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Phytophthora cinnamomi: Population Densities in Forest Soils

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Abstract

Population densities of *Phytophthora cinnamomi*, associated disease and environmental factors were studied concurrently during a 2-year period in three different forest ecosystems. Pathogen populations showed seasonal variation, low values being obtained for winter months associated with soil temperatures less than 10°C. Populations increased with warmer temperatures for spring and summer, but declined during dry periods in late summer or early autumn when the soil water potential was lower than −9 bars, although at that period soil temperatures were favourable. High populations were recorded in autumn, then declined with decrease in soil temperatures during winter. Correlation coefficients indicated a highly significant relationship between pathogen populations and soil temperatures from autumn to early summer, and between soil moisture and pathogen population for summer and autumn, in the Brisbane Ranges independently of site. The same pattern was evident in wetter forests at Narbethong and savannah woodlands at Wilson's Promontory, although results were not significant.

Disease was evident wherever the pathogen occurred among susceptible hosts. The savannah woodland, the dry shrubby sclerophyll forest and the wetter sclerophyll forest all contained susceptible dominants; consequently disease was associated with changes in the forest community such as early death of the understorey, later die-back and death of the trees, and an increase in sedges and in bare ground. Symptoms and deaths increased with time from invasion. The severity of disease and its rate of extension, apart from spread by free water, were associated with environmental factors such as shallow soil, poor drainage and low soil water-holding capacity. These were characteristic of the Brisbane Ranges, where destruction of the forest community was severe and the rate of disease extension rapid. In the deep krasnozem at Narbethong and the deep sands of Wilson's Promontory, destruction was confined to the most susceptible hosts, disease extension was continuous but slow, and deaths occurred in a mosaic throughout the infected zone.

Introduction

Populations of soil-borne pathogens have been studied in relation to root disease in soils carrying agricultural crops (Cook 1973). There is, however, very little recorded information concerning the relation between pathogen populations, environmental factors and root disease in the ecology of native plant communities. Marks *et al.* (1973) reported a highly variable population density of *Phytophthora cinnamomi* Rands in east Gippsland soils which were fired, cleared, cultivated, fertilized and planted with forest trees. Preliminary investigations into populations of *P. cinnamomi* in uncultivated forest soils (Weste and Ruppin 1975) demonstrated considerable seasonal variation. Numbers varied from zero in midwinter to the maximum on the scale (256) in spring and summer. Population densities varied with temperature and, within certain temperature limits, with soil moisture on sites in the Brisbane Ranges.

While a preliminary survey demonstrated the seasonal pattern, possible correlations between pathogen populations and factors such as soil temperature and soil moisture, independent of site and in other forest communities, required a more detailed survey. This paper records a study of *P. cinnamomi* populations within three different forest ecosystems.

Experimental

Ecosystems Selected

Savannah woodland at Wilson's Promontory National Park (Fig. 1). This consists of a two-layered woodland on deep sands on the northern slopes of the Vereker Spur. The tree stratum is dominated by *Banksia serrata* L. with scattered trees of *Eucalyptus obliqua* L'Herit. The understorey dominant is *Xanthorrhoea australis* R.Br., which is associated with woody shrubs such as *Leucopogon virgatus* Labill., *Leptospermum myrsinoides* Schlecht. and *Pultenaea hibbertioides* Hook. Mean annual rainfall is 1100 mm.

Fig. 1. Savannah woodland, Vereker Spur, Wilson's Promontory National Park. The dominant tree is *Banksia serrata* and the dominant shrub is *Xanthorrhoea australis*.

Dry sclerophyll shrubby forest in the Brisbane Ranges (Fig. 2). This is a two-layered forest on shallow soils derived from tertiary sandstone. Soils are duplex, sandy and solodic. The dominant trees are the stringybark eucalypts such as *E. baxteri* (Benth.) Maiden & Blakely, *E. macrorhyncha* F. Muell., and *E. obliqua*. The understorey dominant is *X. australis*, and other woody shrubs are *Isopogon ceratophyllus* R.Br., *Pultenaea pedunculata* HK., *Hibbertia stricta* R.Br., *Epacris impressa* Labill. and *Leucopogon virgatus* Labill. Mean annual rainfall is 635 mm.

A much wetter sclerophyll forest on deep krasnozem loam in the mountains at Narbethong (Fig. 3). This is not entirely a wet sclerophyll forest, being intermediate

Fig. 2. Dry shrubby sclerophyll forest, Brisbane Ranges. The dominant trees are stringybark eucalypts, *E. baxteria* and *E. macrorhyncha*. *X. australis* dominates the understorey, and its symptoms mark the disease front.

