Phytophthora cinnamomi, a Cause of Lethal Disease in Indigenous Plant Communities in Western Australia

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ABSTRACT

In southwestern Australia, more than 80,000 hectares of *Eucalyptus* forest have been destroyed by *Phytophthora cinnamomi*. *Eucalyptus marginata* and many other native plants have been killed in forest, woodland, and heath communities. Isolates of the A_2 strain of *P. cinnamomi* were obtained from wildlings of 55 species in 34 genera among 15 families. Of 49 isolates tested, 48 were pathogenic to *E. marginata* seedlings. Each of nine provenances of *E. marginata* tested was susceptible to a local isolate (I.M.I. 124492) of *P. cinnamomi*. Typical symptoms developed in healthy forest after the soil was inoculated with pure cultures of *P. cinnamomi*, and also with soil taken from beneath nearby diseased forest. No symptoms developed after inoculation with soil taken from unaffected forest.

Apparently, *P. cinnamomi* has been introduced recently and is being dispersed with soil moved during road building and logging operations. Thus, the flora of southwestern Australia is being subjected to a new and powerful force of selection which will greatly affect the further evolution of its composition and character. There appear to be no practicable methods of restricting infestations within their present boundaries, although sanitation measures have been applied to reduce the rate of establishment of new centers of infection.

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Additional key words: disease expression climate and seasonal growth, infection on well-drained soils.

Extensive areas of natural forest in southwestern Australia are afflicted with a destructive disease known as "jarrah dieback" (Fig. 1). The disease occurs in patches (Fig. 2), often bounded sharply by healthy forest. First symptoms are chlorosis and rapid death of many plants of the understory and shrub layers, including many of the state's finest wildflowers. Chlorosis, twig dieback, and death of the stand-dominant *Eucalyptus marginata* Donn. ex Sm. follow, in some cases rapidly and in others after many years. There is a progressive decline in the numbers of plants of susceptible species and a very slow recolonization by the few resistant species. The disease converts evergreen forest to floristically impoverished and open communities of greatly reduced productivity.

The disease, when first noticed in 1921, appeared to be of recent origin and was restricted to a few small areas in the northern part of the forest (W. R. Wallace, *personal communication*). It now occurs throughout the forest range of *E. marginata* in patches of variable size totalling 80,000 hectares of the ca. 1,400,000 hectares of jarrah in state forest reserves. New patches continue to appear, and old ones spread so that the affected area increases by 4%/year.

The continual spread of jarrah dieback causes serious problems of forest management, since *E. marginata* provides 70% of the raw material for Western Australia's forest industry, and affords protective cover to the more important of that state's water catchment areas.

Some earlier descriptions of the disease and speculation as to its cause have been recorded in several unpublished documents held by the Forests Department, Perth, Western Australia (J. H. Harding, 1949; H. D. Waring, 1950; C. D. Hamilton, 1951; W. R. Wallace & A. B. Hatch, 1953). It was reported that the disease was restricted to forest of poor quality on infertile soils, and occurred only in specific topographic situations. As there were no signs of any known pathogen, jarrah dieback was attributed to disturbance of the environment following timber felling, and to those changes in the frequency and severity of forest fires which followed timber felling and the advent of forest management (17). There were various suggestions that such changes might have caused watertables to become less stable, soil nutrient levels to be depleted, toxic levels of ions to accumulate, or insolation at the forest floor to increase to harmful levels.

Until 1960, only the nutrient decline hypothesis had been examined in detail. Small differences were found in nutrient levels of soils and foliage between affected and healthy forest, but it was concluded that these differences were the consequences of death of the forest stand rather than its cause. I reviewed the evidence for increased soil salinity, metal ion toxicity, drought, and waterlogging, and found it inadequate to account for the disease over the entire range of its occurrence.

In 1962, two observations led me to consider the role of root rotting organisms. The first was the development of dieback alongside newly constructed

roadways and its rapid extension into undisturbed forest; the second was the coincidence of deaths of *Pinus radiata* D. Don in shelterbelts with dieback of adjacent jarrah forest at a number of places on the Swan Coastal Plain. The symptoms in pine shelterbelts were identical with those which Newhook (12) attributed to *Phytophthora* spp. Attempts to isolate *Phytophthora* from roots of diseased jarrah, however, were at first unsuccessful. I then demonstrated, in a series of greenhouse and field experiments (13), that soils from forest affected by dieback contained a lethal factor(s) which was not present in soil from unaffected forest. This factor was lethal to *E. marginata* and to several other susceptible species, but had little effect on a number of field-resistant species. Its effects were transmitted in roots and stems of jarrah seedlings, but not in leaves. It was capable of spread uphill on a slope of 30 degrees. The factor was removed entirely by steaming, and its effects were suppressed by fungicidal treatment.

In October 1964, G. A. Zentmyer isolated *Phytophthora cinnamomi* Rands, type A2 (7), from soil beneath dying jarrah. A first account of the disease and its association with *P. cinnamomi* followed (15); this was the first record of the fungus in Western Australia. Subcultures of another Zentmyer isolate, P. 309, from dying jarrah seedlings have been lodged at the Commonwealth Mycological Institute, Kew, Surrey, England (I.M.I. 124492), at the Department of Agriculture, South Perth, Western Australia (WA 422), and at the Forest Research Institute, Kelmscott, Western Australia (C39).

In this paper, the disease and its occurrence are more fully described, similar disease in a number of other indigenous communities in southwestern Australia is reported, proof of the pathogenicity of *P. cinnamomi* is presented, the potential destructiveness of the disease is considered, and the prospects for its control are evaluated.

