CHAPTER 6

The Ecological Implications of Disease in the Southern Forest of South-Western Australia

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Abstract

The ecology of diseases occurring in the forests of south-western Australia are described under five broad types (root cankers, wood rots, stem and branch cankers, leaf spots and diseases of unknown etiology). *Phytophthora cinnamomi* and *P. citricola* are the main pathogens causing root cankers. Recent research has meant that relatively more is known about the distribution and impact of *Armillaria luteobubalina* in forests of south-western Australia than other wood rotting fungi. Even though the incidence of eucalypt dieback-decline has increased in south-western Australia since the 1970s, the contribution of canker fungi to stem and branch death has largely been ignored. There has been no systematic investigation of leaf spot fungi on forest trees in south-western Australia. Brown wood of karri and dieback-decline of marri and wandoo are diseases of unknown etiology. The diseases in southern forests form a complex of interactions between host, pathogen, environment and human activity. However, information is lacking on the specific requirements for pathogen survival, sporulation and spread as well as host infection and susceptibility in the environments experienced in southern forests. Implications for management are discussed.

CURRENT KNOWLEDGE

Introduction

Studies on forest diseases have mainly emphasized the effects of disease on the establishment, growth and productivity of economically important tree species. Often ignored and little studied has been the role plant diseases play in the functioning of forest ecosystems (Edmonds and Sollins 1974). Plant diseases are more often studied from an autecological (the behaviour of individual species or populations in relation to their environment) rather than a synecological or community dynamics perspective.

The pathogens reported causing diseases of the main *Eucalyptus* species occurring in forest south of the Preston River in south-western Australia are listed in Table 1. Lack of information precludes compilation of a similar list for understorey species, although the effect of disease on the understorey will be described where appropriate later. The reported pathogenic organisms in Table 1 are all fungi. This is consistent with the experience in other areas that most diseases of forest trees are caused by fungal pathogens (Schmidt 1978). Most of the discussion will relate to disease caused by fungi; disease caused by abiotic factors will be briefly covered in the section on diseases of unknown etiology.

Fungi are important in the cycling of carbon, water and nutrients within forest ecosystems, although their role in these cycles has been little quantified (Edmonds and Sollins 1974). Fungi lack chlorophyll and are primary consumers (heterotrophs) of plants as parasites or decomposers within the characteristic trophic structure of a forest ecosystem. Some fungi take on different trophic roles during different stages of their life cycle.

Plant pathogens, like other primary consumers, utilize energy from plants by destroying seeds, buds, leaves, stems or roots. In this manner plant pathogens may selectively affect competitive vigour or kill individual plants. However, the potential deleterious affects of pathogens on the vigour of native flora is often ignored. The influence of plant pathogens on intra- and inter-specific competition, the distribution of plant species, the genetic structure of populations and the diversity of individual plant communities is poorly understood (Burdon 1987).

Table 1

Diseases of the *Eucalyptus* species occurring in the Woodchip Licence Area in southwestern Australia. X indicates occurrence of disease, susceptibility obtained from inoculation or observation indicated in parenthesis and reference number in superscript.

Disease/Pathogen	Marri	Karri	Red Flowering Gum	Tingle Yellow	Tingle Red	Jarrah	Wandoo
SOUTH-WESTERN AUSTRALIA							
ROOT CANKERS							
Phytophthora cinnamomi	X(R)[o,y]	(R)[l,y]		(R)[o]	(R)[o]	X(MS)[o,y]	(R)[y]
Phytophthora citricola	(MS)[r]					(MS)[k,r]	
Phytophthora drechsleri						X[s]	
Phytophthora megasperma var. *sojae*						(R-MS)[k,r]	
Phytophthora nicotianae						X[c]	
Phytophthora nicotianae var. *parasitica*						(MS)[k,r]	
WOOD ROTS							
Armillaria luteobubalina (White rot)	X(MS)[h,n,p]	X(MS-MR)[w,n]				X(MS-MR)[h,p]	X(S)[p]
Phellinus gilvus	X[g,x]					X[g,x]	
Phellinus rimosus (White pocket heart rot)							X[g,x]
Piptoporus australiensis (Brown cubical heart rot)		X[g,x]		X[g,x]			
Piptoporus portentosus (Brown rot)	X[g,x]					X[g,x]	
Polyporus pelliculosus (Brown powdery rot)					X[g,x]		
Polyporus tumulosus (Heart rot)		X?[g,x]				X[g,x]	
Poria mutans (Yellow straw rot)						X[g,x]	
BRANCH AND STEM CANKERS							
Botryosphaeria ribis	X[c](R)[q]	X[c]				X[c](MS)[q]	X[c]
Endothia gyrosa	X[c]					X[c]	X[c]
Ramularia pitereka (*Sporotrichum destructor*?)	X(S)[t,z]		X(S)[t,z]				
LEAF SPOTS							
Davisionella eucalypti						X[w]	
Fairmaniella sp.							X[s]
Leptomelanconium australiense			X[v]				
Phoma sp.						X[s]	
UNKNOWN ETIOLOGY							
Brown wood		X					
Crown decline	X						X

Table 1 (*cont'd*)