Fig. 3. Forest site at Narbethong. Die-back symptoms in *E. obliqua* and *E. radiata* 80 m high were host reaction to *P. cinnamomi*.

on the hillsides and grading into wet sclerophyll forest in the valleys. Mean annual rainfall is 1524 mm. The dominant trees are *E. obliqua* and *E. radiata* Sieber ex DC., with a lower tree stratum of *Acacia dealbata* Link and *A. melanoxylon* R.Br. Dominant shrubs are *L. myrsinoides*, *Platylobium obtusangulum* Hook. and *Tetratheca ciliata* Lindl.

Eucalyptus obliqua and some of the shrubs such as *Epacris impressa*, *T. ciliata*, *L. myrsinoides* and *P. obtusangulum* are common to all areas. Each of the three localities carries native vegetation, the growth of natural regeneration following bushfires. The soils have never been cultivated and the forests have not been burnt for at least 30 years.

Aetiology of Disease

Die-back disease occurs in discrete areas in each locality. Symptoms are obvious among species of all strata. Three sampling sites were selected for both Brisbane Ranges and Narbethong and one for Wilson's Promontory and these were used for all subsequent measurements. *P. cinnamomi* was the only pathogen isolated from plating roots of diseased plants surface-sterilized with 70% alcohol on various agar media, or from baiting soil and root samples with lupin roots. Unaffected areas at each locality were tested and found free from the pathogen. When small plots of unaffected vegetation (1 m^2) were inoculated with washed mycelium of *P. cinnamomi*, or soil from die-back areas, a typical die-back disease developed in the understorey and later in the overstorey, and the pathogen was re-isolated; thus a causal relationship was established between this pathogen and disease for each locality (Koch's postulates). Subsequent tests monitored the rate at which (*a*) the pathogen and (*b*) symptoms spread from this inoculum (Weste 1974).

Selection of Sampling Sites

Sampling sites were necessarily confined to the discrete areas of diseased vegetation within each forest community and were each 21 by 30 m. The three sampling sites each for the Brisbane Ranges and Narbethong were not intended as replicates; they were selected as differing in various characteristics such as period of disease and drainage. For example, site 1 in the Brisbane Ranges was on the margin of disease, had an active disease front with an active and advancing pathogen population, which had only been present 6 months. Sites 2 and 3 had been diseased for 3 years, but while drainage was poor in site 2, it was adequate in site 3. These sites were separated by 20 m, had the same type and depth of soil and carried the same type of vegetation.

Site 1 at Narbethong carried undisturbed natural forest; site 2 was planted with *E. obliqua*, and site 3 was planted with *Pinus radiata* D. Don. Site 3 was on a rise facing north, and was warmer and drier than the other sites. All were in the same valley with the same type and depth of soil. Sites were separated by *c.* 500 m.

The Wilson's Promontory site was uniform in soil, aspect and vegetation, so only one site was selected. The low hillside was covered with a mosaic of diseased and unaffected plants.

Each month, samples of soil and roots were collected from four widely scattered diseased plants within each discrete diseased sampling site. These subsamples were bulked together, thoroughly mixed and subsampled (Leeper 1967).

Statistical assistance was obtained both for the planning of this project and for the analysis of results. Sampling on a grid system was not considered practicable

because of the fact that the pathogen, like other obligate root parasites, was concentrated on the roots of susceptible hosts and not uniformly distributed through the soil (Marks *et al.* 1973).

Pathogen Populations

The smallest quantity (air-dry weight) of soil and root sample from which *P. cinnamomi* could be isolated was used as an index of the population density of the pathogen, based on the method developed by Tsao (1960). This method is based on the assumption that the population density index of the pathogen determined by baiting is related to that of the soil from which the samples were collected. Greenhalgh (Plant Research Institute, Burnley, unpubl. data) has demonstrated a direct relationship between the number of chlamydospores of *P. cinnamomi* added to soil and the frequency of detection by lupin baiting. Samples were collected at monthly intervals from each of three sites in the Brisbane Ranges and three at Narbethong, and from the Wilson's Promontory site during a period of 1–3 years. From each site, four subsamples, each 0·5 kg of soil and roots from beneath four scattered diseased hosts, were collected and thoroughly mixed. Three 50 g replicates of each sample were baited each with three germinated seeds of *Lupinus angustifolius* L. (Chee and Newhook 1965). After 48 hours' incubation at 25°C in darkness, surface-sterilized lupin radicles were plated onto antibiotic agar (Eckert and Tsao 1962), from which the characteristic growth of *P. cinnamomi* was easily identified. While the basic samples tested were each 50 g, fractions of these from 1/2 to 1/256 were tested in triplicate from each sample of soil and roots and the reciprocal of the smallest fraction from which the pathogen was isolated was used as the population density index (PDI). Eighty-one baiting tests, each with three possible isolates (from three lupin roots), were processed at a time for each site.

