Root Temperature Effects on the Growth of *Phytophthora cinnamomi* in the Roots of *Eucalyptus marginata* and *E. calophylla*

B. R. Grant and Pauline N. Byrt

Senior lecturer and postdoctoral research associate, respectively, The Russell Grimwade School of Biochemistry, University of Melbourne, Parkville, Victoria 3052, Australia.

This study was made possible by generous support from the Utah Foundation, the Dieback Research Foundation, and the Buckland Foundation.

Accepted for publication 9 August 1983.

ABSTRACT

Grant, B. R., and Byrt, P. N. 1984. Root temperature effects on the growth of *Phytophthora cinnamomi* in the roots of *Eucalyptus marginata* and *E. calophylla*. Phytopathology 74:179-184.

Colonization of the roots of seedling jarrah (*Eucalyptus marginata*) and marri (*E. calophylla*) by the cinnamon fungus (*Phytophthora cinnamomi*) was studied under controlled temperatures and light intensity. Clear differences in pathogen development were shown in the two species. In marri (field-resistant), invasion was restricted after 20–120 mm of the root tissue was colonized. The extent of the invasion within a temperature range of 14–28 C varied, but the growth of the pathogen was always limited in this species. *P. cinnamomi* could be recovered from the zone of healthy tissue adjacent to the lesion for up to 30 days after hyphal growth was contained, but there was no infection of roots not in direct contact with the diseased tissue. In jarrah, a field-susceptible species, a wider variation in the degree of root colonization was observed, although the extension of the pathogen was still dependent upon root temperature. At 14 C, the invasion was restricted to between 30 and 50 mm of the root. At 28 C the fungus spread through the root system, although the total extension varied between seedlings. At intermediate temperatures, the reaction varied from containment of hyphae to systemic invasion of the root system. These differences in the capacity of seedling roots of laboratory grown plants to restrict the invasion by *P. cinnamomi* parallel differences in the sensitivity to the disease shown by mature trees of the same species in the field. They suggest that even in a susceptible species such as jarrah, the degree to which root tissue is colonized is temperature dependent.

Jarrah (*Eucalyptus marginata* Donn ex Sm.) and marri (*E. calophylla* R. Br.) grow in association on a variety of soil types in the southwestern part of Western Australia (W.A.) (11). Jarrah, an important commercial timber species, is susceptible to the disease of jarrah or eucalypt dieback, associated with the invasion of the root system by *Phytophthora cinnamomi* Rands (21). *E. marginata* and *E. calophylla* are members of the subgenera *Monocalyptus* and *Corymbia*, respectively (23). Members of the subgenus *Monocalyptus* are particularly susceptible to attack by *P. cinnamomi*, both in field and in pot trials (17,22,34).

Although *Phytophthora cinnamomi* invades the roots of a wide variety of *Eucalyptus* species at the seedling stage when these are inoculated with zoospores, few differences in the host reaction to the parasite have been detected in the first few hours of invasion (17,28,29). However, it has been shown that roots of resistant species of *Eucalyptus* showed greater increases in the rate of ion leakage (33) and callose deposition (6) than those of susceptible species. In addition, differences in the rates of hyphal growth in seedling roots of tolerant and susceptible eucalypts were found by Byrt and Holland (5) and by Batani (3). In the latter study, however, the differences observed in the early postinfection period had disappeared after 9 days. In studies extending over a longer term, roots of susceptible eucalypt species showed greater water loss following infection (7) and reduction in root system development (9,10) than did those of resistant species.

The purpose of this investigation was to extend the earlier observations made on very young seedlings (5) to older plants, and to determine the extent to which root temperature influenced the development of infection.

MATERIALS AND METHODS

Plant materials. Plants were grown from seeds of a known provenance, supplied by CSIRO, Division of Forest Research, Canberra. Jarrah, *E. marginata*, seed lot 12955, came from the Marriup block, W.A., and marri, *E. calophylla*, seed lot 8855, from the Perth Mundaring division. Marri seeds were surface sterilized with calcium hypochlorite (6%, 1 min) and then germinated directly in a mixture of vermiculite and acid-washed sand (2:1, v/v). Jarrah seeds were surface sterilized with silver-nitrate (0.2%, 1 min), the seed coat was removed, and the seeds were germinated under axenic conditions on moist filter paper for 5–7 days before transfer to the sand-vermiculite mixture. This was essential to obtain synchrony of germination in jarrah. Seedlings were watered with a nutrient solution (modified slightly from that of Ladiges [15]) having the following composition (all values in milligrams per liter): N as NH_4NO_3, 30; P as $NaH_2PO_4 \cdot 2H_2O$, 5; K as K_2SO_4, 10; Ca as $CaCl_2$, 50; Mg as $MgSO_4 \cdot 7H_2O$, 20; B as H_3BO_3, 0.2; Zn as $ZnSO_4 \cdot 7H_2O$, 0.1; Mn as $MnSO_4 \cdot 4H_2O$, 0.1; Mo as $(NH_4)_6Mo_6O_{24} \cdot 4H_2O$, 0.05; Cu as $CuSO_4 \cdot 5H_2O$, 0.02; Fe as FeEDTA, 2.0, pH 6.2. Solution was applied at one-quarter strength for 6 wk, followed by half strength for the next 4 wk.