Disease/Pathogen	Marri	Karri	Red Flowering Gum	Tingle Yellow	Tingle Red	Jarrah	Wandoo
OUTSIDE SOUTH-WESTERN AUSTRALIA							
WOOD ROTS							
Armillaria luteobubalina			X[u]				
Phellinus gilvus		X[i]					
STEM CANKERS							
Cytospora australiae			X[b]				
Endothia gyrosa		X[d]					
Pestalotia disseminata		X[i]					
LEAF SPOTS							
Cercospora eucalypti			X[b]				
Mycosphaerella sp.		X[i]					
Mycotherium amygdalinum			X[m]				
NURSERIES							
DAMPING OFF							
Phytophthora cinnamomi			X[c]				
Phytophthora cryptogea					X[f]		
Phytophthora sp. (unnamed)					X[e]		
STEM CANKER							
Botrytis cinerea		X[s]					
LEAF SPOTS							
Hainesia lythri		X[j]					
Sphaerotheca alchemillae (Powdery Mildew)				X(VS)[a]			

Marri (*Eucalyptus calophylla*)
Karri (*Eucalyptus diversicolor*)
Red Flowering gum (*Eucalyptus ficifolia*)
Tingle
Yellow (*Eucalyptus guilfoylei*)
Red (*Eucalyptus jacksonii*)
Jarrah (*E. marginata*)
Wandoo (*E. wandoo*)

S = Susceptible, R = Resistant, M = Moderate, V = Very.

References: [a]Boesewinkel (1981); [b]Browne and Laurie (1968); [c]Davison and Tay (1983); [d]D'oliveira (1931); [e]Forsberg (1985); [f]Hardy and Sivasithamparam (1988); [g]Hilton (1982); [h]Kile *et al.* (1983); [i]Lundquist and Baxter (1985); [j]Lundquist and Foreman (1986); [k]Newhook and Podger (1972); [l]Palzer and Rockel (1973); [m]Park and Keane (1982); [n]Pearce *et al.* (1986); [o]Podger (1968); [p]Shearer and Tippett (1988); [q]Shearer *et al.* (1987b); [r]Shearer *et al.* (1988); [s]Shivas (1989); [t]Smith (1970); [u]1Smith and Kile (1981); [v]Sutton (1974); [w]Swart (1988); [x]Tamblyn (1937); [y]Tippett *et al.* (1985); [z]Walker and Bertus (1971)

More is known of environmental factors affecting disease and the ecology of pathogens occurring in south-western Australia, than on the implications of disease in an ecosystem context. For this reason the diseases occurring in the forests of south-western Australia are described under five broad types (Table 1). Table 2 summarizes the life cycles and Table 3 possible implications of representative pathogens for the four disease types of known etiology. As an aid to the assessment of potential disease problems, Table 1 also includes those pathogens reported when the *Eucalyptus* species were grown outside of south-western Australia and in nurseries.

DISEASES OF JARRAH, KARRI, MARRI AND WANDOO FORESTS OF SOUTH-WESTERN AUSTRALIA

Root Cankers

Phytophthora species are the main pathogens infecting secondary phloem of roots of *Eucalyptus* in south-western Australia (Table 1). Although mainly root pathogens, *Phytophthora* species can also invade the collar and lower stem. The *Phytophthora* species in Table 1 are introduced soil borne opportunists whose life cycles are characteristically dependent on moisture for sporulation, survival and dispersal (Dell and Malajczuk 1989; Shearer and Tippett 1989). The soils and topography in conjunction with the hydrological cycle and a susceptible plant community of south-western Australia have provided niches whereby *Phytophthora* species can survive dry conditions, despite the harsh dry summers experienced in the region. The interactions that have created the diversity of microenvironments and conditions favourable for sporulation, survival and dispersal of *Phytophthora* species in south-western Australia are detailed in Shearer and Tippett (1989) and can only briefly be described here.

Phytophthora species are probably *r* strategists in the *r-K* continuum of MacArthur and Wilson (1967) as they can quickly reproduce and complete their life cycles (Table 2) when conditions are favourable. The sporangium-zoospore cycle can rapidly produce and release large numbers of zoospores (Table 2) into the soil when conditions are warm and moist. Favourable conditions for sporulation in the mediterranean climate experienced in south-western Australia, are most likely to occur in autumn and spring. Low temperatures in winter and low moisture levels in summer inhibit sporulation. Conditions for survival are not as restrictive as those for sporulation and the fungi survive wherever the soil is moist or infected host tissue is present. Sexual production of oospores is an important form of reproduction of homothallic species such as *P. citricola* and *P. megasperma* var. *sojae* because the thick-walled spores are more resistant to desiccation than zoospores.

Favourable environments for sporulation and survival vary between areas and from year to year. The coincidence of warm moist conditions depends on the commencement and finishing of frontal winter rains, the frequency of summer rain, the occurrence of water-gaining areas from near-surface seepage above impeding horizons within the soil profile, the amount of soil cover, and of disturbance. The distribution of susceptible host material is also an important factor.

Infected moist soil moved by human activity is the main way the *Phytophthora* species are dispersed over large distances. Natural dispersal occurs by water flowing in surface and near-surface drainage systems and by growth through root systems (Table 2).