Environmental Factors

These were measured concurrently with population densities. Soil temperatures were measured by three thermistor probes buried permanently at 5–10 cm at each site. At each sampling, the probes were connected to a Simpson thermometer and soil temperatures recorded. A recording thermograph (O.T.A. earth thermograph 3) with three leads buried in the soil was installed centrally to the plots in each forest community. This recorded maximum and minimum soil temperatures. The accuracy of the instruments was tested regularly by cross-checking. Rainfall was measured on a continuously recording gauge set up at each locality. Soil water potential curves were prepared for each sampling site, by using the psychrometer chamber (Wescor) and the filter paper method (Fawcett and Collis-George 1967). The percentage moisture content of the soil was measured at each sampling date and the water potential determined from the curves. A mechanical analysis was made, and the organic carbon content (Walkley and Black 1934), total nitrogen (Kjeldahl microtechnique, Jackson 1962), exchangeable cations of calcium, magnesium, potassium and sodium (Jackson 1962), total phosphorus (Baagsgard and Sandell 1954), and available phosphorus (Olsen *et al.* 1954) were determined for each soil. Site characteristics such as depth of soil, slope and drainage were recorded for each sampling site. These measurements were taken in the search for predisposing or suppressive factors for die-back disease.

Results

Ecological factors

Some salient differences between the three ecosystems are summarized in Table 1.

At Wilson's Promontory, one large area, *c.* 5000 m^2, of savannah woodland was selected covering a hillside on which die-back disease was moderately uniform and included death of all *Xanthorrhoea australis* and *Isopogon ceratophyllus* and scattered deaths of mature trees of *Banksia serrata* and *Eucalyptus obliqua*. There was a three-fold increase in populations of sedges such as *Lepidosperma concavum* R.Br. and *Hypolaena fastigiata* R.Br. (an increase from 5% to 30% in percentage cover) and some increase in bare ground measured on plots 21 by 30 m. There was no obvious disease front.

Table 1. Comparison of forest communities

Site	Wilson's Promontory	Brisbane Ranges	Narbethong
Altitude (m)	50	400	600
Vegetation	Savannah woodland	Dry sclerophyll shrubby forest	Wetter sclerophyll forest
Dominant (overstorey)	*Banksia serrata*, *E. obliqua*	*Eucalyptus baxteri–E. obliqua*	*E. obliqua*, *E. radiata*
Dominant (understorey)	*Xanthorrhoea australis*	*X. australis*	*Leptospermum myrsinoides*
Soil type	Siliceous sands	Duplex sandy solodic soil	Krasnozem
Soil depth (cm)	130	24	>135

The three sites selected in the Brisbane Ranges were those reported earlier (Weste and Ruppin 1975). One contained an active disease front; the other two had been diseased for 3 years and one was poorly drained. The latter two sites contained 70 and 90% bare ground respectively as a result of die-back disease. The only unaffected understorey plants were the invading sedges *Lepidosperma semiteres* F. Muell. ex Boeck. and *L. concavum*. *Leptospermum myrsinoides* Schlecht. was the only other living understorey plant and it showed severe die-back symptoms. The three sites had clay pans at depths varying from 38 to 61 cm, which were impermeable to water, pathogen and host roots.

The Narbethong soils contained a much greater clay fraction and higher total phosphorus, nitrogen and organic contents than the other soils (Table 2). The Narbethong community was associated with a forest nursery and plantation. The nursery seedlings, then the plantation seedlings and finally the whole plantation and its associated natural regeneration became infected, which resulted in a random distribution of the pathogen. The principal sampling site was outside the plantation in a mature stand, *c.* 80 m high, of *E. obliqua* and *E. radiata*. Thirty-eight or 25% of these died during the investigation, while others exhibited die-back symptoms. The second site was in a provenance trial of *E. obliqua*, five individuals of which died during the study. 92% of the *Tetratheca ciliata* and 50% of the *Platylobium obtusangulum* died in the same period. The third was in a plantation of *Pinus radiata*

D. Don; both of these plantations were 5 years old. The plantation areas had been cleared originally, but the planted trees were overtopped by dense natural regeneration. The pines showed no symptoms, but 12 of the 107 *P. radiata* trees died during the study period, compared with 25% of the large population of *T. ciliata*.