After seedlings had developed to the four-leaf stage, 7–9 wk old, they were transplanted into plastic containers 40 cm in length and 6 × 15 cm in cross-section. One side of these containers was sealed with two layers of transparent plastic sheet, sealed in place with plastic insulation tape, and covered with a close fitting plastic lid. Containers were stacked with their long axes at an angle of 20 degrees to the vertical with the plastic film on the lower side and the plants were grown under the light and temperature regimes described below. Under these conditions, roots developed at the interface of the plastic sheet and the potting mixture. Extension was recorded by tracing the outline of the roots onto a clear, gridded polyacetate sheet, which was aligned relative to reference marks on the box. Lesion extension on inoculated roots was recorded in the same manner. Three to five roots were measured per box and these were measured daily during the period of root extension or lesion extension.

The aerial portions of the plants were exposed to a 16 hr light (24 C), 8 hr dark (19 C) regime in all experiments. The photosynthetically active radiation at the plant surface was 750 μEinsteins $m^{-2} \cdot sec^{-1}$. The temperature of the root systems was controlled independently by inserting the root containers in small incubators which allowed the root zone to remain within 0.5 C of a designated temperature regardless of conditions in the plant

The publication costs of this article were defrayed in part by page charge payment. This article must therefore be hereby marked "*advertisement*" in accordance with 18 U.S.C. § 1734 solely to indicate this fact.

growth chamber. This regime was imposed within 3 days after the seedlings were transplanted so that the roots under study developed at the imposed temperature.

Pathogen. *P. cinnamomi* of the A-2 compatibility type originally isolated from a Brisbane Ranges (Victoria) isolate provided by G. Weste (Commonwealth Mycological Institute [Kew, Surrey, England] accession IMI252489). The preparation of axenic zoospore suspensions has been described previously (4). Zoospore concentrations were determined by direct counting in a haemocytometer and adjusted to 10–20 cells per microliter. Roots were inoculated by placing zoospores in 1–2 μl of suspension delivered from a Gilson pipettor directly on the zone of elongation, 3–5 mm from the root tip. The inoculum was allowed to remain on the root surface in a humid atmosphere for 120 min, before the root box was resealed and the plant returned to the growth chamber. Usually only a single root on each plant was inoculated.

Mycelial inoculations were made with mycelium in 2-mm cubes of agar cut from the edge of an extending hyphal mat grown on a V-8 agar plate and placed with the mycelial surface in contact with the root. Control inoculations were carried out with both water and agar cubes.

Assessment of pathogen extension. Pathogen extension was assessed by (1) direct measurement of lesions, using gridded polyacetate sheets, and (2) by plating of roots to determine the extent of hyphal invasion. As the fungus extended into the brown zone of the root, lesions were not readily distinguished and the extension was estimated indirectly by the appearance of a lesion in lateral roots and by plating out. In plating, roots were removed, surface sterilized with 70% ethanol followed by 0.1% mercuric chloride in 0.03% Triton X-100 and three changes of sterile water. Sections 5 mm long were cut, placed on selective agar (30), and incubated in darkness at 24 C. Sections containing *P. cinnamomi* showed distinct outgrowths of coralloid hyphae after 24–48 hr, and the rapidity with which the hyphae appeared provided a semiquantitative estimate of the amount of mycelium present in each section.

Samples plated from both species at various times after lesions became visible showed a linear relation between visible lesion length, L, and hyphal extension, H, measured by plating. The equation $H = 0.9\,L + 9.9$, in which H and L (in millimeters) gave an average r^2 value of 0.87 for a total of 64 samples.

TABLE 1. Mean rate of root extension of jarrah and marri at different temperatures

Root temperature[b] (C)	Daily increment in root length (mm)[a]	
	Jarrah	Marri
14	10 ± 2	10 ± 2
16	11 ± 2	11 ± 3
24	16 ± 3	22 ± 6
28	24 ± 3	34 ± 9
31	27 ± 6	35 ± 5

[a] Means ± standard error of mean of 12 roots at each temperature, measured daily for 5 days.
[b] Temperature varied within ±0.5 C of the indicated temperature through the heating/cooling cycle in the root boxes. Stem and leaves were maintained at a 24 C day (16 hr) and 19 C night (8 hr) cycle throughout.

TABLE 2. The effect of inoculation type and site on the establishment of *Phytophthora cinnamomi* in jarrah and marri roots at 24 C

Inoculum	Inoculation site (mm from tip)	Infections/trials[a]	
		Jarrah	Marri
Zoospores	0	8/8	8/8
	80	8/8	3/8
Mycelial	0	20/20	20/20
	20	n.d.	6/6
	40	n.d.	8/8
	60	n.d.	1/6

[a] Infection was confirmed in each instance by plating of sequentially sectioned (5-mm) root segments.

RESULTS

Root growth. Both species of eucalypt produced long, unbranched roots within 1–2 wk of transplanting. Marri roots were generally more uniform in size, thicker than jarrah, and grew faster over the upper portion of the temperature range studied (Table 1). The surface of the roots darkened with age, leaving a section of white root at the tip, as previously noted by Palzer (20). Jarrah seedlings produced shorter lengths of white root, which meant that the period over which lesion formation could be observed directly was shorter than with marri. The proportion of white root was higher in the roots grown at higher temperatures in both species. Cross sections of roots grown in the special containers showed no difference in anatomy when compared with those in conventional pots. In addition to the rapidly growing roots described above, both species produced an extensive network of fine fibrous roots at the surface and extending downwards for up to 50 mm into the sand-vermiculite. This surface root system did not become infected during the period of the experiments.