Phytophthora cinnamomi and *P. citricola* are the most frequently recovered and widely distributed *Phytophthora* species in the southern forest region (Table 4). The two *Phytophthora* species have been recovered mainly from jarrah (*E. marginata*) forest, but also from karri (*E. diversicolor*) forest (Schuster 1978; Stukely and Hill[1], personal communication; unpublished reports by CALM's dieback mapping group). Often symptom expression is subtle, resulting in underestimation of the occurrence of the *Phytophthora* species in southern forests. Disease may only be evident as sparsely scattered deaths of susceptible hosts. Large areas are classified as uninterpretable (Table 4) because of either the lack of visible susceptible indicator species on aerial photographs or resistant species have colonized and dominate infected areas in which the susceptible vegetation has been killed. Checks on the ground may improve the precision of interpretation by identifying infection in the shrub layer not evident on aerial photographs. An area infected with *P. cinnamomi* was mapped through a karri stand in this manner (Cell 13 Report by CALM's dieback mapping group). Unrecognized infections in uninterpretable areas have the potential of providing sources of inoculum for inadvertent spread.

Infections associated with *P. citricola* have been noted as part of mapping for *P. cinnamomi*, but the areas infected by *P. citricola* have not been determined. Impact of *P. citricola* is confined to isolated deaths in the shrub layer. However, little is known of the susceptibility of native flora to *P. citricola* infection.

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Table 2

Summary of the ecology of selected fungal pathogens occurring in forests of south-western Australia

Factor	Disease/Pathogen				
	Root cankers (*Phytophthora cinnamomi*)	Wood rots (*Armillaria luteobubalina*)	Wood rots (*Piptoporus australiensis*)	Stem cankers (*Botryosphaeria ribis*)	Leaf spots
Taxonomic class[a]	Oomycetes	Basidiomycetes	Basidiomycetes	Ascomycetes Deuteromycetes	Ascomycetes Deuteromycetes
Introduced	yes	no	no	yes	?
Reproduction	zoospores	basidiospores	basidiospores	conidia	conidia
Primary disease cycle	< yr	< 1- many yr	< 1- many yr	< 1 yr	< 1 yr
Infectious period	< wk-month	1-many months	1-many months	< wk-month	< wk-month
r/*K* strategy	*r*	intermediate	*K*	*r*	*r*
Dispersal	water, soil, root	air, root	air	air, rain-splash	air, rain-splash
Infection	direct/wound	wound/direct	wound	wound/direct	wound/direct
Predisposing factors	moisture defoliation(?) nutrition	drought defoliation nutrition	nutrition(?)	drought defoliation nutrition	moisture nutrition

[a] Alexopoulos (1962)
? = insufficient information

The ecology of *P. citricola* in forests of south-western Australia is poorly understood. *Phytophthora citricola* has a lower temperature optimum for growth in jarrah roots than *P. cinnamomi* (Shearer *et al.* 1987a). Low temperature in winter and autumn may inhibit growth of *P. citricola* less than that of *P. cinnamomi*. Inoculation studies have shown that jarrah is equally susceptible to invasion by *P. citricola* and *P. cinnamomi* (Shearer *et al.* 1988). In contrast, marri (*E. callophylla*) is more susceptible and *B. grandis* more resistant to invasion by *P. citricola* than by *P. cinnamomi*. Lesions of *P. citricola* in *B. grandis* are likely to be confined. Thus infection of the secondary tissue of *B. grandis* is unlikely to be as favourable for survival and dispersal of *P. citricola* as it is for *P. cinnamomi* (Shearer *et al.* 1988). Spread of *P. citricola* must be prevented because the fungus has been frequently recovered from soil in landings and roadside drains, the fungus produces oospores resistant to adverse conditions and the potential host range of the pathogen is poorly understood.

Phytophthora cinnamomi is widely distributed throughout the southern forest (Table 4). Infections are mainly associated with drainage lines or road making and logging activity prior to 1970 (CALM's dieback Mapping group Cell Reports).

Impact of *P. cinnamomi* is mainly confined to death of the shrub layer. Many of the species of the families Proteaceae, Epacridaceae, Dilleniaceae and Myrtaceae make up a large component of the jarrah forest understorey and are susceptible to infection by *P. cinnamomi*. Despite this, the changes in understorey composition caused by *P. cinnamomi* infection are poorly documented for forests of south-western Australia (Shearer and Tippett 1989). In particular, more information is needed on changes in plant species richness following infection and the associated affects on fauna.

Jarrah is the only *Eucalyptus* species in the overstorey susceptible to *P. cinnamomi* (Table 1) and death of jarrah is often confined to isolated clumps on lateritic or poorly drained soils. Most of the jarrah deaths are described as old in the CALM's dieback mapping group Cell Reports, but the time of death is not known. A report by Podger in 1966 (report on a field examination of southern dieback areas, unpublished report of the CSIRO Forest Research Institute, Kelmscott) dates one period when jarrah died in infected southern forest areas.