Table 2. Relevant soil data

Each result is the mean of three determinations

	Brisbane Ranges	Wilson's Promontory	Narbethong	Botany Garden	Dandenongs
Organic matter (%)	1·2	2·5	5·5	7·2	12·2
Nitrogen (ppm)	431·4	753·5	1705·6		1340·3
Phosphorus (total) (ppm)	149·0	55·0	325·3		
Soil pH	5·5	5·0	5·8	5·7	
Clay (%) (<0·04 mm diam.)	2·9	0·6	57·7		

Population measurements

These were low during winter, from the end of May to the beginning of August, when zero population was usually recorded. For example, in the Brisbane Ranges all readings were zero and at Narbethong seven out of 12 were zero. However, the population density index increased rapidly to a maximum in spring and summer

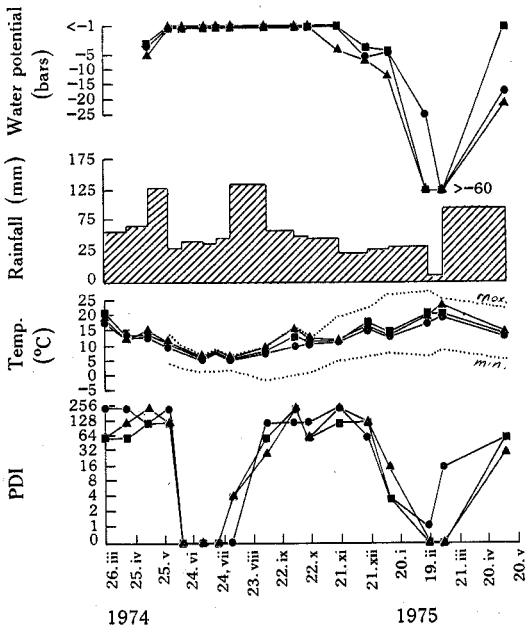

Fig. 4. Pathogen population density, and environmental data for three sites in the Brisbane Ranges. ■ Site 1. ▲ Site 2. ● Site 3.

(September to January). During late summer or early autumn there was a sudden brief decline from 256 to zero. High populations were otherwise recorded during autumn, from March to May. The late summer–early autumn decline was not recorded for Wilson's Promontory (Figs. 4–6).

Environmental factors

Soil temperatures are recorded in Figs. 4–6. During winter, forest soil temperatures remained below 10–12°C for a period of about 3 months. However, winter soil temperatures at Wilson's Promontory were milder and seldom less than 10°C, probably owing to its maritime position.

Soil water potential rather than percentage moisture content was recorded because the former takes cognisance of the varying water availability characteristic of the different soils. The graphs relating soil moisture content to soil water potential for the three forest communities are presented in Fig. 7. The Brisbane Ranges soil was characterized by a poor water-holding capacity; an increase of only 2·5% in moisture content changed the soil from a water potential near the wilting point of most plants to one of saturation. In addition, the soil was shallow with an impermeable clay pan. Several periods of both water saturation and of extreme dryness occurred during most years but from June to November 1974 the soil water potential was

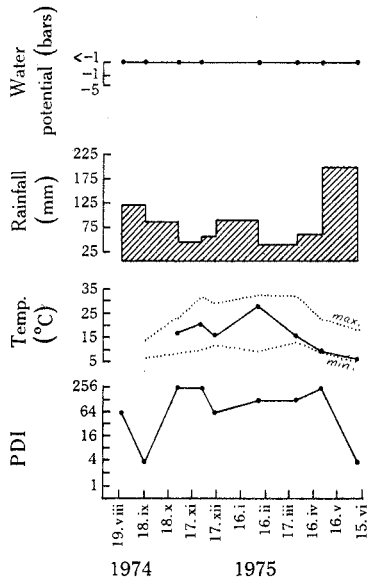

Fig. 5. Pathogen population density and environmental data for one site at Wilson's Promontory.

greater than −1 bar. Wilson's Promontory soil was a nutritionally poor sand, but the soil was deep, drainage excellent and the soil water potential remarkably uniform (Fig. 5) because the locality received frequent coastal rain. The Narbethong soil was a deep friable krasnozem with greater water-holding capacity. Nevertheless, dry periods occurred during late summer or autumn at both the Brisbane Ranges and Narbethong.