Inoculum potential and inoculation site. When the inoculum potential at the zone of elongation was varied between 5 and 50 zoospores per site there was no measurable difference in the time taken for a lesion to appear in either jarrah or marri. Twelve seedlings of each species were used in these tests. Inoculation using

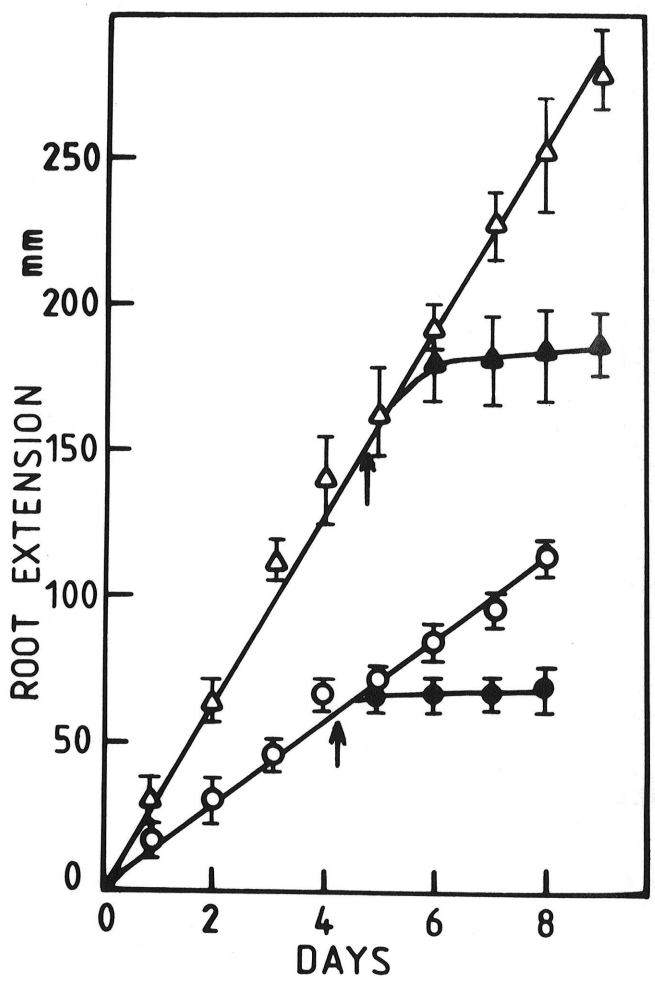

Fig. 1. The effect of inoculation with *Phytophthora cinnamomi* immediately behind the root tip, on root growth in marri (*Eucalyptus calophylla*) (triangles), and jarrah (*E. marginata*) (circles). Closed symbols represent growth after inoculation (arrows indicate time of inoculation). Roots grown at 24 C day, 19 C night. Bars represent two standard deviations, determined with data from 16 plants of each species.

mycelium at similar sites gave identical results to those obtained with zoospores. Inoculation at sites up to 80 mm from the root tip gave 100% infection in jarrah and again the same result was obtained with zoospore and mycelial inocula. In marri roots, inoculation at sites remote from the tip of the root resulted in fewer infections (Table 2), with both zoospore and mycelial inocula. Although the marri root surface becomes hydrophobic beyond 40 mm from the root tip, and thus difficult to inoculate with zoospore suspensions, mycelial inoculation also showed a decrease in the susceptibility of the older tissue to infection. This conclusion is supported by the difference in the progress of the pathogen when inoculated onto sites remote from the root tip, as will be shown later.

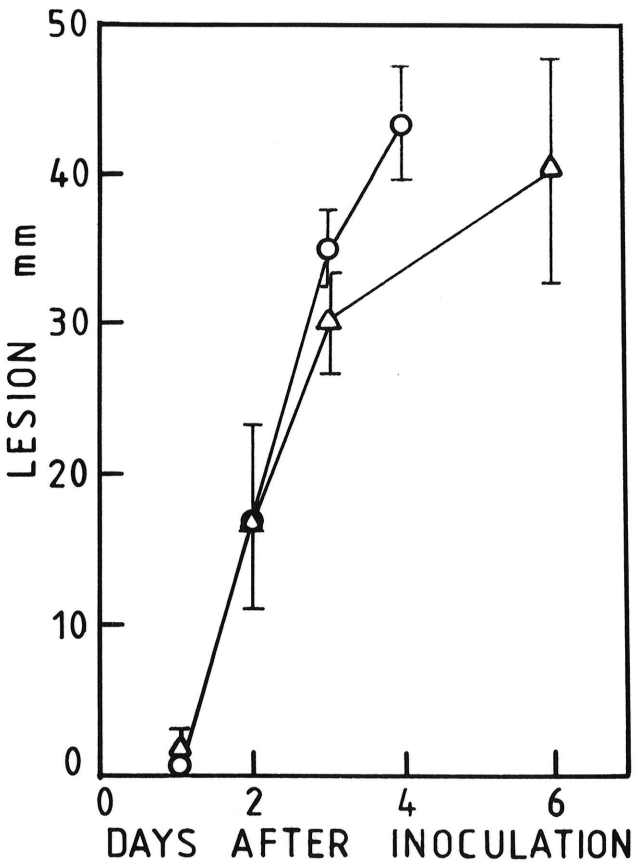

Fig. 2. Lesion extension in roots of marri (triangles) and jarrah (circles) following inoculation with *Phytophthora cinnamomi*. Bars represent two standard deviations. Other conditions were as for Fig. 1.

