A Manual of Diseases of Eucalypts in South-East Asia

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Mycological drawings were prepared by Dr Zi Qing Yuan. The photographs of diseased trees and fungal pathogens were taken by the authors or by their close colleagues, with the exceptions of Fig. 76 which was kindly provided by Ms K. Pongpanich, Figs 92 and 93 by Dr S.S. Lee and Fig. 107 by Professor A.C. Alfenas.

Dedication

With the kind agreement of our coauthor, Zi Qing Yuan, Ken Old and Mike Wingfield would like to dedicate this book to the late Professor Dave French, Department of Plant Pathology, University of Minnesota. For both of us he was an inspirational teacher, mentor and valued friend.
Preface

Eucalypts are second only to pines as the major forest plantation species grown internationally and are of prime importance in the southern hemisphere, much of South-East Asia, southern China and the Indian subcontinent. With a few exceptions, eucalypts are unique to Australia. They have evolved under selection pressure from their major pests and pathogens and from the harsh constraints of the Australian environment. Their capacity for rapid growth, even on difficult sites, their growth habit, ease of vegetative propagation and their desirable product qualities have led to widespread establishment of large eucalypt plantations in many countries of South-East Asia.

Eucalypts, in their native environments are hosts to a very wide range of fungal pathogens, especially those attacking leaves, shoots and stems. The generally broad genetic base of individual species and their presence in heterogeneous forest communities, however, provides significant protection against disease epidemics. In contrast, industrial eucalypt plantations in South-East Asia are typically single species or hybrid plantings, often from a few clones which may share common parentage. Modern propagation techniques, such as shoot multiplication or tissue culture, make it possible to plant large areas with identical clones, with the expectation of uniformly rapid growth and high product quality. Such practices are very dangerous from the standpoint of disease, as pathogens, including endemic fungi and those newly introduced into a plantation region, may cause widespread epidemics. This risk is heightened by movement of ‘improved’ germ-plasm between eucalypt growing regions and even internationally, as pathogens may be transmitted by infested seed or infected planting stock.

Avoidance of major epidemics of eucalypt diseases in South-East Asia requires an increased awareness of the risks from pathogens, inherent in plantation forestry and a systematic approach to disease management. A good knowledge is needed of those diseases, present in plantations, which offer significant threats and of those, which although currently absent, could cause future problems. Based on such knowledge, tree species, provenances and clones can be assessed for their susceptibility to major pathogens and strategies can be devised for the systematic deployment of different clones of widely varying parentage throughout plantation regions. Clonal forestry then has the potential to become a powerful means for the control of plantation diseases, as practised in Brazil and South Africa for eucalypt cankers, rather than increasing the risk of epidemics.

This manual of diseases of eucalypts in South-East Asia will be of assistance to those charged with maintaining the health of eucalypt plantations, in identifying the most common diseases present in their region. It provides recommendations for disease management and offers an introduction to relevant world literature. The manual is a companion to the earlier CIFOR-published "A Manual of Diseases of Tropical Acacias in Australia, South-East Asia and India".

David Kaimowitz
Director General, CIFOR
Introduction

Eucalypt plantations in South-East Asia

*Eucalyptus* species are second to pines in global importance as plantation trees. In the tropics and subtropics they are the most widely planted genus. Data published by FAO (1995) indicated that there were at least 1.4 million ha of formal *Eucalyptus* plantations in the South-East Asian region (Table 1). Midgley and Pinyopusarerk (1996), in reporting these statistics, indicated that the data did not include the equivalent of about 2.0 million ha growing as boundaries around fields and scattered trees. Since 1995 a number of countries, notably China, Laos, Thailand and Vietnam, have accelerated planting programs. It is likely that eucalypt plantations in the South-East Asia region now exceed 2.0 million ha.