Correlation between pathogen population density and environmental factors

The correlation coefficients for the relationships between soil temperature and pathogen population, and between soil moisture and pathogen population, were determined from the records from each site in each forest community and for the total of all sites for each ecosystem. Correlation coefficients for population density and temperature were highly significant ($P < 0{\cdot}001$) for the three sites individually and for the whole area in the Brisbane Ranges from 26 March to 18 December 1974.

During this period, soil moisture remained relatively high and relatively constant. From 18 December 1974 to 7 May 1975, there was a highly significant correlation ($P < 0{\cdot}001$) between soil moisture and pathogen population density for site 2 and a significant correlation ($P < 0{\cdot}05$) for site 1 and no correlation with temperature. Results for Wilson's Promontory and Narbethong were analysed in the same way, but were not significant, partly because there were fewer results to analyse, measurements being recorded over a shorter period of time. The trends in the relationship between the various parameters can be observed from Figs. 4–6.

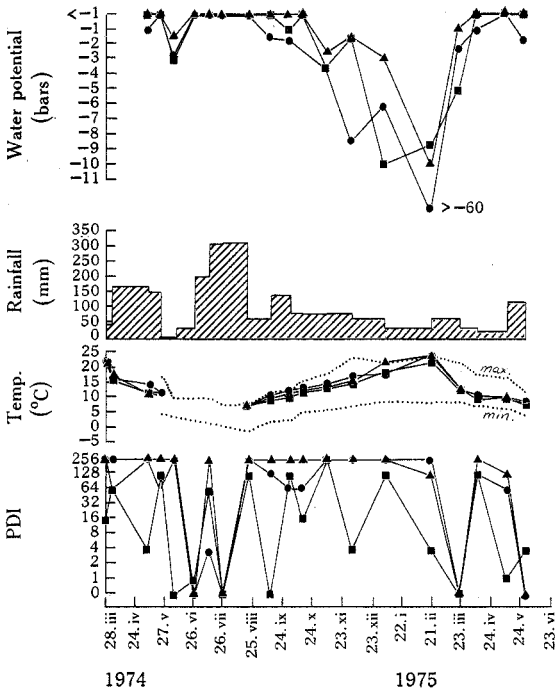

Fig. 6. Pathogen population density and environmental data for three sites at Narbethong. ■ Site 1. ▲ Site 2. ● Site 3.

For all sites, pathogen populations were small in winter (PDI 0–4) as long as the maximum soil temperatures remained less than 10°C. When they rose above 10°, for example briefly in July at Narbethong, pathogen populations increased rapidly. There was no reduction in pathogen populations when high soil temperatures were recorded provided the soil water potential was higher than −9 bars. A soil temperature maximum of 33° at Wilson's Promontory was associated with a PDI of 128 at a time when the soil water potential was greater than −1 bar.

Pathogen populations declined in late summer to early autumn on sites in the Brisbane Ranges and Narbethong when the soil water potential declined to values lower than −9 bars, although temperatures remained optimal. Immediately soil water potentials increased, pathogen populations also increased.

The results from each forest community are briefly reported.

Wilson's Promontory

The climate is maritime and the soil water potential remained higher than −1 bar throughout the year owing to frequent coastal rain. The pathogen population density

varied with soil temperature but not significantly. The coastal winter is relatively mild, and soil temperatures less than 10°C were only recorded twice in June and September. These were the only two occasions on which small populations (PDI 4) were recorded (Fig. 5).

Brisbane Ranges

Small populations (PDI 0–4) were recorded during winter (from the end of May to mid August), when soil temperatures were less than 10°C, although soil water potentials were higher than −1 bar. Large pathogen population densities (PDI 64–256) were recorded for spring and summer (late August to late January), when soil temperatures varied from 12 to 25° and soil water potentials were higher than −10 bars. Pathogen populations declined to zero in late summer and early autumn for February and early March, although soil temperatures were optimal for the pathogen (20–25°). However, at this period the soil water potential was lower than −60 bars. Rain at the end of March was followed by increased pathogen populations until winter, when the June reduction in population was associated with low soil temperatures. Site 3, which was poorly drained, showed a slower decline in both soil water potential and population densities during autumn (Fig. 4).