Table 3

Summary of the implications of infection of selected fungal pathogens occurring in forests of south-western Australia

Factor	Disease/Pathogen				
	Root cankers (*Phytophthora cinnamomi*)	Wood rots (*Armillaria luteobubalina*)	Wood rots (*Piptoporus australiensis*)	Stem cankers (*Botryosphaeria ribis*)	Leaf spots
Host range	very wide	very wide	narrow	wide	narrow?
Impact on:					
Understorey	death	death	none	?	?
Regeneration	death	death	none	?	?
Overstorey	decline & death	decline & death	decline & death	decline & death	decline
Effect on diversity	reduction	reduction	none	?	?
Part of natural succession	not in past, but now an influence	yes	yes	probably[a]	probably
Occurrence of damage in host	all stages	all stages	thinning,harvest rotation, utilization	all stages	all stages
Affects growth of *Eucalyptus* host	reduce(?)	reduce(?)	none(?)	reduce(?)	none(?)
Canopy reduction	yes	yes	no	yes	some
Influences energy & nutrient cycling	yes	yes	yes	yes	yes
Hazard rating	yes	no	no	no	no
Rating of risk of infection	possible	no	no	no	no
Control options:					
Minimize disturbance	yes	yes	yes	yes	yes
Current hygiene practices	applicable	applicable	n/a	n/a	n/a
Reduction by fire of:	susceptible hosts	inoculum	inoculum	inoculum	inoculum
Reduction by antagonists of:	inoculum	substrate & inoculum	substrate & inoculum	?	?
Enhance host resistance	?	reduce stress	reduce stress	reduce stress	reduce stress
Fertilization	?	?	?	?	?
Chemical	spot infections	n/a	n/a	n/a	n/a
Resistance	in rehabilitation	in rehabilitation	?	?	?

? = insufficient information
n/a = not applicable
[a] endemic fungi would

Research conducted in the northern jarrah forest provides most of the information on the ecology of *P. cinnamomi* in south-western Australia (Shearer and Tippett 1989). Less research has been done on *P. cinnamomi* in southern forests; this is documented in four publications (Palzer and Rockel 1973; Christensen 1975; Schuster 1978; Strelein 1988) and a report (Grant and Blankendaal 1988). Because of differences in climate, landform, soils, vegetation and the intensity of human activity between the regions, results from research in northern forest cannot be extrapolated south of the Preston River (Shearer and Tippett 1989).

Christensen (1975) monitored moisture and temperature in the top 75 mm of soil and found that removal of canopy, scrub or litter cover from the soil increased the coincidence of warm moist conditions

Table 4

Incidence of *Phytophthora cinnamomi*, *Phytophthora citricola* and *Armillaria luteobubalina* in southern jarrah forest compiled from maps and reports of the Dieback Mapping Group, Inventory and Planning

Block	Area (ha)	Year Inter-preted	*Phytophthora cinnamomi*			*Phytophthora citricola*	*Armillaria luteobubalina*
			Area inter-pretable (%)	Interpreted area infected (%)	Infections (No.)	Infections (No.)	Infections (No.)
Carey	2195	1982	52	37	80	0	0
Coonan-Warrup	8100	1982	-	-	20	0	25
Iffley-Strickland	2535	1982	81	53	62	2	1
Easter-Andrew-Barlee	9352	1982/83	54	31	152	12	38
Mattaband-Burnside	5520	1983	62	44	15	2	2
Spring-Poorginup	3910	1984	35	72	22	0	6
Carter-Thornton-Yornup-Alco	9750	1984/85	-	-	56	15	16
Crowea	80	1986	-	-	9	0	0
Dordagup	308	1986	66	8	55	0	2
Mack	296	1986	-	-	3	0	0
Wye-Deep-Sharpe	15250	-	-	-	118	0	6

- no data

favourable for disease development. A small increase in temperature towards the end of winter increased the duration of the period when conditions were favourable for development of *P. cinnamomi* in spring. However, the results of Christensen's (1975) study underestimate the likely effects of environment on development of *P. cinnamomi* in the southern region and differences between regions. Temperature and moisture were monitored at only four sites over 18 months during a period of below-average rainfall. Furthermore, the existence of an environment favourable to sporulation, survival and dispersal of *P. cinnamomi* at depth below the soil surface was not appreciated at the time and the study did not include the potential effects of summer rain on disease development. No determination has been made of the population dynamics of *P. cinnamomi* in the range of environments and soils of southern forest. Information is lacking on the specific requirements for pathogen survival, sporulation and spread as well as host infection and susceptibility in southern forest soils. This information is needed to predict the development of *P. cinnamomi* in the range of environments experienced in southern forest and the effects of disturbance.

Southern forest sites that are likely to be favourable for increase and spread of *P. cinnamomi* and host infection can be identified from the work of Schuster (1978), Strelein (1988) and Grant and Blankendaal (1988). Schuster (1978) observed greatest impact of *P. cinnamomi* in the overstorey in sites with shallow duplex and gravelly-sand lateritic soil. In the development of a site-vegetation classification system for the southern jarrah forest, Strelein (1988) found greatest incidence of *P. cinnamomi* in less fertile, high rainfall, poorly drained sites associated with southern jarrah site-vegetation types P, R and S.

Grant and Blankendaal (1988) have used Strelein's site-vegetation types and landform to develop a hazard rating system for *P. cinnamomi* in southern jarrah forest. High hazard was associated with southern types P and S and landform units Dwellingup, Trent, Ellis, Mungardup and Caldyanup. These upland types or landform units are associated with lateritic or duplex soils that impede the drainage of water. Low hazard was associated with types T, S and K and the Crowea and Bevan landform units in dissected valleys with fertile loamy soils. Both high and low hazard were associated with the following landform units: Hester on ridge crests with gravelly-sand over duricrust; the units Collis, Mattaband and Keystone with yellow duplex soils; and the stream unit in minor valleys with swampy floors. The hazard rating system has only recently been introduced and mapping commenced. Mapping of hazard must have high priority to accurately determine the proportion of southern forest at risk from *P. cinnamomi* infection.