Table 1. Estimated areas of *Eucalyptus* plantations in the South-East Asian region in 1995 (FAO)

<table>
<thead>
<tr>
<th>Country</th>
<th>Area (ha)</th>
</tr>
</thead>
<tbody>
<tr>
<td>China</td>
<td>670 000</td>
</tr>
<tr>
<td>Indonesia</td>
<td>80 000</td>
</tr>
<tr>
<td>Laos</td>
<td>62 000</td>
</tr>
<tr>
<td>Malaysia</td>
<td>8 000</td>
</tr>
<tr>
<td>Myanmar</td>
<td>40 000</td>
</tr>
<tr>
<td>Philippines</td>
<td>10 000</td>
</tr>
<tr>
<td>Thailand</td>
<td>195 000</td>
</tr>
<tr>
<td>Vietnam</td>
<td>350 000</td>
</tr>
<tr>
<td>Total</td>
<td>1 415 000</td>
</tr>
</tbody>
</table>

In addition to large-scale plantations and farm plantings grown to supply fibre for industrial plants, eucalypts are highly valued in rural communities for a wide range of uses. These include fuel, poles, small lumber and furniture, essential oils and tannins (Midgley and Pinyopusarerk 1996). The main species grown include *Eucalyptus camaldulensis*, *E. tereticornis*, *E. urophylla* and *E. grandis* in subtropical and tropical regions, and *E. globulus* in more temperate climes. There is a strong trend for plantations to be established from clones of trees, often inter-specific hybrids, selected for good growth and product quality. Great benefits can be gained from such clonal forestry but this practice requires particular vigilance with regard to the susceptibility of individual clones to pests and diseases. A thorough knowledge of the nature and biology of these agents is thus required. In the equatorial humid tropics, acacias have become more widely planted than eucalypts, due partly to the high levels of leaf and shoot diseases sustained by *Eucalyptus* plantations, which depress growth rates and affect product quality. These diseases are the focus of this manual, which is complementary to a manual of diseases of tropical acacias published earlier (Old *et al.* 2000).
**Eucalyptus** diseases and the scope of this manual

Tree diseases can be grouped according to the stage of growth of the plant, that is, seedlings in nurseries or trees after out-planting, and by the part of the tree affected. Although tree disease problems often originate in nurseries, management solutions are commonly available. Disease outbreaks in plantations are less readily contained, diagnosis is more difficult and management options are few. For these reasons the primary purpose of the manual is to provide a field guide for identification of eucalypt diseases by plantation managers and their staff. Diseases are dealt with in turn, based on the part of the tree affected, namely foliage, branches, stems and roots, internal defect and decay development in non-living tissue, and a section is devoted to nursery diseases. A description is also given of a rust pathogen, not found as yet in South-East Asia, which could cause serious disease if accidentally introduced into the region.

Until recently there was no published work that provided a comprehensive account of eucalypt diseases and pathogens. The need for such a reference document has been met by Keane et al. (2000), who have provided an account of eucalypt diseases worldwide. In contrast, this manual is a guide to the more damaging diseases of *Eucalyptus* plantations that have been encountered by the authors and their colleagues during disease surveys, consultancies and research and development projects in South-East Asia (primarily East Timor, Indonesia, Laos, Thailand and Vietnam) over the past decade. In view of the very large numbers of foliar and stem diseases recorded on *Eucalyptus*, both in this region and worldwide (Sankaran et al. 1995), and budgetary constraints, the guide could not be comprehensive. The forest health specialist who critically examines diseased *Eucalyptus* specimens will find many genera and species of fungal pathogens not covered here. An attempt has been made, however, to include the most serious eucalypt pathogens found in the region and also some diseases that may not cause significant damage, but have conspicuous symptoms.

Each disease is attributed to one or more pathogens and is described using standard headings. Accounts are based on first-hand experience of these diseases in South-East Asia, supplemented by information from other regions and selected literature references. Original colour photographs of symptoms, and in many cases, sporing structures of pathogens are presented. Line drawings of fungal characteristics used in identification to genus or species level have been prepared from fresh or herbarium specimens for users of the manual who have access to a microscope and basic plant pathology skills (Johnston and Booth 1983). A key is provided to assist in diagnosis, and a glossary of terms that may be unfamiliar to non-specialists.