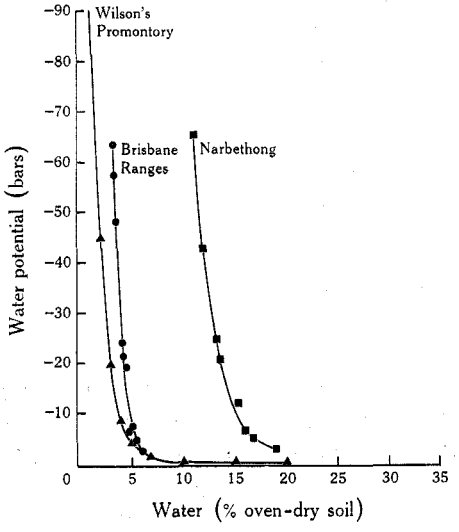

Fig. 7. Curves relating soil moisture percentage to soil water potential for soils from experimental sites.

Narbethong

Zero populations were recorded for the winter months (May to August), as long as soil temperatures remained less than 10°C. During July a brief rise in soil temperature was associated with a brief increase in pathogen population. Population density was maximum (PDI 256) during spring and summer (from late August to late February), when soil temperatures varied from 12 to 25° and the soil water potential was higher than −9 bars. In autumn, at the end of March 1975, the pathogen population fell to zero although soil temperatures remained favourable. At this time the soil water potential was lower than −9 bars for all sites and lower than −60 bars for the dry site (Fig. 6).

Comparative effect of disease on the ecosystems

As a result of root disease due to *P. cinnamomi*, changes occurred in the ecosystems. The first and most obvious sign at each of the three localities was the destruction of the understorey. Both total numbers of plants and numbers of species were reduced and the percentage of bare ground increased. About 12 months later, trees began to show die-back symptoms and either died or persisted with a reduced canopy. Detailed results have been reported for the Brisbane Ranges (Weste and Ruppin 1975); those for Narbethong and Wilson's Promontory will be published in a later paper. With the reduction in root absorption, vegetative cover and hence transpiration, sites which have been severely diseased for more than 3 years appeared considerably wetter, light increased at ground level and there occurred a gradual invasion by sedges such as *Gahnia radula, Lepidosperma* spp. and *Hypolaena fastigiata.* Some tolerant species such as *L. myrsinoides* and *P. obtusangulum* persisted although exhibiting symptoms of chlorosis and die-back. *P. cinnamomi* was readily isolated from the roots of both remaining shrubs and surviving ecualypts but not from sedges or grasses. Soil analyses at diseased sites showed a decreased organic content and decreased total nitrogen, both probably the result of the reduction in plant cover, and this was associated with a reduction in soil microflora. An assessment of an area diseased for 12 months or more revealed a damp site suitable for sporangial formation and zoospore release by *P. cinnamomi* and poor in microflora and therefore competitive soil colonization. This means that the diseased site was degraded. With the elimination of the shrubby understorey and the reduction in tree numbers, the shrubby sclerophyll forest became a woodland, either a sedge or a grassy woodland depending on factors such as soil moisture and fertility (Weste *et al.* 1973).

Discussion

These investigations have supported the previous conclusion (Weste and Ruppin 1975) that in plant communities invaded by *P. cinnamomi* the most important factor in disease development was the association between pathogen and susceptible host. Structural dominants of Victorian plant communities studied were highly susceptible, so that the presence of the pathogen was associated with severe disease. Since a structural dominant (by definition) influences the type of plant community, the disease and death of such dominants has resulted in the destruction first of the plant community (Fig. 2) and later of the entire ecosystem. Examples of Victorian plant communities affected in this way (Weste and Marks 1974) include the stringybark forests dominated by *E. baxteri* and *E. macrorhyncha*, the messmate–peppermint forests dominated by *E. obliqua* and *E. radiata*, the ash forests dominated by *E. regnans*, the *Banksia* woodlands dominated by *B. serrata*, and the east Gippsland coastal forests dominated by *E. sieberi.* Similarly, heathlands and swamps develop severe disease when the dominant heath species is *Epacris impressa* or the swamp heath, *Sprengelia incarnata* Sm., which are both highly susceptible.

Within the limits of the ecosystems studied, these investigations have demonstrated that environmental factors do not determine whether or not disease develops. Disease development depends on the susceptibility of the structural dominants. However, these experiments have demonstrated that environmental factors are most important in determining pathogen population densities and hence potentially the severity of disease and its rate of spread.