MATERIALS AND METHODS.—Descriptions of symptoms and of the occurrence of the disease in relation to environmental factors are based on observations over 7 years. Three extensive surveys were made throughout the jarrah forest. Data on the relative susceptibility of the various species and size classes were obtained by recording plant numbers and mortalities and by measuring the heights and girths of all plants of 20 species on transects in 21 severely affected forest and woodland communities. The transects totalled 10.8 hectares, and covered a wide range of variation of stand height, stand structure, species composition, and geographic origin.

Tests for constant association of *P. cinnamomi* with the disease were made in two ways. Root and stem tissues of a wide range of naturally occurring plants with advanced symptoms were surface-sterilized and plated on selective medium (6). Soil samples from 39 affected and 33 unaffected stands nearby were tested for *P. cinnamomi* by baiting with lupin radicles (2). From each stand, five samples were obtained. Each was a composite of five subsamples of roots and soil from the 0- to 15-cm level. Each sample was mixed and about 300 g were wetted and baited for 24 hr with 10 newly germinated seed of blue lupin (*Lupinus angustifolium* L.). Samples which did not produce lesions on the roots of the lupin baits were exposed for a further 24 hr. The roots of all lupins were then excised and plated on selective medium. This was necessary because infected lupin radicles did not always produce the visible lesions described by Chee & Newhook (2).

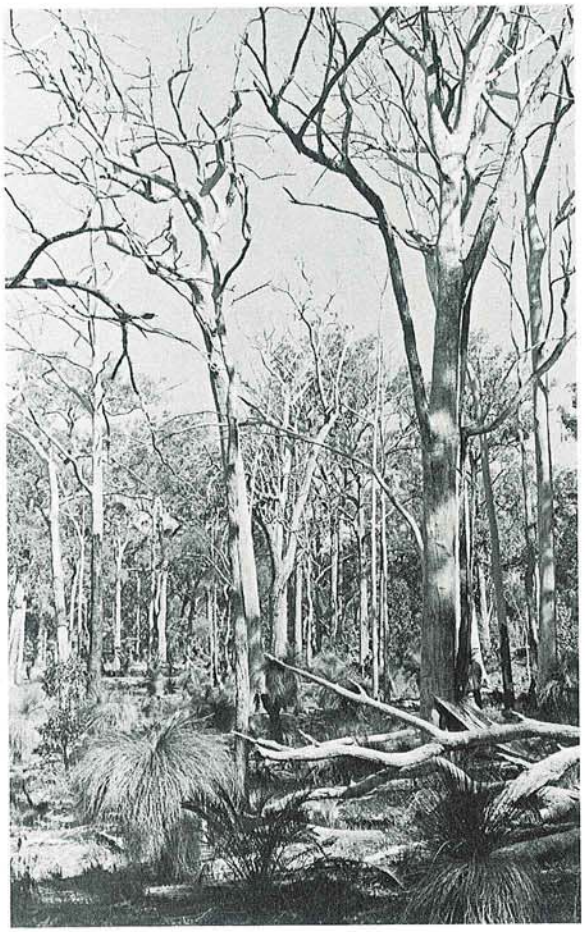

Fig. 1. Dieback of jarrah (*Eucalyptus marginata*). The dense understory of *Banksia grandis* (normally present in healthy forest, see left background) has been killed and its remnants have decayed. The "grass trees" are *Xanthorrhoea preissii*, one of which (left foreground) has recently died, as indicated by the collapsed crown. In the center foreground is an unhealthy specimen of the cycad *Macrozamia reidlei*.

The identity of isolates of *P. cinnamomi* was checked by comparing growth rates and mycelial characters with those of I.M.I. 124492 after 96 hr of growth at 26 C on 10 ml of potato-dextrose agar (PDA).

Pathogenicity tests were made on seedlings in a greenhouse and on trees in a mature jarrah community. In the greenhouse, pathogenicity tests were made with (i) 49 Western Australian isolates on