Postinoculation events. The pattern of extension of *P. cinnamomi* within the host root differed in the two *Eucalyptus* species, and also varied with the temperature of the root system and the site of inoculation. With inoculations immediately behind the root tip, and the root system maintained at 24 C day, 19 C night, roots ceased extension within 24 hr, while those that received water or agar block inoculations continued to grow until the root tip approached the base of the container (Fig. 1). The decrease in the rate of extension in the inoculated roots preceded the appearance of a lesion and was the first indication of infection. Between 24 and 48 hr after inoculation a lesion appeared and initially extended at comparable rates in both species (Fig. 2). Lesion extension was more rapid towards the tip; this was shown clearly when inoculations were made at some distance from the root tip (Fig. 3). By the third day after inoculation, differences in the progress of invasion of the two species became apparent.

In marri, lesion extension slowed and then ceased, leaving a well-defined necrotic zone at the tip of an otherwise healthy root. Plating showed that *P. cinnamomi* was present in the lesioned section and up to 15 mm beyond. However, there was no further extension of hyphae, even after an interval of 30 days (Fig. 4). As the root system of the plant developed and the inoculated roots matured, it became impossible to distinguish the margin of the original lesion. However, when plants were held for 4 wk and longer, the lesioned section rotted, and could then be distinguished visually from the healthy brown tissue adjacent to it.

New roots developed above the site of the initial lesion and remained free of infection unless in direct contact with lesioned sections of the inoculated root. This freedom from secondary infection was a major difference between plants grown in drained sand-vermiculite and those grown in water culture (3).

In jarrah (24 C) lesion extension slowed, but did not cease after 3 days (Fig. 5) and by this time had extended into the brown region of

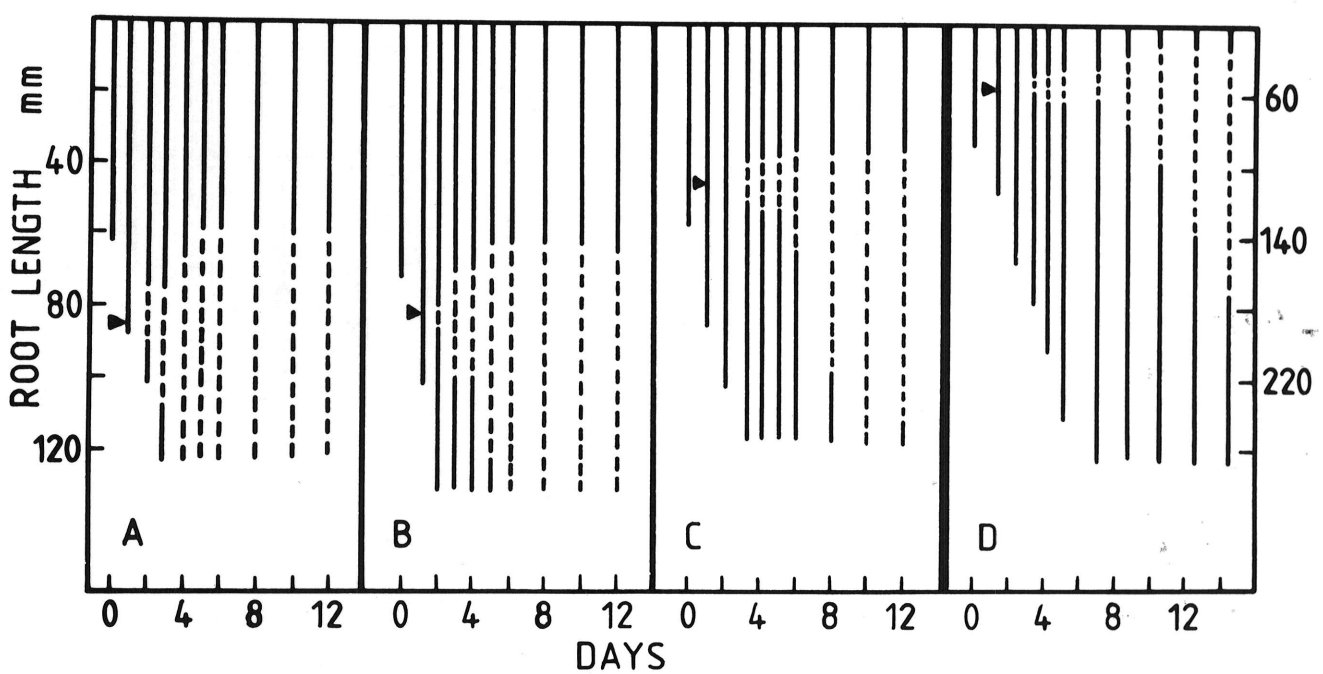

Fig. 3. Influence of the site of inoculation on the pattern of extension of lesions caused by *Phytophthora cinnamomi* in marri roots grown at 24 C. Arrow indicates site of inoculation: **A**, at the root tip; **B**, 20 mm behind the root tip; **C**, 40 mm behind the root tip; and **D**, 60 mm behind the root tip. The broken lines represent the sites of visible lesions. The greater length of root growth which took place when the inoculations were 60 mm from the tip required that frame D of the figure be drawn to a different scale.

the root, limiting further direct measurement. Plating of infected roots showed that hyphal extension varied from one seedling to another, but was generally greater than in marri (Fig. 4). The scatter of the data points for jarrah in this figure indicates the variable reaction observed in the seedling population in this species. At one extreme, the pathogen was recovered only from the inoculated root and had extended only a few centimeters at the time of sampling, 9 days postinoculation. Seedlings showing the capacity to restrict invasion to this extent at 24 C were rare and only four were found in 160 tested. At the other extreme, extension of the pathogen through the inoculated root continued, reached the hypocotyl, and entered the other long roots, infecting laterals en route. This degree of susceptibility was also rare; only 12 seedlings reacted in this way in samples tested to date. In the majority, hyphae extended through the inoculated root and infected laterals on that root. However, the rate of extension was reduced in the older tissue. As in marri, there was no evidence of secondary infection unless developing roots came into direct contact with a lesioned section.