References


### Key to diseases and pathogens described in this manual

#### Foliar diseases

<table>
<thead>
<tr>
<th>A</th>
<th>Mycelial growth superficial</th>
<th>Page number</th>
</tr>
</thead>
<tbody>
<tr>
<td>A1</td>
<td>Hyphae white, powdery appearance</td>
<td>Oidium</td>
</tr>
<tr>
<td>A2</td>
<td>Hyphae black, perithecia may be present</td>
<td>Meliola</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>B</th>
<th>Causing necrotic spots, spores borne freely on lesion surfaces</th>
<th>Page number</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td>Cylindrical, septate spores</td>
<td>Cylindrocladium</td>
</tr>
<tr>
<td>B2</td>
<td>Slender, tapered, septate spores</td>
<td>Pseudocercospora</td>
</tr>
<tr>
<td>B3</td>
<td>Spheroidal, aseptate, yellow spores or brown, septate stalked spores</td>
<td>Puccinia</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>C</th>
<th>Causing necrotic spots, spores borne within cup-, flask-shaped or spheroidal fruiting bodies embedded in leaf lesions and extruded when moist</th>
<th>Page number</th>
</tr>
</thead>
<tbody>
<tr>
<td>C1</td>
<td>Cup-shaped fruiting bodies containing spheroidal spores</td>
<td>Cryptosporiopsis</td>
</tr>
<tr>
<td>C2</td>
<td>Spheroidal, sub-stomatal fruiting bodies containing two-celled ascospores</td>
<td>Mycosphaerella</td>
</tr>
<tr>
<td>C3</td>
<td>Flask-shaped fruiting bodies extruding hair-like columns of slender, septate spores</td>
<td>Phaeophleospora</td>
</tr>
<tr>
<td>C4</td>
<td>Flask-shaped fruiting bodies containing darkly pigmented spores</td>
<td>Microsphaeropsis</td>
</tr>
<tr>
<td>C4.1</td>
<td>Spores spheroidal, thick walled with truncated collar</td>
<td>Coniella</td>
</tr>
<tr>
<td>C4.2</td>
<td>Spores lemon shaped</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>D</th>
<th>Causing corky spots, spores borne within elongate to branched fruiting bodies scattered on the surface of lesions</th>
<th>Page number</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Aulographina</td>
<td></td>
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</tbody>
</table>

#### Stem cankers

| A  | White to pink mycelium superficial on bark | Erythricium | 55 |

<table>
<thead>
<tr>
<th>B</th>
<th>Mycelium not superficial, fruiting bodies embedded in bark or on the bark surface</th>
<th>Page number</th>
</tr>
</thead>
<tbody>
<tr>
<td>B1</td>
<td>Flask-shaped fruiting bodies containing ascospores and/or conidia</td>
<td>Cryphonectria eucalypti</td>
</tr>
<tr>
<td>B1.1</td>
<td>Sexual and asexual fruiting bodies embedded in yellow or orange stromata</td>
<td>Cryphonectria eucalypti</td>
</tr>
<tr>
<td>B1.2</td>
<td>Sexual fruiting bodies with long necks, bases embedded in stroma. Asexual fruiting bodies on bark surface</td>
<td>Cryphonectria cubensis</td>
</tr>
<tr>
<td>B1.3</td>
<td>Fruiting bodies embedded in black stromata</td>
<td>Valsa</td>
</tr>
</tbody>
</table>
B2 Fruiting bodies not flask-shaped, ascospores and conidia formed in chambers within black stromata

Botryosphaeria 60

B3 Minute, sub-epidermal fruiting bodies containing ellipsoidal, brown conidia

Coniothyrium 50

**Bacterial wilt**

**Rots of woody stems and roots**

A Range of basidiomycete species including;