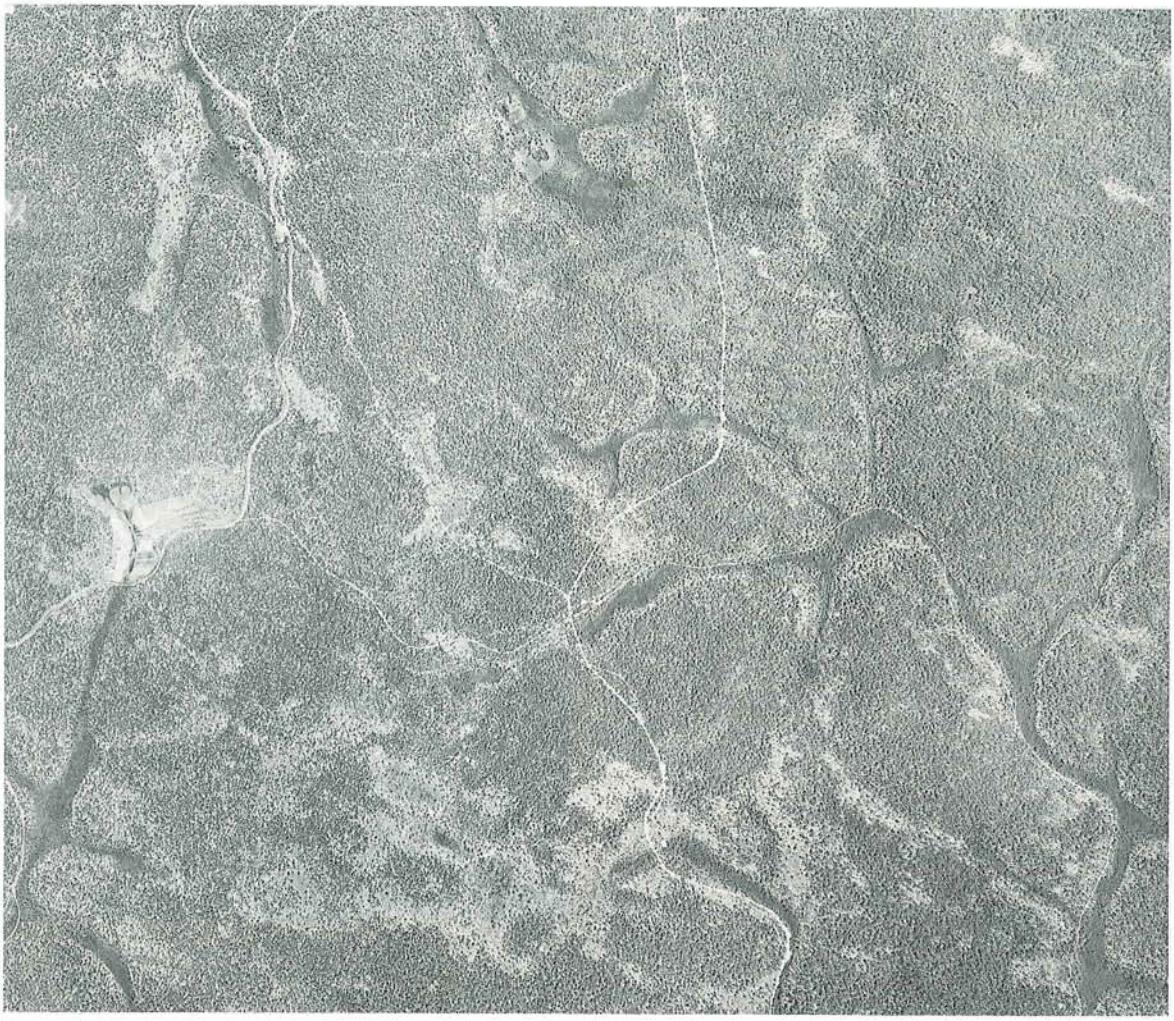

Fig. 2. Aerial view from 6,700 m above jarrah forest near Willowdale. Photographed 19 January 1965 by Western Australian Department of Lands and Survey (photograph No. WA909 Pinjarra Run 17 No. 5063 6 inches). The dark gray, closely matted areas are swamps and flats along watercourses with a narrow fringe of field resistant *Eucalyptus megacarpa* and *E. patens*. The greater part of the area is covered by an *E. marginata-E. calophylla* community with a dense understory of *Banksia grandis*. Healthy stands are midgray and of granular texture. Pale-gray areas of blotchy texture are dieback-affected. The pale tone is due to increased light reflection from the forest floor after death of the *Banksia* understory. White lines indicate the location of roads and tracks. The pattern of association of dieback with tracks and drainage lines may be seen over much of the area.

a single source of *E. marginata*; (ii) a single isolate, I.M.I. 124492, on nine different sources of *E. marginata*; (iii) one isolate, I.M.I. 124492, on 16 other indigenous species. All test plants were grown from seed in freely drained 6-inch pots of steam-sterilized sand. Pure cultures for inoculation were raised on V-8 juice agar, cornmeal agar (CMA), or PDA. At the end of each experiment, root pieces from both inoculated and control treatments were plated on Eckert & Tsao's medium (6). Field tests were made by inoculating soil beneath healthy forest with pure cultures of *P. cinnamomi* and with soil transferred from diseased forest.

RESULTS.–Symptoms, in *E. marginata* and other species, typical of jarrah dieback were found in many plant communities and in a wide range of environments in southwestern Australia.

Symptoms in E. marginata.–In some apparently healthy trees, death occurs suddenly, but mostly there is a gradual deterioration of crown health with chlorosis, dieback of small branches, microphylly of new leaves, and the replacement of the primary leaf-bearing system by epicormic shoots on large branches and stems. The epicormic shoots, in turn, deteriorate. Such trees may live for many years. Mortality in 79 trees averaged 5%/year over 2 years, and at another place, the rate in 79 trees averaged 1.7%/year over 6 years. At a third locality, 4 of 17

marked trees died in 4 years. The growth of diseased trees is very slow. Girth increment on six diseased trees averaged only 0.08 cm/year for 3 years compared with an average of 0.63 cm/year for the same period on 10 trees of similar size nearby in healthy forest.

Symptoms in other species.—The initial symptom in all species is severe and rapid foliar chlorosis. In the case of perennials with rhizomes, such as *Dasypogon* sp. and *Dryandra nivea* R. Br., some shoots may develop severe chlorosis while others remain apparently healthy. In species with woody, underground, perennating organs; e.g., *Podocarpus drouyniana* F. Muell., *Adenanthos* sp., and *Lasiopetalum floribundum* Benth., chlorosis is followed by dieback of the parts aboveground and their replacement by stunted shoots bearing dwarfed and chlorotic foliage.

In *Banksia grandis* Willd., twig dieback is uncommon; death of the entire tree usually follows soon after the first signs of chlorosis. Where the course of the disease is protracted, there is premature basipetal leaf-shed and stunted development of the new season's foliage; the trees then have a hollow rosetted crown of strongly recurved and dull leaves. Few trees live for more than 3 years after the first appearance of symptoms.

In some hosts, root rot is largely confined to small roots, but in chlorotic *B. grandis*, roots up to 10-cm in diam may be rotted. In many plants of the shrub layer, extensive root and collar rot are common.