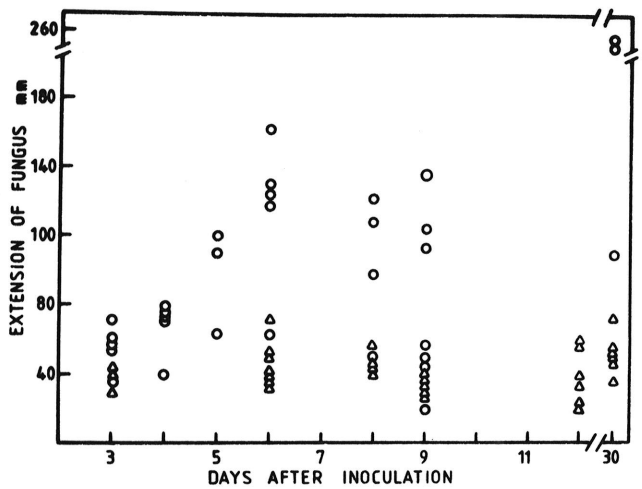

Fig. 4. Variation in the extent of hyphal extension of *Phytophthora cinnamomi* in jarrah (circles) and marri (triangles) roots as a function of time. Each point represents a single inoculated root, and the total extension was measured by plating root pieces. Conditions were as for Fig. 1.

Effect of temperature. Root temperature influenced both the rate of extension of *P. cinnamomi* and, in jarrah, the outcome of the invasion. Although marri seedlings limited the extension of *P. cinnamomi* over the full range of temperatures tested (14–31 C), the length of root infected varied from 50 ± 10 mm at 28 C to 20 ± 5 at 14 C (Fig. 6). At 14 C, 3 days elapsed before a lesion appeared, while at 28 C a lesion was visible within 16 hr of inoculation. However, once the lesion did appear its rate of extension was similar regardless of the temperature. Therefore, the difference in the length of root infected at the different temperatures reflected the variation in the lag between inoculation and lesion appearance rather than variation in hyphal extension rate. Measurements made at intermediate temperatures, 17, 19, and 26 C (*unpublished*) gave total hyphal extensions intermediate between the extremes shown in Fig. 6. At 28 C, some marri seedlings showed a slow extension of hyphae beyond the lesion, suggesting that the capacity to contain infection was beginning to break down. In a further series of experiments in which roots were grown at 31 C, no lesion formed following inoculation, and *P. cinnamomi* was not recovered when the roots were plated 7 days later. When these same plants were root pruned, and new roots developed at 28 C, a second inoculation gave 100% infection.

Hyphal extension in jarrah roots was also temperature dependent (Fig. 5) but more extensive at each temperature than in marri. At 28 C, there was no indication that infection was

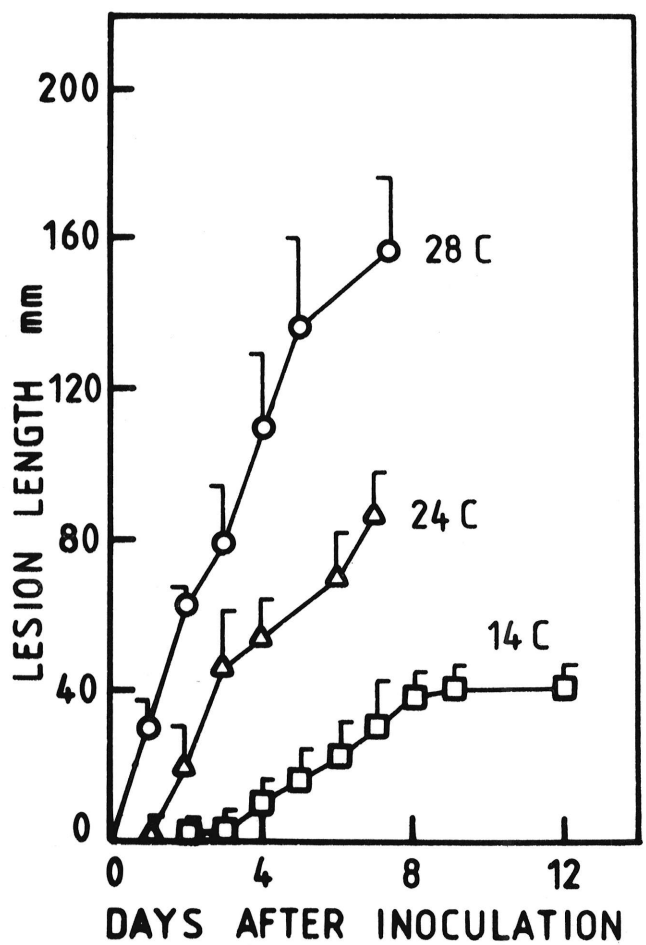

Fig. 5. The effect of temperature on the extent of lesion development in jarrah roots. Conditions were as for Fig. 6.