Associations between population density, soil temperature and soil moisture were demonstrated for 1973–1974 in the Brisbane Ranges (Weste and Ruppin 1975). The results presented in this paper demonstrate the association for 1974 and 1975 in the Brisbane Ranges, Narbethong and Wilson's Promontory (Figs. 4–6). The correlation coefficient demonstrates a highly significant relationship ($P < 0{\cdot}01$) between population density and soil temperature from autumn to late spring in the Brisbane Ranges. During this period, soil water potential was uniform and greater than -1 bar, and the pathogen population was primarily temperature-controlled. Results were highly significant both for individual plots and for the total area, therefore independent of the differences between the plots. Low winter temperatures were correlated with a significant reduction in the soil population of *P. cinnamomi*. Others (Zentmyer and Marshall 1959; Kuhlman 1964; Podger 1968; Shea 1975) have demonstrated that temperatures above 12°C are required for sporangial production and infection of roots by *P. cinnamomi*. In the Brisbane Ranges and at Narbethong, population densities were zero when the mean soil temperature was less than 10°. However, minimum temperatures did not fall below zero on any site and evidently the pathogen was not destroyed by low temperature because populations rapidly increased with the return of warm temperatures. Maximum soil temperatures were not recorded above those satisfactory for growth *in vitro* of *P. cinnamomi* (Shepherd and Pratt 1974).

The effect of soil temperature on population numbers was often dramatic. When the temperature fell below 10°C, the PDI declined from 128 to zero in 13 days, for example between 30 May and 12 June in the Brisbane Ranges and between 23 May and 6 June at Narbethong. During favourable conditions in pot tests (Weste, unpubl. data) the pathogen grew on roots as mycelium producing sporangia, zoospores and occasional resistant chlamydospores. With adverse conditions, the pathogen survived only as resistant spores or inside host roots. With higher soil temperatures, the population increase was more gradual over 54 days. Within a favourable temperature range, the population density of *P. cinnamomi* was correlated with soil moisture in the Brisbane Ranges, the correlation being highly significant in summer for site 2 and significant in winter for site 3. Cook (1973) has reported optimal growth for *P. cinnamomi* at water potentials of -5 bars. Adebayo and Harris (1971) demonstrated that the effect of soil water potential on the growth of *P. cinnamomi* varied with the soil texture; for example, growth retardation occurred at -5 bars in sandy loam, at -10 bars in silt loam and at an intermediate value for sand. During most of 1973–1974 soils were sufficiently moist for growth of the fungus. However, soils with water potentials lower than -9 bars limited populations at times between December 1973 and May 1974 (Figs. 4, 6), as for example in the Brisbane Ranges (on 19 February and 4 March soil water potentials were lower than -60 bars) and at Narbethong (on 2 March soil water potentials were -60 bars). Zero PDI values were recorded on all sites except site 3 in the Brisbane Ranges, which was a water-gaining site. These results were highly significant for two sites in the Brisbane Ranges. Two days after rain the PDI had increased from 0 to 128 on site 1 in the Brisbane Ranges (Weste and Ruppin 1975). The rapid increase in the pathogen population was probably due to germination of the chlamydospores, and the production of sporangia and zoospores in free water. A population decline with low soil water potential and its recovery after rain is shown for the Brisbane Ranges in

February 1975 (Fig. 4) and for Narbethong in March 1974 and 1975 (Fig. 6). The deeper Narbethong soils with higher clay and organic components appear to have a buffering effect compared with the Brisbane Ranges.

The methods used to measure pathogen population density were not entirely satisfactory. For example, a zero population was recorded for winter, yet the pathogen population increased immediately with the return of favourable conditions such as soil temperatures greater than 10°C and soil water potentials greater than −9 bars, hence the fungus must be capable of rapid reproduction and dispersal as soon as the environment becomes suitable. *P. cinnamomi* may survive unfavourable conditions as oospores or chlamydospores. Only the A_2 mating type has been isolated from these soils and oospores have never been observed in our studies, but chlamydospores have been frequently observed to form inside root tissues and to germinate quite readily. They may produce sporangia and zoospores, thus rapidly increasing population densities. Duncan (1976) used the maximum likelihood or most probable number method of population analysis, which assumes random distribution of inoculum in the soil. *P. cinnamomi* is not either evenly or randomly distributed in the soil of uncultivated forests, but is concentrated along the fine roots of hosts (Marks *et al.* 1973), as would be expected for obligate root parasites. Pathogen populations increased with time from invasion for a while then declined with the decline in population of susceptible hosts. Maximum populations were obtained on the margin of a diseased area (Fig. 4) where there was a dense supply of susceptible fine roots (Weste *et al.* 1973). Experimental evidence has demonstrated that pathogen populations fluctuated with seasonal variations of soil temperature and soil moisture (Figs. 4–6). Otrosina and Marx (1975) recorded wide variations in soil populations of *P. cinnamomi*, the numbers being greater beneath the more susceptible *Pinus echinata* Mill. than beneath *P. taeda* L. They were unable to relate population variability to seasonal conditions, but recorded an unusually mild winter and cool summer during the sampling year. Marks *et al.* (1973) reported a variable population density in cultivated east Gippsland soils with a decline during winter associated with lower soil temperatures. East Gippsland receives regular summer rains.