Similar disease in other communities.—Symptoms similar to those in jarrah forest were found in a number of other indigenous communities: (i) sclerophyll shrub woodland where there is mass dying in the overstory and shrub layers; *Banksia* sp., *Casuarina fraseriana* Miq., *E. marginata, E. todtiana* F. Muell., *Xanthorrhoea preissii* Endl., *Macrozamia reidlei* (Gaud.) C. A. Gardn., *Casuarina humilis* Otto & Dietr., *Adenanthos obovata* Labill., *Petrophila* sp., *Hibbertia* sp., and *Leucopogon* sp. are all severely affected; but *Melaleuca parviflora* Lindl. and *Nuytsia floribunda* (Labill.) R. Br. are apparently unaffected; (ii) low sclerophyll shrubland on skeletal soils where *Casuarina humilis, Petrophila biloba* R. Br., *X. preissii, Hovea pungens* Benth., *Macrozamia reidlei, Leucopogon polymorphus* Sond., *Verticordia plumosa* (Desf.) Domin., *Verticordia huegelii* Endl., and *Hibbertia subvaginata* (Stend.) Ostf. are killed; (iii) moorland heaths on peaty sands where stunted *E. marginata, Banksia littoralis* R. Br., *Podocarpus drouyniana, Adenanthos obovata, Pultenaea* sp., *Aotus passerinoides* Meissn., and *Sphenotoma squarrosum* (R. Br.) G. Don. are killed; (iv) *Melaleuca parviflora-E. rudis* association in seasonal swamps where *Thomasia pauciflora* Lindl., *Leucopogon australis* R. Br., *Aotus ericoides* (Vent.) G. Don., *Patersonia occidentalis* R. Br., *Adenanthos obovata, Xanthorrhoea gracilis* Endl., *Verticordia densiflora* Lindl., and *Leptospermum ellipticum* Endl. are killed, whereas *Melaleuca parviflora, E. rudis, Nuytsia floribunda, Lepidosperma tetraquetrum* Nees, and *Acacia urophylla* Benth. are relatively resistant; (v) dry sclerophyll forest in *E. wandoo-E. marginata* association where *E. wandoo* Blakely and *E. calophylla* R. Br. ex Lindl. are little affected, but *E. marginata, Banksia grandis, Xanthorrhoea preissii, Macrozamia reidlei,* and *Casuarina humilis* are killed. (vi) Wet sclerophyll forest, where the stand dominants, *E. diversicolor* F. Muell. and *E. calophylla*, are apparently unaffected, but chlorosis and death of *Banksia grandis, Macrozamia reidlei,* and a *Leucopogon* sp. occur.

The effect of the disease on the communities varies considerably, depending upon the relative abundance of field resistant species. In extreme cases, communities may be almost entirely destroyed.

Relative susceptibility of species.—Among the forest eucalypts, only the dominant species *E. marginata* is highly susceptible. Of 2,018 trees taller than 1.8 m, 68% were *E. marginata*; of these, 52% were dead and the remainder mainly affected severely. Although few *E. calophylla* were dead, this common associate of *E. marginata* is frequently chlorotic, slow to recolonize dieback areas, and does not grow as well as in unaffected forest. The other five forest eucalypts encountered, *E. patens* Benth., *E. wandoo, E. megacarpa* F. Muell., *E. rudis* Endl., and *E. laeliae* Podger & Chippendale are apparently unaffected. They are minor species of limited occurrence, and do not commonly colonize other sites where jarrah has died. Throughout the region, tree species of the Proteaceae, which form the forest understory or dominate low woodland, are highly susceptible; of 1,505 *Banksia grandis* trees taller than 1.8 m, 92% were dead. In each of *Persoonia longifolia* R. Br., *B. menziesii* R. Br., *B. ilicifolia* R. Br., *B. littoralis* R. Br., and *B. attenuata* R. Br., more than 65% of all trees taller than 1.8 m were dead. Among other minor tree species, *Casuarina fraseriana* (15% of all trees dead) and *E. todtiana* (13%) were susceptible, whereas *Nuytsia floribunda* and *Melaleuca parviflora* were unaffected. In the large shrub layer, *Casuarina humilis* (46%), *Macrozamia reidlei* (27%), and *Xanthorrhoea preissii* (25%) were susceptible. In the small shrub layer, many species in Proteaceae, Epacridaceae, Dilleniaceae, Papilionaceae, and Myrtaceae were highly susceptible. Members of Restionaceae and Cyperaceae were generally unaffected. There seemed to be no association of susceptibility with occurrence in particular habitats; e.g., *B. verticillata*, which is restricted to well-drained sites, *B. littoralis*, restricted to poorly drained swamp sites, and *B. attenuata*, restricted to deep, well-drained sands, are all highly susceptible.

Evidence that the susceptibility of *E. marginata* and *Banksia* spp. temporarily decreases after the seedling stage is shown in the lower rate of mortality of trees less than 1.8 m tall. Chi-square analysis of the height class distribution of mortalities in samples of 6,093 *E. marginata* and 2,248 *Banksia grandis* individuals indicates that a high level of susceptibility is reached at 6 m and 1.8 m, respectively, and is not influenced by further increases in height (13).

Occurrence of the disease.—The disease occurs in

forest, woodland, shrub, and heath communities of sclerophyllous evergreens south of latitude 30°S and west of longitude 118°E in an area bounded on the northeast by the 500 mm isohyet.

Throughout this region, the climatic type is regular Mediterranean with cool wet winters and hot dry summers. Jarrah dieback is found throughout the range of climate in which *E. marginata* occurs. Although it is most extensive in those parts of the forest which have both high levels of winter rainfall and high summer temperatures, it also has been found in areas whose mean annual rainfall is as low as 625 mm.

The disease affects communities in each of the major physiographic subdivisions and in almost every geomorphic unit within them. It is also found in almost the entire range of topographic situations, but is much more frequent along drainage lines and in broad valleys than on upper slopes and ridges. The incidence of dieback in 2,800 hectares of forest in the South Dandalup catchment was found to be independent of aspect and of slope up to 10 degrees. Although the disease may be found on slopes up to 25 degrees, the deeply dissected valleys of the major river systems, to which such steep slopes are generally confined, are almost entirely free of dieback.