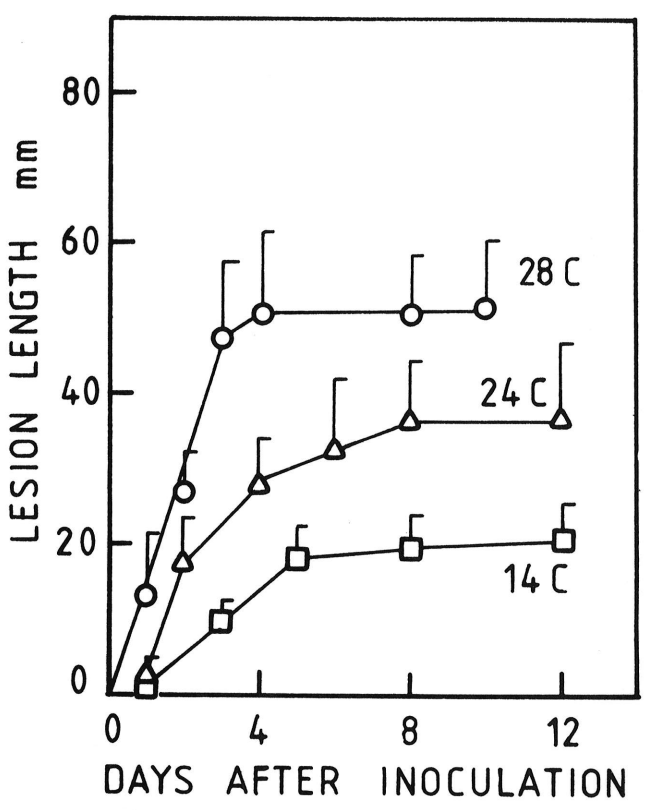

Fig. 6. The effect of temperature on the extent of lesion development in marri roots. Bars represent one standard deviation. In all cases the leaves and stems were maintained at 24 C day and 19 C night. Inoculations were made immediately behind the root tip. There were eight plants in each treatment.

contained, or restricted. In contrast, at 14 C, the extension of the pathogen was contained, and the jarrah behaved as though it were resistant to attack by *P. cinnamomi*. At 24 C, hyphal extension slowed after 3 days but still continued. At 17 C, (*unpublished*) many seedlings limited hyphal extension after 8–10 days, at 21 C fewer did so, while above 26 C none showed this response, although again hyphal extension slowed in older tissue.

Effect of inoculation site. Inoculation of marri at sites remote from the tip gave a lower rate of success as the distance increased at 16 and 24 C, but at 28 C all sites were susceptible (Table 3). There was also an increase in the time taken before a lesion appeared when inoculation was remote from the tip. In Fig. 3, the pattern of lesion development is shown in four individual roots inoculated at different sites. In roots inoculated 40 and 60 mm from the tip, growth continued for longer than with tip inoculations. However, root growth always ceased before the lesion reached the tip. When the lesion appeared it extended rapidly in the direction of the tip, but made little advance into the mature regions of the root (Fig. 3).

Inoculations at sites remote from the root tip were also made in roots grown at 16 and 28 C. In general, the results were similar to those illustrated in Fig. 3. There were two differences. At 16 C, inoculations made 40 and 60 mm from the tip produced lesions, but the development of the lesion was restricted to between 10 and 15 mm. When these roots were plated at 14 days, no fungus was recovered from the root tissue. At 28 C, tip inoculation produced a typical lesion which extended for 60–70 mm and then ceased, similar to the data shown in Fig. 6. However, in two seedlings in the trial of eight, hyphae were recovered for up to 50 mm beyond the lesion site in apparently healthy root.

DISCUSSION

The results show that two species of eucalypt, jarrah (*E. marginata*), a susceptible species, and marri (*E. calophylla*), a resistant species, differ in capacity to restrict root invasion by *P. cinnamomi*. Preliminary results with other species of eucalypt (*E. sieberi, E. maculata, E. obliqua*, and *E. botryoides*) (B. R. Grant, *unpublished*) suggest that these differences extend throughout the genus and correlate with the degree of field tolerance. Differences in rates of hyphal extension in the roots of resistant and susceptible eucalypt species have been found previously (5,9,10,28) and differences in the extent of root damage in *E. sieberi* (susceptible) and *E. maculata* (resistant) following infection by *P. cinnamomi* were reported by Halsall (9). However, our results show that in the resistant species the rate of extension of the pathogen is not only slower, but ceases completely in the majority of individuals. Results of one earlier study (3) showed initial differences in the capacity of jarrah and marri to resist invasion by *P. cinnamomi*, but since the plants were grown in water culture, continuing secondary infection of the root system resulted in the breakdown of resistance, a point recognized in the study. Secondary infection was not encountered under the conditions of these experiments, but it is not known whether zoospores were not produced or whether drainage prevented zoospore migration to potential infection sites.

Sporangial production has been noted in infected seedling tissues of nonhosts grown axenically on moist filter paper (33), which suggests that failure to infect new root tissue is probably not due to host suppression of sporulation. Results of studies with a wide variety of other plant species (12,32) show that *P. cinnamomi* invades both host and nonhost; these and our results indicate that differences in susceptibility are a result of events in the postinvasion phase.

Neither of the *Eucalyptus* species studied exhibited a typical hypersensitive response to *P. cinnamomi* even under conditions where the invasion was restricted. The pathogen always extended several centimeters within the root before hyphal growth ceased and there was no visible tissue damage at this point. This restriction was not the result of hyphal death, since the fungus was isolated from the zone above the lesioned area up to 1 mo after growth ceased. We have subsequently shown (B. R. Grant, *unpublished*) that in the nonhost, maize (*Zea mays*), in which lesion formation is much more restricted, a similar situation occurs. Recent reports of recovering *P. cinnamomi* from large roots of naturally infected jarrah (8,26) suggest that the pathogen survives in mature trees in the field without causing death of the tissue immediately surrounding the hyphae.