The Victorian environment, apart from mountains or deserts, appears peculiarly suited to the requirements of *P. cinnamomi*, having frequent warm wet periods favouring sporangial production, zoospore release and dispersal, and root infection. The Western Australian jarrah forests have only brief periods when soil temperatures and moisture are suitable for growth and infection by *P. cinnamomi* (Shea 1975). In addition, many Victorian forest communities experience dry periods with severe water stress. Under these conditions, decay and death of the fine roots is probably fatal to the infected forest flora. Victorian forests may present a special hazard, for three reasons: the recent introduction of the pathogen (Weste 1974*a*); the susceptibility of many structural dominants such as the renantherous eucalypts (Marks *et al.* 1973); and the exigencies of the climate, the relevance of which is demonstrated by the results presented in this paper and by Weste and Ruppin (1975). A suppressive soil would therefore be of great value in Victorian natural communities. So far, no uncultivated Victorian soil has been found suppressive *sensu* Broadbent and Baker (1974), but there is a range of disease liability from dry sclerophyll plant communities where the disease hazard is maximal to wet sclerophyll forest on the richer krasnozem loams where disease hazard remains lower. A range of disease liability has been demonstrated for the three forest communities described in this paper.

The other environmental characters measured, such as drainage, slope, depth of soil, soil–water relations, organic and nutrient content and microbial populations, are all factors important in determining the severity of the disease and its rate of spread (Weste *et al.* 1975). Disease extension has been measured at 400 m per year with water run-off both at Wilson's Promontory and in the Brisbane Ranges. But disease extension through soil on more or less level ground varies from 18 m per year in deep sands at Wilson's Promontory to 171 m per year in shallow soils of the Brisbane Ranges (Weste and Law 1973).

The shallow soil of the Brisbane Ranges has important consequences in that both fungus and host roots occupy a relatively small volume of soil, thus presenting the pathogen with a high concentration of substrate. This relatively small volume of soil is easily saturated with rain water and may become anaerobic, hence damaging for host roots, while free water favours fungal zoospore formation, dispersal and infection. In comparison, the pathogen while concentrated round host roots is also dispersed throughout the 80 cm of the deep soils characteristic of Narbethong and Wilson's Promontory (Weste and Law 1973). Soils from the Brisbane Ranges are characterized by a particularly steep water potential curve (Fig. 7), which means that a difference of only $2\frac{1}{2}\%$ in moisture content represents a change from water saturation, promoting zoospore formation, release and dispersal, to a water stress which endangers plant survival. These soils were also characterized by a low microbial population and so are not likely to be suppressive (Weste and Vithanage 1977).

A rating system may be devised involving both susceptibility of dominants and environmental factors, to estimate the potential disease hazard for Victorian dry sclerophyll forests as in the Brisbane Ranges, to minimum hazard in wet sclerophyll forests such as Sherbrooke forest (Weste and Marks 1974). If disease due to *P. cinnamomi* were controlled entirely by environment, it would be difficult to understand why any disease was found in Sherbrooke forest, and certainly why it was severe at Narbethong since both areas have well-drained friable krasnozem loam and high reliable rainfall. As with other pathogens, disease is the process of a change in relationship between host, pathogen and environment, all three of which interact in disease induction.

Conclusions

The results of this investigation have demonstrated that disease due to *P. cinnamomi* depends on the introduction of the pathogen and the susceptibility of the structural dominants in the forest community; and that the severity of the disease and its rate of extension depend on the population density of the pathogen and certain environmental factors such as soil temperature and soil water potential. Pathogen populations in three diseased forest communities were correlated first with temperature, and within a suitable temperature range with soil water potential, independent of site. This study has exposed the lack of information concerning the survival of the pathogen during adverse conditions and the mechanism by which population densities increase with the return of suitable conditions.

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