In most of the area, the soils are lateritic podzolics, infertile, moderately acidic (pH 5.5-6.5), and low in exchangeable cations. Texture, soil depth, and drainage vary, but shallow sands, silts, and gravelly sands over indurated laterite or dense clays predominate. Although dieback may be found in the entire range of soil types, including deep, well-drained gravels and aeolian sands (13), it is not common on the red and yellow podzolics of the major river valleys.

Similarly, there seems to be no particular intensity of cutting or fire, level of total biomass, stand structure, or species composition in which the disease may not be found (13). There is, however, a marked tendency for it to be most extensive in areas with a history of frequent or recent utilization. The largest areas which remain essentially unaffected are among those which have not been logged or intensively roaded, and those which were logged before the widespread use of heavy earth-moving machinery. Aerial photographs show a strong association between the occurrence of dieback and roadways. Though much of the road system in the forest follows gentle terrain close to drainage lines, dieback is also present along direct routes constructed across ridge systems (Fig. 2).

That the disease is not due to the direct effects of logging is suggested by (i) its absence in extensive areas which have been logged heavily; (ii) its recent development in areas of dense, vigorous, second-growth forest resulting from cutting more than 50 years ago; and (iii) its development in areas which have never been logged. Although a few patches of dieback have been located in areas remote from logging or roading, in each case there was evidence of the passage of vehicles or heavy equipment in the course of fire suppression, mining exploration, or firewood cutting.

Spread of the disease.—Spread of dieback patches outward from apparent foci of infection is evident on aerial photographs taken in 1951, 1960, and 1965. There is also evidence of spread in the gradients of disease severity within patches. This is seen in both the severity of symptoms in jarrah and in the reduced numbers of plants of susceptible species inwards from the boundary with healthy forest.

The rate and pattern of spread may be extremely irregular. The boundary at one place may appear to have stabilized for several years while it extends rapidly at another; later the apparently dormant boundary may extend. Rates of spread are most rapid down drainage lines, especially on poorly drained sites. On such sites in the southern part of the forest, some boundaries have extended up to 40 m in a year. On gently undulating terrain in the north, spread is much slower, especially uphill. Spread along the contour on well-drained soil in one such place was 3.6 m/year during 30 months of measurement. On well-drained soils, even downhill spread may be relatively slow; on a small, gently sloping dune of deep, freely drained Gavin sand, dieback in *Banksia* woodland spread only 40 m from the crest in 5 years.

The importance of high soil moisture levels in the development of this disease is suggested by its frequent occurrence near and rapid spread from culverts and drains along roadways; and by its rapid spread and severe effects around bores where water from deep mines is pumped onto the forest floor.

Constant association of P. cinnamomi with the disease.—*P. cinnamomi* was isolated from soil beneath diseased communities at each of the 39 locations tested. At the 29 jarrah forest and 4 *Banksia* woodland locations where it was also possible to obtain samples in healthy communities nearby, *P. cinnamomi* was isolated from the diseased community, but in no case was it detected in samples from healthy stands.

P. cinnamomi was isolated from surface-sterilized tissues of wildlings of the following 55 species of indigenous plants in 15 families: Cycadaceae, *Macrozamia reidlei* (Gaud) C. A. Gard.; Podocarpaceae, *Podocarpus drouyniana* F. Muell.; Xanthorrhoeaceae, *Lomandra* sp.; Haemodoraceae, *Conostylis setigera* R. Br.; Casuarinaceae, *Casuarina fraseriana* Miq.; Proteaceae, *Adenanthos obovata* Labill., *Banksia attenuata* R. Br., *Banksia grandis* Willd., *Banksia ilicifolia* R. Br., *Banksia littoralis* R. Br., *Banksia menziesii* R. Br., *Banksia quercifolia* R. Br., *Isopogon attenuatus* R. Br., *Isopogon formosus* R. Br., *Persoonia longifolia* R. Br., *Xylomelum occidentale* R. Br.; Leguminosae, *Acacia huegellii* Benth., *Aotus ericoides* G. Don., *Aotus passerinoides* Meissn., *Bossiaea eriocarpa* Benth., *Burtonia conferta* DC., *Dillwynia uncinata* (Turcz.) C. A. Gardn., *Hovea elliptica* (Sm.) DC., *Pultenaea reticulata* Benth., *Pultenaea* sp.; Tremandraceae, *Tetratheca viminea* Lindl.; Euphorbiaceae, *Amperea ericoides* A. Juss.; Sterculiaceae, *Lasiopetalum floribundum* Benth., *Thomasia grandiflora* Lindl., *Thomasia pauciflora* Lindl.; Dilleniaceae, *Hibbertia acerosa* (R. Br.)

Benth., *Hibbertia cunninghamii* (Benth.) Steud., *Hibbertia subvaginata* (Steud.) Ostf., *Hibbertia vaginata* (Benth.) F. Muell., *Hibbertia* sp.; Myrtaceae, *Agonis hypericifolia* Schau., *Beaufortia sparsa* R. Br., *Calythrix flavescens* A. Cunn., *Eucalyptus marginata* Donn. ex Sm., *Hypocalymma angustifolium* Endl., *Hypocalymma cordifolium* Lehm. Schau., *Hypocalymma robustum* Endl., *Leptospermum ellipticum* Endl., *Verticordia densiflora* Lindl., *Verticordia huegellii* Endl., *Verticordia plumosa* Domin.; Apiaceae, *Platysace compressa* (Labill.) Norman.; Epacridaceae, *Leucopogon australis* R. Br., *Leucopogon glabellus* R. Br., *Leucopogon lasiostachyus* Stschegl., *Leucopogon pulchellis* Sond., *Leucopogon verticillatus* R. Br., *Monotoca tamarascina* F. Muell., *Sphenotoma squarrosum* (R. Br.) G. Don.; Goodeniaceae, *Dampieria linearis* Lindl. The fungus was isolated mainly from root tissues, but also from stem tissues up to 22 cm above the root collar in *Adenanthos obovata, Thomasia* spp., *Hibbertia* spp., *Hypocalymma* spp., and *Leucopogon* spp. Isolates were obtained from both woody tissues and living bark in the roots and lignotubers of older *E. marginata* seedlings. In late summer, *P. cinnamomi* was obtained from *Banksia* spp. only in roots 5-mm in diam or larger. Examination of thin sections showed that hyphae had invaded the pith of woody roots via the medullary rays. I confirmed the identity of the invading mycelium as *P. cinnamomi* by dissecting portions of the thin sections and plating them on Eckert & Tsao's medium (6).