Armillaria
Ganoderma
Phellinus 71

**Nursery diseases**

A Pre-emergence and post-emergence damping-off

Phytophthora
Pythium 79

B Web blight

Rhizoctonia 84

C Powdery mildew

Oidium 88
Black mildew

Disease
Black mildew

Causal organism
This disease is caused by fungi belonging to the genus *Meliola*, family Meliolaceae, order Meliolales (Ascomycota). The Meliolales are obligate parasites producing a variety of structures which penetrate into the host cells. *Meliola amphitricha* Fr., *M. densa* Cooke and *M. eucalypti* F. Stevens & Roldan ex Hansford have been recorded on leaves of *Eucalyptus* spp. The black mildews are often confused with the common sooty moulds which are superficial, epiphytic saprophytes and also with members of the genus *Meliolina*, family Meliolinaceae, order Dothideales.

Host range and distribution
Species of *Meliola* are primarily tropical. *Meliola amphitricha* was recorded on *Eucalyptus* sp. from Queensland and Victoria, Australia (Cooke 1892) and *M. densa* on *Eucalyptus* spp., *Callistemon viminalis* and *Tristania conferta* from Queensland (Simmonds 1966), and on *E. tereticornis* from Papua New Guinea (Shaw 1984). *Meliola densa* was also recorded on *Eugenia* sp. from India (Hosagoudar et al. 1994) and on a hybrid of *E. pellita* x *E. brassiana* in a provenance and family trial in Melville Island, Northern Territory of Australia (the authors, unpublished data). *Meliola eucalypti* was identified on *Eucalyptus* sp. from the Philippines (Hansford 1962).

Symptoms
Species of *Meliola* grow on the surfaces of leaves and stems and form thick, black, radiate, velvety colonies of up to 1 cm diameter (Fig.1). In cases of heavy infestation, the entire leaf surface may be covered by the fungus. The infection is usually more frequent on upper than lower leaf surfaces. Sometimes young stems and twigs can also be infected. Numerous minute spherical fruiting bodies develop on the fungal thallus on the leaf surface (Figs 1, 3). In *Meliola densa* on *E. pellita* x *E. brassiana* these ascocarps have pigmented walls bearing setae and contain sac-shaped asci with two pigmented ascospores (Figs 2, 3).

Pathology
No in-depth studies have been carried out with the black mildews of *Eucalyptus*. The ascospores of *Meliola* are generally believed to germinate on the surface of the host leaves, immediately producing capitate hyphopodia. Mycelium then grows on the surface of leaves, obtaining nourishment through haustoria that extend into the epidermal cells from the hyphopodia and spread to form a colony. The close association of *Meliola* with scale insects and mealy bugs on mango suggests that the honeydew excreted by these insects provides a rich food source for growth and establishment of black mildews (Lim and Khoo 1985). Water splash and crawling insects may be their main dispersal agents.
Impact

Little information is available on the impact of black mildews on growth of eucalypts. De Guzman (1977) indicated that heavily infected phyllodes of acacias turned yellow and abscissed prematurely, with repeated infection leading to stunting of seedlings. On older trees, however, black mildew does not cause any serious damage although it may be common on the foliage. A study on biochemical changes in the leaves of ebony trees affected with a black mildew (M. diospyri Yates) indicated a reduction, within infected leaves, of primary metabolites necessary for the production of soluble sugars and starch, total protein, amino acid and chlorophyll (Hosagoudar et al. 1997).

Control and management

Control of the disease is seldom necessary as it has little impact on the host. If infestation is heavy, however, black mildew can be controlled by spraying fungicides and insecticides to eliminate scale insects and mealybugs.