The slower penetration of older root tissue of marri, coupled with lower success in establishing infection, suggests that in this species there is constitutive resistance to invasion by *P. cinnamomi* in mature tissue. Although the effects of tissue age on the rate of invasion by *P. cinnamomi* was shown most clearly in marri, it was also evident in the jarrah seedlings, which also showed a decrease in the rate of hyphal extension in older tissue. Differences in the spread of *P. megasperma* var. *sojae* were also noted in soybean hypocotyl tissue of different ages (16,27).

The effect of temperature on the progress of the disease in both host species is more complex than a simple alteration in growth rate of the pathogen. Measurements of growth of this strain of *P. cinnamomi* on V-8 agar showed that growth extension took place between 5 and 35 C with optimum between 28 and 30 C (D. Phillips, *personal communication*). This is similar to the results obtained with other strains (36) and shows that, in *Eucalyptus*, host resistance to invasion at a root temperature of 31 C is not due to exceeding the thermal death point of the particular strain of *P. cinnamomi* being tested. Similarly, failure to colonize jarrah at 14 C cannot be due to an effect on the parasite alone, and it is clear that at low temperatures susceptibility gives way to resistance.

A temperature-induced alteration in the nonhost reaction to invasion has recently been reported in the interactions of soybean cultivars and *P. megasperma* (31). In this case, too, resistance gave way to susceptibility as temperature increased.

There are a number of cases in which temperature effects on susceptibility of host tissue have been noted (2,13,18,31,36). In two of these (13,18), accumulation of phytoalexin has been shown to be temperature sensitive, and the level of this accumulation correlated strongly with the expression of resistance.

Although our data at present could be interpreted by postulating the presence of a constitutive inhibitor of fungal growth, whose concentration is dependent upon species, tissue age, and temperature, they also show distinct similarities to results reported with systems in which the role of phytoalexins has been unequivocally established (1,2,16,19,27). Further investigation is required to establish whether there are increases in the concentration of fungitoxic compounds in root tissue of *Eucalyptus* in direct response to invasion. The discovery of a temperature-conditional resistance in at least one member of the genus will be of assistance in work on a species in which genetically defined lines are at present not available.

It has been recognized since the work of Kuhlman (14) and Roth and Kuhlman (24) that soil temperature plays a significant role in the development of diseases caused by *P. cinnamomi* (35). The effect of environmental conditions on attack by *P. cinnamomi* in jarrah forests was summarized by Shea (25). In previous studies, however, the emphasis has been on the effect of soil temperature on pathogen survival and sporulation. Our results show that soil, and hence root temperature, can play a major role in the capacity of *Eucalyptus* species to contain invasion after the initial infection has been established.

TABLE 3. The effect of temperature and location of the inoculation site on the establishment of infection[a] in marri roots after inoculation with *Phytophthora cinnamomi* at different locations

Site of inoculation (distance from tip [mm])	Pathogen recovered/inoculation[a]		
	16 C	24 C	28 C
0	6/6	6/6	6/6
20	2/4	6/6	4/4
40	1/4	4/4	4/4
60	1/6	1/4	4/4

[a] Mycelial inoculum was used throughout.