Pathogenicity tests of 49 isolates against jarrah.—Jarrah seedlings were grown from seed collected from high-quality forest at a single location near Plavins. After 12 weeks' growth, single pots containing 7-19 seedlings were inoculated, each with one of the 49 isolates of *P. cinnamomi*. Twenty-seven isolates from 24 hosts in the jarrah forest, eight from hosts in *Banksia* woodland, twelve from lupin baiting of jarrah forest soils, one from soil beneath *E. diversicolor* forest, and one from beneath a dying *Pinus pinaster* Aitch. shelterbelt were tested.

Each pot was inoculated with a 6-day-old culture grown on 10 ml of PDA which was cut into 8 pieces; these were placed at 1- to 3-cm depth amongst the seedlings. Twenty pots were similarly treated with noninoculated PDA as control.

At 150 days, death of plants had occurred in all inoculated pots excepting that inoculated with the *Banksia ilicifolia* isolate, and in all surviving plants there was extensive root rot from which *P. cinnamomi* was reisolated. In the control plots there were no deaths and no recoveries of *P. cinnamomi*.

Pathogenicity tests with nine provenances of E. marginata.—Seed was collected at nine places throughout the range of *E. marginata*, including three outliers. The sources included stunted populations in extreme habitats such as those on wet moorland heaths at Wye Plains, and on a dry site on Mt. Lesueur. There were two pots for each provenance. After 20 weeks of growth, one pot of each provenance was inoculated with isolate I.M.I. 124492 in the manner previously described.

The experiment was terminated 16 weeks after inoculation, and the root systems were assessed for root rot by three observers. In all provenances, the mean degree of root rot was much higher in the inoculated treatment. There were, however, considerable differences between provenances in the degree of root rot. *P. cinnamomi* was recovered from root tissues in each of the inoculated pots, but not from any control pot.

Pathogenicity tests with 16 indigenous species.—Seed of each of the species listed in Table 1 was sown on each of two pots. One pot of each species was inoculated with I.M.I. 124492 (on 5 ml of CMA) 60 days after the slowest species had germinated, and again at 150 days. Controls were similarly treated with 5 ml of noninoculated CMA. At 220 days, all plants were assessed for root rot by three observers (Table 1).

For fourteen of the species, the relative degree of root rot is consistent with the relative susceptibilities of the species in the field. The data for *X. preissii* are surprising, as are the unexpectedly high levels of root rot in *E. megacarpa* and in the control pots of *E. wandoo, Casuarina humilis,* and *X. preissii*. The reasons for these results and for the failure to reisolate *P. cinnamomi* from two of the three *Banksia* species are not understood.

Field inoculation tests.—Three healthy stands were selected in poor-quality forest of mean dominant height 21-24 m, growing in shallow, coarse, gravelly sand, over massive laterite on gently undulating terrain of 240- to 300-m elevation. So that large areas of unaffected forest would not be endangered, the test stands were located in areas of generally high disease incidence, but isolated from diseased areas by at least 100 m of healthy forest.

Fifty-two plots were located among thickets of susceptible species, 20 plots each in two stands, and twelve in the third. Each plot was 3 m in diameter and separated from other plots by at least 15 m. In each stand, all plots were ranked according to the total number of plants of susceptible species, then stratified into groups of four plots as close as possible to uniform stocking. One replicate of each of four treatments was randomly assigned to each group.

The treatments were: (i) control, inoculation holes prepared but no inoculum added; (ii) inoculation with soil from unaffected forest; (iii) inoculation with soil from diseased forest; (iv) inoculation with pure culture of isolate I.M.I. 124492.

P. cinnamomi was incubated for 13 days at 26 C in 800 ml of pea broth prepared according to the method of Chee & Newhook (2). The stock was blended for 10 sec and diluted to prepare 130 lots of 250 ml, each containing 6 ml of stock culture. Noninoculated pea broth was used as control.

Soil was obtained from 0- to 15-cm depth around chlorotic plants near the active edge of a dieback patch. Coarse gravel was screened from the soil, but all large roots were broken and returned to the stock of inoculum. Roots and soil were mixed, and 260 lots of 400 ml were prepared. Soil from unaffected forest

TABLE 1. Pathogenicity tests with *Phytophthora cinnamomi* isolate I.M.I. 124492 against 16 indigenous species of varying field susceptibility

Species	Field[a] susceptibility	Mean root rot[b]		Recovery of *P. cinnamomi* from inoculated plants	
		Inoculated	Control	Pieces plated	Positive
Banksia grandis	VH	3.8	2.3	22	0
Banksia menziesii	VH	3.2	2.0	2	0
Banksia quercifolia	VH	4.1	1.0	18	11
Xylomelum occidentale	VH	3.2	2.1	5	0
Casuarina humilis	VH	5.0	2.9	176	81
Beaufortia sparsa	H	3.8	0.0	17	6
Xanthorrhoea preissii	H	3.0	3.0	38	0
Macrozamia reidlei	H	3.0	1.5		
Eucalyptus todtiana	H	4.2	1.8	43	27
E. patens	L	2.0	1.6	8	0
E. calophylla	L	2.7	2.4	9	0
E. wandoo	L	3.0	3.0	40	0
E. megacarpa	L	3.0	2.1	27	11
E. rudis	L	2.1	2.0	26	0
E. laeliae	L	2.1	2.3	20	0
E. haematoxylon	L	2.0	2.0	14	0

[a] VH = very high; H = high; L = low.
[b] Root rot scale 0, 1 = 1-5%; 2 = 6-20%; 3 = 20-50%; 4 = 50-90%; 5 = 90%+.