References

**Figure 1.** Black mildew on a leaf surface, denser bodies are fruiting bodies (perithecia)

**Figure 2.** *Meliola densa*: A. Seta, bar = 40 µm; B. pigmented hyphae; C. hyphopodia, bar = 12.5 µm; D. perithecium wall; E-F. septate pigmented ascospores, bar = 12.5 µm
Figure 3. *Meliola densa* on *Eucalyptus pellita*: A. perithecia surrounded by sterile hyphae (setae); B. longitudinal section of a perithegium (half); C. asci containing two mature ascospores; D. ascospores; E. hyphae with lateral cells: capitate hyphopodia (swollen) and a mucronate hyphopodium (horn-like); F. a seta. Bar = 80 µm for A; 30 µm for B; 45 µm for C; 25 µm for D and E; and 40 µm for F.
Cryptosporiopsis leaf and shoot blight

Disease
Cryptosporiopsis leaf and shoot blight

Causal organism
Cryptosporiopsis eucalypti Sankaran & B. Sutton

Host range and known distribution
Not known to occur on hosts other than Eucalyptus spp., C. eucalypti has been collected by the co-authors from Eucalyptus with leaf spot or shoot blight symptoms in Australia, Japan, Laos, Indonesia, Sri Lanka, Thailand and Vietnam, (Old and Yuan 1994, Old et al. 2002) South Africa and Uruguay (Wingfield unpublished). Other reports are from Brazil (Ferreira et al. 1998), Australia, India and Hawaii (Sankaran et al. 1995) and New Zealand (Gadgil and Dick 1999).

Symptoms
Symptoms of C. eucalypti infection develop on both leaves and shoots of eucalypts. Leaf spots occur on both sides of the leaves and vary in size, shape and colour, within and between Eucalyptus species. For example, there are at least four lesion types on E. camaldulensis. These include large, brown, spreading necrotic lesions leading to a leaf blight symptom; circular or sub-circular spots 1-2 cm in diameter; irregular chocolate-brown or greyish spots covering much of the leaf area (Fig. 5); irregular roughened or corky lesions with eruption and necrosis of epidermal tissue, sometimes localised along veins, on which the fungus fruits. Terminal shoots of young trees can be totally defoliated and are commonly blighted (Fig. 6).

Conidiomata develop on foliar lesions on blighted shoots and have also been found associated with cankers on small-diameter woody branches. Fruiting bodies are cup-shaped when moist with pigmented margins, bearing creamy masses of macroconidia (Fig. 7). The conidiomata are scattered irregularly on lesions and erupt through the epidermis or stem periderm but can be quite inconspicuous when leaves are dry. Macroconidia are thick walled and ellipsoid to elongate-ellipsoid in shape with distinctive protuberant attachment scars (Figs 8, 9).

Pathology
Cryptosporiopsis eucalypti can exist as a canker pathogen in woody stem tissue, so that inoculum persists during dry months when conditions are not favourable for leaf and shoot blight. During the onset of epidemic disease, leaf spots develop and affected leaves are eventually shed. The most damaging phase of the disease, however, is blight and dieback of terminal shoots. The typically conical shape of fast-growing plantation trees becomes flattened, main stems suffer dieback and multiple branching, apical dominance is reduced.
and growth can be stunted. Canker pathogens, such as *Cytospora* sp. and *Lasiodiplodia theobromae*, invade affected stems and trees may be killed.

**Impacts**

Impacts in plantations vary from scattered lesions, especially on lower crowns and coppice shoots, to severe defoliation and death of shoots in the crowns of susceptible trees. The limited information available from observations in Vietnam suggests that *E. camaldulensis* ssp. *obtusa* is relatively susceptible, *E. camaldulensis* ssp. *simulata* and *E. tereticornis* are moderately resistant whereas *E. pellita* appears to be highly resistant. Differences in the susceptibility to *C. eucalypti* leaf and shoot blight of *E. camaldulensis* occurred at the species, provenance and family levels (Old *et al.* 2002).