It is generally considered that impact of *P. cinnamomi* is less in southern jarrah forest than in northern jarrah forest (Strelein 1988). Reasons for this difference are based mainly on empirical observation. It cannot be implied from the difference between the two regions that the implications of *P. cinnamomi* infection is less in southern than northern forest. Interactions between historical and environmental

factors have effected differences in disease incidence and impact between the two regions.

Historically, southern jarrah forest has been exposed to less disturbance that favours spread and intensification of *P. cinnamomi* than northern forest. Most of the northern forest has been logged at least two or three times (Abbott and Loneragan 1986) while southern forest has mainly been logged only once. Southern forest has not been exposed to the widespread mineral exploration and the road construction and earth movement associated with bauxite mining carried out in northern forest.

Strelein (1988) and Christensen (1975) suggest that disturbance from more intensive management may lead to intensification of disease owing to *P. cinnamomi* in southern forest. Accurate predictions are difficult, however, without more information on the influence of the microclimates experienced in southern forest on the development of *P. cinnamomi*. Furthermore, the use of integrated control can minimize the effects of disturbance on *P. cinnamomi* intensification and spread. Hygiene procedures already in practice (Underwood and Murch 1984) need to be integrated with control methods that reduce the rate of disease development. However, integrated control measures require further development before practical application (Shearer and Tippett 1989).

Localized death of jarrah has occurred in infected areas of southern forest, but little is known of the timing of these deaths and the climatic conditions when they occurred. The hazard rating system of Grant and Blankendaal (1988) allows the mapping of high hazard sites, but cannot predict how quickly disease will be expressed following infection. Prediction of the rate of disease expression in sites of different hazard ratings needs a better understanding, than is presently available, of the occurrence of conditions favourable for *P. cinnamomi* sporulation and survival and host infection in southern forest sites, especially at depth in the soil profile.

Host studies have shown that site factors affect the susceptibility of jarrah to invasion by *P. cinnamomi* (Tippett *et al.* 1987). The susceptibility of jarrah on sites of different hazard ratings has yet to be assessed. Insufficient is known of the physiological status of host plants on different sites under different climatic and management conditions to determine when and for how long some species are vulnerable to infection and invasion by *P. cinnamomi* (Shearer and Tippett 1989). On intermediate hazard sites where jarrah is likely to survive, the effect of *P. cinnamomi* infection on regeneration, growth and leaf area is poorly understood (Table 3).

Wood Rots

Wood rotting fungi have cellulolytic and lignolitic enzymes and are important contributors to nutrient cycling within an ecosystem through the decomposition of plant material including standing timber. Table 1 lists only decay organisms that attack the sapwood or heartwood of standing trees. The volume of timber lost through decay has not been determined for forests of south-western Australia, despite the growing appreciation of the need for better utilization and conservation of the present timber resource.

Little research has been undertaken on the ecology of wood rots caused by basidiomycetes (Table 1) that are native to south-western Australia (Table 2). Tamblyn (1937) determined the identity and incidence of a number of decay fungi in south-western forests (Table 1) and the taxonomy was updated by Hilton (1982, 1988). A general description of wood rotting fungi attacking standing jarrah is given by Hilton *et al.* (1989).

Although specific details are lacking, the life cycles of the wood rotting fungi occurring in forests of south-western Australia (Table 2) are comparable to similar organisms in other forests. Wood rotting fungi can be considered as intermediate or *K* strategists in the *r-K* continuum (MacArther and Wilson 1967) as their life cycles are mainly dependent on the ability to maintain stable population carrying capacity (*K*) from annual reproduction (Table 2). This is in contrast to the *r* strategists, such as the causal fungi of root and stem cankers, that have fluctuating population levels and rapid reproduction (Table 2) in response to transient favourable conditions in a fluctuating environment.

Infection from wood decay fungi occurs mainly from aerial dispersed basidiospores or through mycelium in root systems. Basidiospores, formed by sexual recombination of gametes, are shed in winter from annual or perennial fruiting bodies found on decayed roots and stems of dead and living trees. Fruiting bodies of *A. luteobubalina* are mainly produced in June and July (Pearce *et al.* 1986; Shearer and Tippett 1988), but the environmental stimuli favouring fruiting at this time of the year are poorly understood. Dead trees following wildfire, or karri stumps left after logging may provide a long-term food base for sporulation of *Phelinus* species that cause serious heartwood decay of standing trees (Pearce and Malajczuk 1990a).

Infection points for entry of germinating basidiospores are mainly provided by wounds caused by fire, logging injuries, dead or broken limbs or insect damage. The effect of environment on the survival and infectivity of basidiospores of most decay

fungi is poorly understood (Merrill 1970) and has not been determined in south-western Australian forests. The distribution of infection points and aerial dispersed basidiospores results in a discontinuous, discrete distribution of infections of different genotypes. The number and distribution of different genotypes for a particular decay organism can provide an estimate of the frequency of infection from basidiospores, but no analysis of this type has been done in forests of south-western Australia.