TABLE 2. Mortality per cent in plants of Proteaceae and Dilleniaceae (pooled data) 460 days after inoculation of three jarrah forest stands with uninfested forest soil, with infested forest soil, and with pure culture of *Phytophthora cinnamomi*

Inoculation treatment	Stand						Totals	
	A		B		C			
	No. plants	% Dead	No. plants	% Dead	No. plants	% Dead	No. plants	% Dead
Control (none)	220	0.9	200	1.5	201	0.5	621	1.0
Uninfested forest soil	175	1.1	256	1.2	321	0.3	752	0.8
Infested forest soil	202	19.3	196	8.7	214	9.8	612	12.6
P. cinnamomi culture	321	10.3	302	4.3	189	5.3	812	6.9

was prepared in a similar manner.

In late October 1965, one lot of the appropriate inoculum was placed at depths of 10-15 cm in each of 10 holes adjacent to host plants and, if possible, at equal intervals within the plot. The holes were then refilled, including those in the noninoculated control plots.

Data on environmental conditions after inoculation included continuous records of rainfall, and soil temperatures at a depth of 4 cm in similar forest nearby. For each of the 3 weeks after inoculation, these records show more than 250 mm of rain, and mean daily periods of 24, 21, and 18 hr, respectively, during which soil temperatures were within the range for sporangial production by *P. cinnamomi*, as reported by Chee & Newhook (3).

In August 1966, plants of the Proteaceae and Dilleniaceae in the understory and shrub layers of each plot were counted. First-year seedlings were excluded so that the high death rates which are normal during the first summer would not obscure treatment effects. A recount was made in February 1967 (Table 2), when all plots were also assessed for the presence of dieback symptoms in the understory and shrub layers.

Typical dieback symptoms followed inoculation with pure cultures, and with soil from diseased forest in all three stands. Symptoms developed in either the understory or the shrub layer in 10 of 13 plots inoculated with infested soil and 6 of 13 inoculated with *P. cinnamomi* culture. There were no symptoms in plots inoculated with noninfested soil and none in the control plots. By May 1968, dieback symptoms had spread almost 20 m downhill from an inoculated plot in stand A.

Disease symptoms in jarrah appeared much more slowly than in species of the understory and shrub layer. By February 1971, however, severe symptoms had appeared in most jarrah in stand B, and one pole-sized tree 15 m tall had died. No results of the effect of inoculation on jarrah are available from stands A and C after February 1967, since these were inadvertently destroyed by salvage logging and pole cutting.

In May 1966, two soil samples, each a composite of 10 subsamples, were taken from around host

species in each plot, with minimum disturbance to the stand. The soils were lupin-baited for *P. cinnamomi*. In February 1967, a further sample was obtained by destructive sampling in order to obtain a high proportion of root material in each subsample. *P. cinnamomi* was reisolated from six plots in which dieback symptoms appeared after inoculation with soil from diseased forest, and in five plots where symptoms followed pure culture inoculation. The fungus was also isolated from four plots in which symptoms had not appeared by February 1967. There were no recoveries from the control or healthy forest soil treatments.

DISCUSSION.—The evidence presented in this paper leaves little doubt that *P. cinnamomi* is the cause of both jarrah dieback and similar disease in other indigenous plant communities in southwestern Australia.

The disease syndrome is consistent with the effects of a recently introduced pathogen. However, it has also been proposed (E. Bjorkmann, *unpublished data*; B. H. Pratt et al., *unpublished data*) that *P. cinnamomi* is probably an ubiquitous native in southern Australia where it is normally tolerated by its hosts but which causes damage if activated by disturbance of the plant communities. This supposes that the communities and the pathogen are normally in delicate balance with one another, and with their environments, a premise which is not consistent with the history and characteristics of the vegetation. Churchill (4) presented palynological and radiocarbon evidence that the present dominants in southwestern forests have dominated the pollen spectra for at least the past 7,000 years. He showed that wildfires were present throughout that time. Gardner (9) discussed the remarkable adaptations of the flora to fire. In historical times, even the most severe wildfires in intensively logged jarrah forest had little effect on species composition (17). The characteristics of the flora are those of stable communities. Although there is highly developed speciation, a high degree of endemism at species level (1, 8), and great diversity of floral morphology and color on the one hand, there is, on the other, marked uniformity of vegetative characteristics among widely separated taxa (8). These are characters of a highly evolved flora which, in relative isolation, has developed great taxonomic diversity and evolved growth forms which are adapted to the environment in which it has survived for a long time. It is therefore difficult to envisage how the several families which are highly susceptible to *P. cinnamomi* could have attained such an important position in the flora had the fungus been present throughout that time.

The pattern of disease development is also inconsistent with the hypothesis that *P. cinnamomi* is ubiquitous, but stimulated to activity only by disturbance. Severe dieback is found on sites favorable to jarrah where there has been little disturbance, yet it has failed to develop on many marginal sites which have been disturbed repeatedly by logging and burning. The hypothesis would also need to explain how the effects of a disturbance might be delayed for as long as 50 years. Though extensive utilization (and thus disturbance) of the jarrah forests commenced about 1850 and was at its maximum between 1870 and 1920 (17), jarrah dieback was restricted to a few small areas until as late as 1928. Furthermore, the disease has appeared only recently in some 40- to 50-year-old stands of dense and vigorous second growth which have remained undisturbed since the heavy logging which preceded regeneration.