LITERATURE CITED

1. Bailey, J. A., and Rowell, P. M. 1980. Viability of *Colletotrichum lindemuthianum* in hypersensitive cells of *Phaseolus vulgaris*. Physiol. Plant Pathol. 17:341-345.
2. Bailey, J. A., Rowell, P. M., and Arnold, G. M. 1980. The temporal relationship between host cell death, phytoalexin accumulation and fungal inhibition during hypersensitive reactions of *Phaseolus vulgaris* to *Colletotrichum lindemuthianum*. Physiol. Plant Pathol. 17:329-339.
3. Batini, F. E. 1974. Susceptibility of *Eucalyptus marginata* and *Eucalyptus calophylla* seedlings to infection by *Phytophthora cinnamomi* in nutrient solution. Forests Department, Western Australia, Res. Pap. 14.
4. Byrt, P. N., and Grant, B. R. 1979. Some conditions governing zoospore production in axenic cultures of *Phytophthora cinnamomi* Rands. Aust. J. Bot. 27:103-115.
5. Byrt, P. N., and Holland, A. A. 1978. Infection of axenic *Eucalyptus* seedlings with *Phytophthora cinnamomi*. Aust. J. Bot. 26:169-176.
6. Cahill, D., and Weste, G. 1983. The formation of callose deposits as a response to infection with *P. cinnamomi*. Trans. Br. Mycol. Soc. 80:23-29.
7. Dawson, P., and Weste, G. 1982. Changes in water relations associated with infection by *P. cinnamomi*. Aust. J. Bot. 30:393-400.
8. Dell, B., and Wallace, I. M. 1981. Recovery of *Phytophthora cinnamomi* from naturally infected jarrah roots. Austr. Plant Pathol. 10:1-2.
9. Halsall, D. M. 1978. A comparison of *Phytophthora cinnamomi* infection in *Eucalyptus sieberi*, a susceptible species, and *Eucalyptus maculata*, a field-resistant species. Aust. J. Bot. 26:643-655.
10. Halsall, D. M. 1980. Calcium nutrition and the infection of eucalypt seedlings by *Phytophthora cinnamomi*. Aust. J. Bot. 28:19-25.
11. Havel, J. J. 1975. Site-vegetation mapping in the northern jarrah forest (Darling Range). 1. Definition of site-vegetation types. Western Australian For. Dep. Bull. 86.
12. Hinch, J., and Weste, G. 1979. Behaviour of *Phytophthora cinnamomi* zoospores on roots of Australian forest species. Aust. J. Bot. 27:679-691.
13. Holliday, M. J., Long, M., and Keen, N. T. 1981. Manipulation of the temperature-sensitive interaction between soybean leaves and *Pseudomona syringae* pv. *glycinea*—Implications on the nature of determinative events modulating hypersensitive resistance. Physiol. Plant Pathol. 19:209-216.
14. Kuhlman, E. G. 1964. Survival and pathogenicity of *Phytophthora cinnamomi* in several western Oregon soils. For. Sci. 10:151-158.
15. Ladiges, P. 1977. Differential susceptibility of two populations of *Eucalyptus viminalis* Labill to iron chlorosis. Plant Soil 48:581-597.
16. Lazarovits, G., Stössel, P., and Ward, E. W. B. 1981. Age-related changes in specificity and glyceollin production in the hypocotyl reactions of soybeans to *Phytophthora megasperma* var. *sojae*. Phytopathology 71:94-97.
17. Malajczuk, N., McComb, A. J., and Parker, C. A. 1977. Infection by *Phytophthora cinnamomi* Rands of roots of *Eucalyptus calophylla* R. Br. and *Eucalyptus marginata* Donn ex Sm. Aust. J. Bot. 25:483-500.
18. Mayama, S., Hayeshi, S., Yamamoto, R., and Tarna, T. 1982. The effects of elevated temperature and α-aminooxyacetate on the accumulation of avenalumins in oat leaves infected with *Puccinia coronata* f. sp. *avenae*. Physiol. Plant Pathol. 20:305-312.
19. Molot, P. M., Mas, P., Conus, M., and Ferriere, H. 1981. Relations between capsidiol concentration, speed of fungal invasion and level of induced resistance in cultivars of pepper (*Capsicum annuum*) susceptible or resistant to *Phytophthora capsici*. Physiol. Plant Pathol. 18:379-389.
20. Palzer, C. 1976. Zoospore inoculum potential of *Phytophthora cinnamomi*. Ph.D. thesis, University of Western Australia, Nedlands.
21. Podger, F. D. 1972. *Phytophthora cinnamomi*, a cause of lethal disease in indigenous plant communities in West Australia. Phytopathology 62:972-981.
22. Podger, F. D., and Batini, F. 1971. Susceptibility to *Phytophthora cinnamomi* root rot of thirty-six species of eucalypts. Aust. For. Res. 5:9-20.
23. Pryor, L. D., and Johnson, L. A. S. 1971. A Classification of the Eucalypts. Aust. National University Press, Canberra. 102 pp.
24. Roth, L. F., and Kuhlman, E. G. 1966. *Phytophthora cinnamomi*, an unlikely threat to Douglas-fir forestry. J. For. 61:199-205.
25. Shea, S. R. 1975. Environmental factors of the northern jarrah forest in relation to pathogenicity and survival of *Phytophthora cinnamomi*. For. Dep. Bull. 85: Perth, WA. 85 pp.
26. Shearer, B. L., Shea, S. R., and Fairman, R. G. 1981. Infection of the stem and large roots of *Eucalyptus marginata* by *Phytophthora cinnamomi*. Austr. Plant Pathol. 10:2-3.
27. Stössel, P., Lazarovits, G., and Ward, E. W. B. 1980. Penetration and growth of compatible and incompatible races of *Phytophthora megasperma* var. *sojae* in soybean hypocotyl tissue of different ages. Can. J. Bot. 58:2594-2601.
28. Tippett, J. T., Holland, A. A., Marks, G. C., and O'Brien, T. P. 1976. Penetration of *Phytophthora cinnamomi* into disease tolerant and susceptible eucalypts. Arch. Microbiol. 108:231-242.
29. Tippett, J. T., O'Brien, T. P., and Holland, A. A. 1977. Ultrastructural changes in eucalypt roots caused by *Phytophthora cinnamomi*. Physiol. Plant Pathol. 11:279-286.
30. Tsao, P. H., and Ocana, G. 1969. Selective isolation of species of *Phytophthora* from natural soils on an improved antibiotic medium. Nature (Lond.) 223:636-638.
31. Ward, E. W. B., and Lazarovits, G. 1982. Temperature induced changes in specificity in the interaction of soy beans with *Phytophthora megasperma* f. sp. *glycinea*. Phytopathology 72:826-830.
32. Weste, G. 1975. Comparative pathogenicity of root parasites to wheat seedlings. Trans. Br. Mycol. Soc. 64:43-53.
33. Weste, G., and Cahill, D. 1982. Changes in root tissue associated with infection by *Phytophthora cinnamomi*. Phytopathol. Z. 103:97-108.
34. Weste, G., Cook, D., and Taylor, P. 1973. The invasion of native forest by *Phytophthora cinnamomi*. II. Post-infection vegetation patterns, regeneration, decline in inoculum and attempted control. Aust. J. Bot. 21:31-39
35. Zentmyer, G. A. 1980. *Phytophthora cinnamomi* and the diseases it causes. Phytopathol. Monogr. 10, American Phytopathological Society, St. Paul, MN. 96 pp.
36. Zentmyer, G. A. 1981. The effect of temperature on growth and pathogenesis of *Phytophthora cinnamomi* and on growth of its avocado host. Phytopathology 71:925-928.