**Control and management**

As for other leaf and shoot blight diseases in eucalypt plantations, the only feasible management option is selection of disease-resistant trees (Fig. 4). Depending on management objectives, this can be a choice of species or provenances that, on the basis of field trials, appear relatively resistant to *C. eucalypti* leaf blight. Where species can be readily grown from cuttings, selection and propagation of selected, highly resistant individuals can be practised. Old *et al.* (2002) found that all six provenances studied and many families of *E. camaldulensis* contained highly resistant trees, many with good silvicultural characteristics. These selections could then either be established as clonal seed orchards or propagated as cuttings and used as clonal plantations in disease-prone areas. Selection of resistant trees requires some knowledge of leaf and shoot pathology as *C. eucalypti* leaf blight can be confused with other diseases, for example cylindrocladium leaf blight and foliar diseases caused by *Mycosphaerella* spp. and their anamorphs.

**References**


Figure 4. *Eucalyptus camaldulensis* clonal trial infected by *Cryptosporiopsis eucalypti* with resistant and susceptible trees

Figure 5. Leaf spot caused by *C. eucalypti*

Figure 6. *E. camaldulensis* seedling defoliated by *C. eucalypti*; healthy family in background

Figure 7. Masses of conidia oozing from pycnidia onto leaf surface

Figure 8. Macroconidia of *C. eucalypti*, bar = 32 μm
Figure 9. *Cryptosporiopsis eucalypti* on *Eucalyptus camaldulensis*: A. conidia; B. conidia attached to conidiogenous cells; C. conidiogenous cells; D. conidia; E. longitudinal section of conidioma. Bar = 20 µm for A-D; and 40 µm for E.
Cylindrocladium foliar spot and foliar blight

Disease
Cylindrocladium foliar blight

Causal organisms
Cylindrocladium spp. are widespread and damaging pathogens of a very wide range of plant hosts including eucalypts. Cylindrocladium spp. have sexual states (teleomorphs) in the genus Calonectria de Not. There have been two major reviews of Cylindrocladium in the last decade, (Crous and Wingfield 1994, Crous 2002). In the latter publication, Crous has distinguished 39 Cylindrocladium spp. Of these 24 are listed as pathogens of Eucalyptus spp. and 15 of these have been found in South-East Asia.

Host range
These fungi are most commonly found as the Cylindrocladium anamorph (asexual state) and those most commonly recorded often have very wide host ranges. For example C. reteaudii (as C. quinqueseptatum) has been recorded from many hosts in northern Australia, South-East Asia and India.

Known distribution
Distribution maps for all known species are provided in Crous (2002). Some species, e.g. C. reteaudii, occur primarily in tropical regions of South-East Asia, India and northern Australia. C. pauciramosum C.L. Schoch & Crous occurs in many countries around the world and may have been formerly confused with C. scoparium, which appears to be limited in its confirmed distribution to North and South America (Crous 2002). Other widely distributed species, known to attack eucalypts, and which occur in South-East Asia include C. insulare C.L. Schoch & Crous, C. parasiticum Crous, M.J. Wingf. & Alfenas, C. floridanum Sobers & E.P. Seym., C. theae (Petch) Subram. and C. pteridis F.A. Wolf.

Symptoms
The most common and severe causal agent of cylindrocladium leaf blight in South-East Asia is C. reteaudii (Bugn.) Boesew. (syn. C. quinqueseptatum Boedijn & Reitsma), which is responsible for epidemic disease in several countries including Australia (Fig. 10), India, Vietnam, Laos and parts of Thailand. The disease first shows as greyish water-soaked spots on young leaves (Figs 11, 13, 16). These spots coalesce and develop into extensive necrotic areas. Large numbers of shining white spores can be seen at the margin of lesions, on older necrotic portions of leaves, especially along midribs on the abaxial surfaces, and on fine shoots (Fig. 12). Under favourable conditions of high humidity and frequent rainfall, necrotic lesions cover the entire area of the leaf and fruit profusely on young shoot tips which are killed, resulting in leaf and shoot blight symptoms (Bolland et al. 1985).
Conidia of *Cylindrocladium* are typically cylindrical in shape with one or more cross walls (septa). Figures 14, 15 and 17 illustrate fruiting structures produced by *C. reteaudii*, including six-celled macroconidia, two-celled microconidia, vesicles at the tips of sterile hyphae and barrel-shaped phialides which give rise to the conidia. The fungus also forms pigmented chlamydospores, swollen hyphal cells that develop pigmentation and are resistant to biodegradation, thereby aiding survival in soil.