Recent research on *Armillaria luteobubalina* in the karri and jarrah forest (Kile *et al.* 1983; Pearce *et al.* 1986; Shearer and Tippett 1988) has meant that relatively more is known about the distribution and impact of *A. luteobubalina* in forests of south-western Australia than the other wood rotting fungi. *Armillaria luteobubalina* is a primary pathogen widely distributed throughout south-western Australia (Pearce *et al.* 1986; Shearer and Tippett 1988; Table 4). The location, but not the size of infection centres have been determined during routine mapping (Table 4) and the area of forest affected by *A. luteobubalina* is not known. The pathogen spreads within infection centres by mycelial growth through roots. New infections are established by contact between roots and stems, and dead roots and stumps increase the inoculum level. Disease caused by *A. luteobubalina* in forests in Victoria spreads at a rate of 0.5-2.0 m/year (Kile 1981), but no estimates are available for south-western Australia.

Armillaria luteobubalina infects a wide range of plant species from diverse families (Pearce *et al.* 1986; Shearer and Tippett 1988), but changes in species richness within affected areas have not been determined. Many hosts that resist infection by *P. cinnamomi*, are susceptible to *A. luteobubalina* (Shearer and Tippett 1988).

Impact of *A. luteobubalina* in the jarrah forest varies between plant community and climatic zone (Shearer and Tippett 1988). Patch death of *E. wandoo*, *B. grandis* and *Xanthorrhoea preisii*, and crown decline and scattered death of marri and jarrah is associated with *A. luteobubalina* in intermediate-low rainfall zones receiving < 1000 mm p.a. Damage to trees in the high rainfall zone (> 1100 mm p.a.) of the jarrah forest is not as severe as in eastern low rainfall zone forest, despite the fact that the fungus is well established and fruits prolifically. Within high rainfall-zone infection centres, marri, jarrah and *B. grandis* exhibit crown decline but there are few and scattered mortalities. Scattered deaths of overstorey species partly reflects variation in the response to infection within a species; some individuals resist infection while others of the same species die when infection reaches the base of the stem. The reasons for this variation is not known and the contribution of stress, inoculum potential, site, soil and climatic factors or the genotype of host or pathogen to variation in response to infection has yet to be determined (Shearer and Tippett 1988).

Impact of *A. luteobubalina* in the karri forest changes with age of the forest (Pearce *et al.* 1986). The pathogen kills vigorous karri saplings in the youngest stands and a significant loss of regeneration trees can occur in infected areas. Loss in older infected stands (5-15 years old) is mainly confined to death of subdominant and suppressed trees and *A. luteobubalina* would play a role in the thinning of natural stands. The deaths occur mainly within a 10-m radius of infected karri and marri stumps.

The impact of *A. luteobubalina* in karri stands probably reflects changes in the balance between inoculum potential of the pathogen, host resistance as a function of vigour and tree size, and environmental stress during the life of the stand (Pearce *et al.* 1986). *Armillaria luteobubalina* invades the root system of stumps following logging (Kile 1981; Shearer and Tippett 1988) and the inoculum potential of the fungus is probably greatest in young generation stands. Infected stump roots represent a large source of inoculum compared with the relatively small diameter of young karri sapling roots and the inoculum potential of the fungus probably overrides host resistance factors (Pearce *et al.* 1986). Dominant trees in older stands are probably sufficiently vigorous to contain lesions of *A. luteobubalina* and prevent girdling of the root crown and death. However, infection of dominant trees, although contained, weakens roots and may predispose the trees to windfall (Pearce *et al.* 1986).

Logging prescriptions could increase the incidence of disease caused by *A. luteobubalina* in the forest by increasing the amount of inoculum present (Pearce *et al.* 1986; Kellas *et al.* 1987). The frequency of cutting mixed eucalypt forests of Victoria had a greater effect on disease development by *A. luteobubalina* than cutting intensity *per se*. Regular creation of stumps through frequent cutting increases both the inoculum level and the probability of remaining trees being in close proximity to inoculum (Kellas *et al.* 1987). However, the implications of cutting frequency also depend on the distribution of the fungus and the effect of environmental factors on disease expression.

As native organisms it is likely that the decay fungi have always caused some disease as part of the functioning of the ecosystems in which they occur (Table 3). For example, high intensity fire kills karri trees and stimulates regeneration, providing similar opportunities for *A. luteobubalina* as in cut-over

forests. Nevertheless care must be taken that forest operations do not shift the balance in favour of the fungi by providing infection sites through wounding and a large food base for reproduction. Information on the distribution, incidence and ecology of fungi causing decay is needed to predict the effects of forest operations on decay organisms under different climatic and site conditions.

Stem and Branch Cankers

Even though the incidence of eucalypt dieback-decline has increased in south-western Australia since the 1970s (Kimber 1980), the contribution of canker fungi to stem and branch death has largely been ignored. Mortality and decline of marri and red flowering gum (*E. ficifolia*) was associated with stem cankers in the mid 1930s (Smith 1970). Davison and Tay (1983) identified a number of pathogenic fungi associated with stem and branch cankers of forest trees in south-western Australia. Canker fungi were associated with a complex of factors causing crown decline of wandoo (*E. wandoo*) (Albone 1989). Severe cankering of marri in southern forest is causing concern (C. Muller[2] *personal communication*), but has not been investigated.

