was higher than in samples from other areas

on the cap rock. Removal of soil from this plot revealed numerous situations where *B. grandis* roots occupied the same root channels as jarrah.

Sporangial formation on *Banksia* leaf disks was high during autumn (May) but declined as soil temperatures decreased. There was no significant difference ($P = 0.01$) between the number of sporangia formed in the surface soil and at the interface of the concreted lateritic layer. Sporangia formed as deep as 60 cm at the interface of the lateritic layer as shown in Figure 5, which represents the pattern of sporangial formation recorded from one of the series of disks inserted in June.

The fungus was consistently recovered from soil samples at the interface of the concreted lateritic layer at distances of 50–70 cm downslope of the zoospore introduction points 24 hr after inoculation. On sites where the concreted layer was not present, the fungus was detected in soil vertically below the introduction

Table 1. Detection of *Phytophthora cinnamomi* in random surface and subsurface soil samples from an area undergoing mass decline of jarrah

Depth (cm)	No. samples	Recovery of *P. cinnamomi* (%)	No. propagules/g dry soil
Surface samples			
<5	150	10	0.84[a] (0.36–1.95)[b]
Samples from interface with concreted lateritic layer			
<12	35	40	0.96[a] (0.53–2.69)[b]
<22	36	50	1.03 (0.87–2.73)
<32	22	68	0.69 (0.50–2.73)
<42	25	80	1.00 (0.85–2.72)
<52	17	71	0.50 (0.33–1.65)
<62	8	88	1.14 (0.20–1.65)
Mean	147	61	1.13

[a] Mean of positive samples.
[b] Asymmetric confidence interval of 95%.
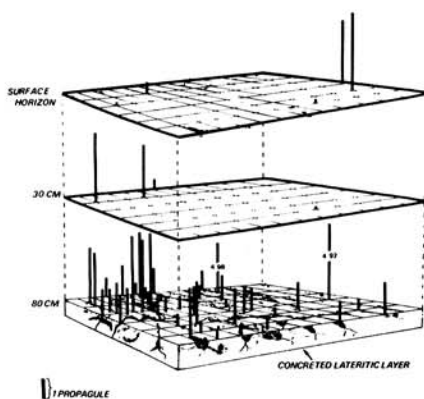
Fig. 3. Distribution of *Phytophthora cinnamomi* in soil above a concreted lateritic layer on a site of severe disease. Vertical bars represent propagule numbers per gram of oven-dry soil.
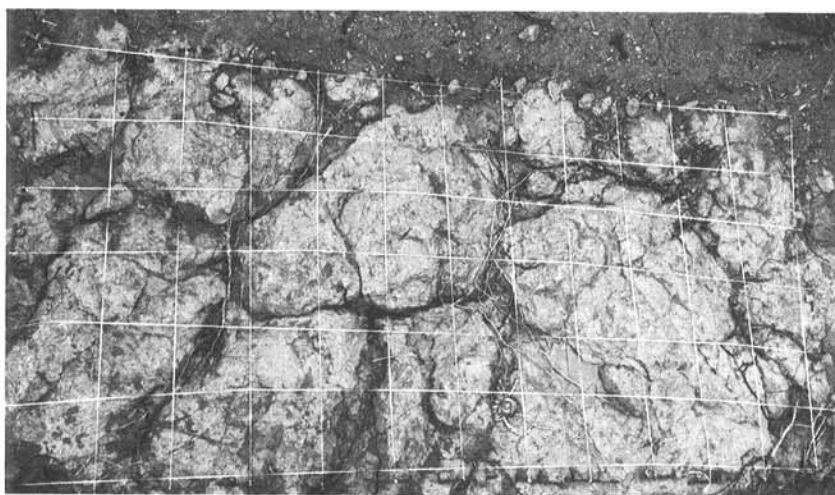
Fig. 4. Exposed layer of concreted sheet laterite forming the base of the plot shown in Figure 3. Tape denotes meter square sampling grid.
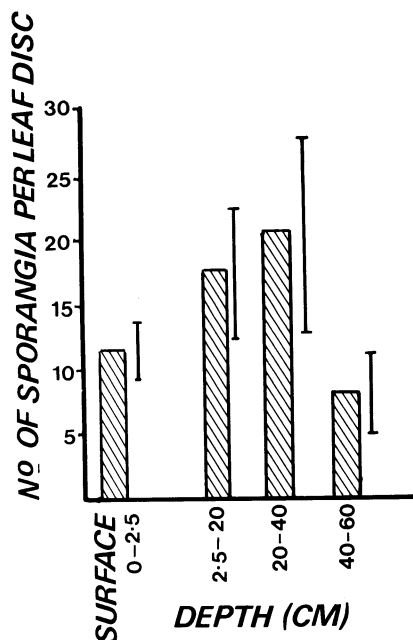
Fig. 5. Sporangia formed on *Banksia grandis* leaf disks in the surface soil horizon and at the interface of the concreted lateritic layer 14 days after insertion in June 1982. Bars indicate 95% confidence limits.

point but not downslope. No recoveries of *P. cinnamomi* were recorded from controls. In a separate trial, zoospores were detected in water flowing at the interface of a concreted lateritic layer 30 cm below the soil surface, 2 m downslope from a trench that had been filled with water and inoculated with a zoospore suspension.