**Pathology**

*Cylindrocladium* spp. cause a variety of diseases in the nursery and in plantations including root and collar rot, shoot blight, leaf blight and foliar spots (Crous *et al.* 1991, Sharma and Mohanan 1982, 1991). Epidemic disease is favoured by high rainfall and humidity (Sharma and Mohanan 1991). Booth *et al.* (2000) used bioclimatic mapping to predict high hazard areas for *C. reteaudii* leaf blight for South-East Asia and other parts of the world. Good agreement was found between predicted high hazard areas and locations where epidemics occurred in South-East Asia. Annual rainfall of >1400 mm and minimum temperatures of the coldest month >16°C were useful predictors of high hazard. The spread of the disease is by means of conidia, (Figs 12, 14, 15, 17) which are borne in vast numbers on leaf surfaces. During heavy rain, these spores are splashed into the air and infect nearby trees. *Cylindrocladium* species are commonly able to survive in soil by means of thick-walled chlamydospores, and these propagules are probably responsible for initial infections within eucalypt stands. Infections usually appear on the foliage of lower branches and spread upwards into the crown. Although diseased foliage can be found in crowns of large trees, disease is most obvious in saplings and pole-sized trees, in which defoliation can be very extensive (Fig. 10).

**Impacts**

*Cylindrocladium* foliar blight is a major problem on eucalypts grown in the humid tropics. In high-rainfall regions of south-eastern and central Vietnam, repeated defoliation of susceptible provenances of *E. camaldulensis* has led to crown dieback with secondary infection by canker fungi, loss of form and even death. *Eucalyptus urophylla* has also suffered significant damage in central Vietnam. Overall, losses to cylindrocladium leaf blight have contributed to the generally poor growth rates of *E. camaldulensis* in disease-prone regions of Vietnam (often less than 10 m$^3$ ha$^{-1}$ y$^{-1}$ on a 5-6 year rotation).

**Control**

In nurseries, *Cylindrocladium* infection can be effectively controlled by carbendazim applied as a foliar spray or soil drench (Sharma *et al.* 1984, Ferreira 1994). In plantations, however, it is not economically feasible to control the disease by fungicidal treatment. The disease on eucalypts has been serious enough to warrant selection for resistant species, provenances, families and clones (Blum *et al.* 1992, Nghia and Old 1997, Sharma *et al.* 1999). *Eucalyptus pellita* has been found to be resistant to *C. reteaudii* leaf blight in northern Queensland and in Vietnam, where *E. brassiana* has also performed well.

**References**


Figure 10. *Eucalyptus grandis* x *E. urophylla* clone defoliated by *Cylindrocladium reteaudii* (syn. *C. quinquesepatum*)

Figure 11. *E. camaldulensis* showing leaf spot caused by *C. reteaudii* (upper leaf surface)

Figure 12. Conidiophores of *C. reteaudii* on shoot surface, bar = 250 µm

Figure 13. *E. camaldulensis* showing leaf spot caused by *C. reteaudii* (lower leaf surface)

Figure 14. Conidiophores with vesicle (swollen tip of projecting hypha), bar = 48 µm