Table 1 lists the primary pathogens that cause perennial cankers. Not included are fungi that are frequently isolated from cankers but are nonagressive facultative parasite (e.g. *Cytospora eucalypticola*). *Botryosphaeria ribis* is an aggressive pathogen widely distributed in tropical and temperate regions and able to infect at least 34 genera and 20 families of plants (Smith 1934). Whereas *B. ribis* is possibly an introduced pathogen (Davison and Tay 1983), *Ramularia pitereka* is probably native to south-western Australia (Walker and Bertus 1971). Although Davison and Tay (1983) consider *Endothia gyrosa* to be native to the region, Walker (1987) questions the native status of this pathogen.

How the canker causing fungi complete their life cycles in south-western forest is poorly understood. This is complicated by uncertainties in the identity of canker fungi. The identity of the *Endothia gyrosa* in south-western forests was recently confirmed by isozyme analysis against voucher specimens (E. Davison[3], *personal communication*). The fungus causing the canker on red flowering gum was called *Sporotrichum destructor*, but the name was never validly published and no description of the fungus exists in the literature. The fungus causing cankering of red flowering gum and marri may be *R. pitereka*, but the identity remains uncertain until fresh isolates can be examined (Walker and Bertus 1971).

Canker fungi are opportunistic *r* strategists (Table 2). They sporulate in dead bark and are dispersed as sexually produced ascospores in wind currents or asexually produced pycnidiospores in rain splash. Entry of germinating spores is probably gained through lenticels or wounds from branch stubs, broken branches and insect damage. Phloem and sapwood invasion results in sunken cracked areas on the stem that may expose the xylem and exude kino. Girdling by cankers can result in gradual decline from death of twigs and lateral branches to rapid death of leaders. No determination has been made of the losses of leaf area and tree function from stem and branch death in forests of south-western Australia. Progressive canker development may lead to death of diseased trees (Smith 1970; Shearer *et al.* 1987b).

Disease caused by canker fungi can be aggravated by transient stress factors (Schoeneweiss 1975). Trees planted outside the normal geographic range may experience environmental stress with an associated decline in resistance to infection by canker organisms (Shearer *et al.* 1987b). Stress from defoliation by insects (Abbott, this volume) can predispose trees to canker organisms (Schoeneweiss 1975).

Leaf Spots

There has been no systematic investigation of leaf spot fungi on forest trees in south-western Australia similar to studies in eastern Australia (e.g. Park and Keane 1982). Table 1 list leaf spot fungi collected and identified during the investigation of other pathogenic organisms. More species, than in Table 1, would be described from a systematic survey of leaf spots on eucalypts in south-western Australia.

In general leaf spots are opportunistic *r* strategists (Table 2). They sporulate in lesions and are dispersed as sexually produced ascospores in wind currents or asexually produced conidia in air and pycnidiospores in rain splash. Many leaf spot fungi are weak facultative pathogens and are associated with insect damage to leaves.

Most of the leaf spot fungi of eucalypts would be native to south-western Australia and play an important role in litter decomposition and nutrient cycling. Defoliation of eucalypt seedlings by host-specific leaf pathogens affect the composition of regeneration in forests of eastern Australia (Burdon and Chilvers 1974). Severe defoliation of eucalypts by leaf spots has not been reported in Western Australia. Karri seedlings are very susceptible to leaf infection by

2 C. Muller, CALM, Bunbury

3 E. Davison, CALM, Como

powdery mildews in the nursery (Table 1). The effect of powdery mildew infection on karri seedling susceptibility and establishment in the field is not known.

Unknown Etiology

Brown wood of karri and dieback-decline of marri and wandoo are diseases of unknown etiology. Browning of wood occurs frequently in regrowth karri sawlogs and also occurs in logs from regrowth forests. It causes concern owing to reduced recovery and increased milling costs. The cause of brown rot and its effect on timber durability and permeability are currently being investigated.

Some areas of marri and wandoo in forests and rural areas have suffered from decline and some death over the last decade (Kimber 1980; Tippett and Shea 1985). However, investigation of the causes of dieback-decline has received little attention. Damage from frost, drought, salinity, disturbance, pests and pathogens are suggested causes, but the etiology is still uncertain. The succession of factors, such as climatic, edaphic or biotic stress that predispose trees to infection by pathogens, often complicates the determination of the cause. Deforestation through dieback-decline has obvious implications for wildlife habitats, conservation of roadside verges, amenity values such as the provision of shade and the control of salinity and erosion by reaforestation.

CONCLUSIONS

Systematic surveys are required to improve information on the identity and incidence of pathogens causing disease in forests of south-western Australia. Such surveys would identify previously unrecognized pathogens, as illustrated by the survey on cankers by Davison and Tay (1983). Ranking of the relative importance of disease would then be possible according to deleterious affects on conservation and production values.