Another hypothesis, which seems not to have been previously suggested, is that *P. cinnamomi* is native but limited in its natural occurrence, and has only recently begun to spread into the affected communities. If this were so, it might be expected that the plant communities in which *P. cinnamomi* occurred naturally would be generally resistant to the fungus. In fact, there is no plant community in the jarrah forest which is not somewhere severely affected. The many species of the genus *Banksia*, which occurs across the entire range of environmental conditions and is represented in every plant community, are all highly susceptible.

Both the physical and historical patterns of development of jarrah dieback are explained more readily in terms of the introduction of an unspecialized pathogen to previously stable plant communities which never before have been exposed to its selective pressure.

Although *P. cinnamomi* is known to have a wide host range and to cause serious disease elsewhere, its extensive effects in Western Australia, including the complete collapse of floristically complex communities, are unusual. Until recently, there had been no other report of coincident death of a wide range of species in natural communities. However, Podger & Ashton (14) reported a similar disease in dry sclerophyll woodland in the Brisbane Ranges, southeastern Australia. There are, in both the jarrah forest and the Brisbane Ranges, environmental conditions which are unusually favorable for the activity of the pathogen and which tend to increase the vulnerability of host plants.

In all of the affected communities, woody perennials dominate; there is a paucity of herbaceous shrubs, and grasses are of minor significance. The root systems of many of the susceptible species are extensively invaded. Thus, there is an unusually abundant food base for the development of inoculum in root systems which are in intimate and almost unbroken contact throughout large areas of the susceptible communities.

The soils have extremely low rates of microbial activity, as indicated by the very slow breakdown of litter (10). This may be due partly to the low base status of the soils and partly to climate (10). Activity of organisms with effects antagonistic to *P. cinnamomi* may, therefore, be unusually low.

The light-textured surface soils of the jarrah forest and of the Brisbane Ranges allow rapid wetting of the rooting zone, and thus aid dispersal of zoospores. At field capacity, the soils have low moisture contents due to their high content of nonabsorbing gravels and

their low clay and colloid content. Even light falls of rain may bring a large volume of soil to field capacity, and thus expose many roots to the chance of infection by zoospores. Although massive laterite or dense clay at shallow depth impedes drainage in much of the area, there are deep, well-drained soils on which the fungus has caused severe damage. It is often suggested that long periods of soil saturation are necessary for infection. However, it is readily demonstrated by the lupin-baiting method that infection can occur in less than 24 hr after the wetting of a previously dry soil.

Soil temperatures which favor rapid growth and sporulation of *P. cinnamomi* are infrequent during the winter period of maximum rainfall. Nevertheless, the soils are frequently wetted during spring and autumn and by occasional summer storms. The length and frequency of periods during which soil moisture and temperature are limiting for zoospore production and dispersal are less likely to be critical for survival of the pathogen where *P. cinnamomi* invades and grows in large organs, than in the feeder-root necrosis diseases of *Pinus* and other species. It is possible that, as long as the host lives, *P. cinnamomi* might continue to grow within the large roots of some hosts such as *Banksia* independently of soil moisture conditions.

The seasonal pattern of plant growth in relation to climatic stress is also an important factor in determining the severity of effects of *P. cinnamomi* root rot. Summer in these areas is usually hot and dry, and is marked by declining soil moisture reserves which may fall to wilting point early in the season, at least in the surface horizons. Summer is also the period of maximum shoot extension and flowering in *E. marginata* (11), and in many other species. Specht & Rayson (16) observed that shoot extension is "out of phase" with present climates in much of the sclerophyll flora of southern Australia. Thus, trees damaged by root rot in autumn and spring enter a period of maximum growth and environmental stress with root systems unfit to meet their needs.

Jarrah dieback is already a serious problem. In affected areas, its destructive effects include nearly all those listed as possible by Davidson & Buchanan (5).

Apart from causing direct losses, the disease leaves forest managers with the alternatives of managing poorly stocked and slow-growing stands of less desirable species or of species substitution on infertile soils in a difficult climate. The extent of the threat which *P. cinnamomi* presents to the native communities depends upon the proportion of unaffected forest which is susceptible, and on the rate at which the fungus is dispersed and spreads. It would seem to be capable of establishing and causing disease wherever it is introduced in southwestern Australia. Dieback already occurs in situations which cover almost the entire range of variation in site quality, stand structure, soil type, topography, and fire and cutting history (13). The only exceptions are stands on the red-brown loams of major river valleys. However, such areas have been less intensively logged, and thus have probably not been exposed frequently to the chance of infection. In any case, the area of such soils is small and is being planted to *Pinus*.

Although there is evidence of variation in the resistance of *E. marginata*, it seems unlikely that there will be sufficient resistance to prevent serious damage wherever *P. cinnamomi* establishes. Present knowledge of the factors affecting the disease does not permit prediction of the rates of spread or intensification of existing infections.

P. cinnamomi has appreciable competitive saprophytic ability, a capacity for long saprophytic survival in relatively small substrates (18), and a rapid rate of multiplication. When these facts are considered along with the already widespread distribution of the fungus, the large number of species affected, the great length of the existing interface between diseased and healthy forest, and the difficulties of trenching in indurated laterite, there seems to be little prospect for eradicating the fungus or restricting the infestation within its current boundaries. Therefore, it is imperative that the rate of transfer of inoculum to unaffected forest is reduced by modification of management procedures. Plant communities in which the effects of *P. cinnamomi* may not be as marked as in the jarrah forest should not be overlooked as possible sources of inoculum. Many of the necessary control procedures have already been implemented by the Forests Department of Western Australia.

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