DISCUSSION

It has been difficult to explain the ability of *P. cinnamomi* to cause rapid decline and death of jarrah forests on apparently free draining sites in a region that has a Mediterranean climate. The discovery that *P. cinnamomi* can occur at high densities in the soil below the surface horizons where a layer of concreted laterite is present and that zoospores of the fungus can be laterally transmitted at the interface of this layer partially resolves this question. This study, together with our previous report (9) of infection of the vertical root system of jarrah trees by *P. cinnamomi*, forms the basis for an explanation of how *P. cinnamomi* can cause mass collapse of a jarrah forest.

Jarrah trees, which maintain a high rate of transpiration throughout the summer months (1,2), depend on their vertical root system to maintain internal water balance during the extended period of annual drought. On specific site types, the presence of a concreted layer of sheet laterite in the soil profile provides conditions favorable for reproduction and lateral movement of *P. cinnamomi* at the surface of the layer. Vertical roots pass through the concreted lateritic layer via root channels, where they commonly form fine roots. Vertical roots of the highly susceptible *B. grandis* understory (7) often occur in the same root channels. The presence of infected *B. grandis* roots, and the fact that root channels form depressions in the lateritic layer that accumulate water, cause zoospores to be concentrated around the vertical roots. Only relatively small extensions of *P. cinnamomi* into the vertical roots via fine roots formed in the root channels are necessary to cause death of the root at its junction with the laterite layer. Death of vertical roots results in the cessation of water uptake from deep sources and desiccation of the trees.

Preliminary surveys indicate that a large proportion of the forest area where mass decline occurred in 1947–1965 had soil profile characteristics similar to those we have described (S. R. Shea, *unpublished*). It is possible that large areas of forest that do not have these soil characteristics are less susceptible to *P. cinnamomi* than has previously been assumed.

LITERATURE CITED

1. Doley, D. 1967. Water relations of *Eucalyptus marginata* Sm. under natural conditions. Ecology 55:597-614.
2. Kimber, P. C. 1974. The root system of jarrah (*Eucalyptus marginata*). West. Aust. For. Dep. Res. Pap. 10. 3 pp.
3. Mircetich, S. M., Zentmyer, G. A., and Kendrick, J. B., Jr. 1968. Physiology of germination of chlamydospores of *Phytophthora cinnamomi*. Phytopathology 58:666-671.
4. Mulcahy, M. J., Churchward, H. M., and Dimmock, G. M. 1972. Landforms and soils on an uplifted peneplain in the Darling Range, Western Australia. Aust. J. Soil Res. 10:1-14.
5. Podger, F. D. 1972. *Phytophthora cinnamomi*, a cause of lethal disease in indigenous plant communities in Western Australia. Phytopathology 62:972-981.
6. Shea, S. R. 1975. Environmental factors of the northern jarrah forest in relation to pathogenicity and survival of *Phytophthora cinnamomi*. West. Aust. For. Dep. Bull. 85. 83 pp.
7. Shea, S. R. 1979. *Phytophthora cinnamomi* (Rands). A collar rot pathogen of *Banksia grandis* Willd. Aust. Plant Pathol. 8:33-34.
8. Shea, S. R., Gillen, K. J., and Leppard, W. I. 1980. Seasonal variation in population levels of *Phytophthora cinnamomi* Rands in soil in diseased, freely-drained *Eucalyptus marginata* Sm. sites in the northern jarrah forest of south-western Australia. Prot. Ecol. 2:135-156.
9. Shea, S. R., Shearer, B. L., and Tippett, J. 1982. Recovery of *Phytophthora cinnamomi* Rands from vertical roots of Jarrah (*Eucalyptus marginata* Sm.). Australas. Plant Pathol. 11:25-28.
10. Shearer, B. L., Shea, S. R., and Fairman, R. G. 1981. Infection of the stem and large roots of *Eucalyptus marginata* by *Phytophthora cinnamomi*. Australas. Plant Pathol. 10:2-3.
11. Tsao, P. H., and Guy, S. O. 1977. Inhibition of *Mortierella* and *Pythium* in a *Phytophthora*-isolation medium containing Hymexazol. Phytopathology 67:796-801.
12. Zentmyer, G. A. 1980. *Phytophthora cinnamomi* and the Diseases it Causes. Monogr. 10. American Phytopathological Society, St. Paul, MN. 96 pp.