As part of the functioning of ecosystems, pathogens continually modify the plant populations in which they occur. The effects of disease on plant populations are thus more often a reflection of persistent long-term changes than of devastating damage. Disease causing devastating damage is the exception, being more the symptom of imbalance mainly brought about by human intervention. The initiation and rate of increase and spread of disease is considered to be mitigated by the functional diversity of forest ecosystems (Schmidt 1978; Dinoor and Eshed 1984). Functional diversity comprises the characteristic structural, macro and microenvironmental and genetic diversity of a forest ecosystem that act collectively to impede disease development in space and time (Schmidt 1978). The ability of a system to cope with disease is also related to the length of time host and pathogen have coevolved. Coevolution may have a greater stabilizing role than diversity, in mitigating the effects of disease in some instances (May 1976).

Functional diversity and coevolution of host and pathogen cannot buffer ecosystems against introduced pathogens with a wide host range. At least three of the pathogens listed in Table 1 have been introduced into south-western Australia, namely *B. ribis* (Davison and Tay 1983), *E. gyrosa* (Walker 1987) and *P. cinnamomi* (Newhook and Podger 1972). *Phytophthora* species other than *P. cinnamomi* may also have been introduced into south-western Australia (Shearer and Tippett 1989). These pathogens are widely distributed in temperate and sub-tropical regions of the world and infect a wide range of plant species from diverse families. The impact of *P. cinnamomi* infection on the jarrah forest is an exceptional example of an introduced pathogen with a wide host range causing great damage to a diverse but mainly susceptible plant community (Shearer and Tippett 1989).

Predictions of the implication of disease requires an understanding of the varied life cycles of the pathogens causing disease in southern forest (Table 2). Many of the pathogens listed in Table 1 are natives of south-western Australia and play a part in the functioning of forest ecosystems (Table 3). However, the life cycles of *Phytophthora* species other than *P. cinnamomi*, and the fungi causing wood decay, stem and branch cankers, and leaf spots have not been investigated and are poorly understood in south-western Australia. The cause of a number of diseases has still to be determined (Table 1).

The diseases in southern forests form a complex of interactions between host, pathogen, environment and human activity. More information is needed on the specific requirements for pathogen survival, sporulation and spread as well as host infection and susceptibility in the environments experienced in southern forests. The effects of potential changes in climate on disease requires a much better understanding than is available at present of the life cycles of pathogens in southern forest. The effects of insect pests on life cycles of pathogens needs to be determined. Insect attack increases the incidence of wounds and stress from defoliation and predisposes trees to infection.

Human actions affect different parts of the life cycle of a pathogen to either aggravate or control disease in forest ecosystems. Human activity introduced *Phytophthora* species and *B. ribis* into

diverse but susceptible plant communities of forests of south-western Australia. Movement of infected soils by human activity is an important mechanism of dispersal of *Phytophthora* species and has necessitated the commitment of considerable resources into hygiene measures to prevent and minimize this type of spread. Fungi causing wood decay, cankers and leaf spots are mainly dispersed by natural means (Table 2), but care must be taken to ensure that human activity does not cause wounds favourable to infection (Table 2). Cutting frequency and regeneration from coppice stumps can exacerbate the incidence of wood decay. Fire may cause wounds favourable for infection by decay fungi, but destroy inoculum of canker and leaf spot fungi. Stand management must aim to prevent stress and physiological imbalance that may predispose a tree species to infection.

The life cycles of different pathogens can be linked by the stimuli and interactions occurring within forest ecosystems. Care must therefore be taken to ensure that the management of a forest does not consider a few pathogens to the exclusion of others that may occur.

CURRENT RESEARCH

Identification of the organisms causing brown wood in karri is the only current research on disease being undertaken in southern forest. *Armillaria luteobubalina* is an intermediate-*K* strategist and probably sensitive to control by reducing the food base for reproduction. The use of decay fungi to reduce stumps as a food base for *A. luteobubalina* in southern forest has been investigated (Pearce and Malajczuk 1990b).

Research on diseases in the forest is mainly being carried out north of the Preston River or in woodlands and shrublands of the coastal plain. Research is continuing on the factors affecting the development and control of *P. cinnamomi* in the northern jarrah forest (Shearer and Tippett 1989) and coastal plain (Shearer and Hill 1989). Current research on *A. luteobubalina* has determined the impact of the pathogen in the wandoo forest and coastal shrublands. Methods for pathogenicity tests are being developed. A survey of dieback-decline of wandoo has been completed but the data requires analysis (Albone 1989).

There is no research in progress on the cause of dieback-decline diseases and cankering of marri. No determination is being made of the environmental factors affecting the life cycles of *Phytophthora* species, *A. luteobubalina* and other decay organisms, stem cankers and dieback-decline of unknown etiology in southern forest.

HIGH PRIORITY ADDITIONAL RESEARCH REQUIREMENTS

Shearer and Tippett (1989) detail research priority areas and questions for *P. cinnamomi* and other *Phytophthora* species. Research on diseases in southern forests needing immediate attention are listed in order of priority:

- Determine the cause of cankering and dieback decline of marri and options for control.
- Develop a data base of the susceptibility of plant species to *Phytophthora* species and determine the long-term effects of infection on community diversity.
- Determine the relationships between climatic events and the processes affecting the development of *P. cinnamomi* and *P. citricola* and host infection in soil profiles of southern forest. Use the information to apply risk and hazard rating systems.
- Identify and rank diseases in southern forest from an assessment of their impact and potential threat to forest health.

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