Figure 15. Macroconidia with, typically, 3-5 septa, bar = 32 µm

Figure 16. *E. pellita* with leaf spot caused by *C. reteaudii*
Figure 17. *Cylindrocladium reteaudii* (syn. *C. quinqueseptatum*) on *Eucalyptus camaldulensis* from Vietnam: A. conidiophore terminating in a vesicle; B. typical branched conidiophores, and conidia; C. vesicles; D. phialides, cells which produce conidia; E. more densely branched conidiophores on agar culture; F. two-celled microconidia produced on agar culture; G. chlamydospores produced on agar culture. Bar = 30 µm for A-C and E-G; and 15 µm for D.
Mycosphaerella leaf diseases

Diseases
Leaf spot, leaf blotch or leaf crinkle, depending on symptomology

Causal organisms
Worldwide more than thirty distinct species of Mycosphaerella are recognised on Eucalyptus (Park et al. 2000), causing a great variety of symptoms. The genus Mycosphaerella as found on Eucalyptus has not been clearly defined and may actually represent several distinct genera. This possibility is supported by the association, with different Mycosphaerella species, of a wide range of anamorphic states. Anamorphs include Colletogloeum, Colletogloeopsis, Coniothyrium, Phaeoleospora, Pseudocercospora, Sonderhenia, Stagonospora, Stenella and Uwebraunia. Several of these anamorphs are discussed in the sections on Phaeoleospora and Pseudocercospora spp.

Significant changes in name combinations and association of named anamorphs with Mycosphaerella have often been often made. This provides considerable difficulty for the non-taxonomist in assessing which species are present in plantations and how their observations relate to published information. Names used in older literature might not accurately represent the fungus in question and these should be used with some circumspection. Descriptions of 10 Mycosphaerella spp. found on Eucalyptus, their synonyms, anamorphs and hosts are provided in Set 124, IMI Descriptions of Fungi and Bacteria (Crous et al. 1995), the most recent taxonomic treatment being provided by Crous (1998). Useful characteristics for identification are lesion morphology and their position on the leaf (Figs 18, 19, 21), fruiting body morphology (Figs 20, 23), ascospore and conidial size and shape and the mode of germination of ascospores (Figs 22, 23). Molecular methods are increasingly being used, to support more traditional approaches, in the identification of species of Mycosphaerella (Carnegie et al. 2001, Crous et al. 2001).

Collections of several Mycosphaerella species, associated with Eucalyptus, have been made in East Timor (Old unpublished), Indonesia (Crous and Alfenas 1995, Crous and Wingfield 1997), Thailand (Pongpanich unpublished) and Vietnam (Yuan unpublished). No systematic attempt has been made, however, to determine which are the most common and damaging Mycosphaerella species in South-East Asia. For this reason the main biological, morphological and pathological characteristics of the fungi will be summarised at the generic level, drawing largely on information derived from temperate species.

Host range
It is likely that all Eucalyptus spp. can be infected by one or more species of Mycosphaerella. For example, Crous et al. (1995) listed more than 50 hosts for M. cryptica (Cooke) Hansf. across all Eucalyptus subgenera. Juvenile, intermediate and adult foliage are susceptible to infection by this pathogen. In their check-list of fungi recorded on Eucalyptus, Sankaran et al. (1995) provided host lists for many Mycosphaerella spp. and their anamorphs. Host
records for fungi collected in Australia often include several to many *Eucalyptus* spp. whereas overseas records of *Mycosphaerella* on plantation eucalypts are sometimes restricted to a single species. This may reflect the limited range of *Eucalyptus* spp. present in exotic plantations, rather than host specificity and is probably also related to difficulties experienced in distinguishing between species.

**Distribution**

*Mycosphaerella* spp. have been recorded on *Eucalyptus* in all States and most forested regions of Australia although the knowledge of the species present and their impacts on tree health is incomplete (Park *et al.* 2000). These pathogens are also present in virtually all countries where significant exotic eucalypt plantations have been established, with most information being available from South Africa (Crous and Wingfield 1996), Brazil (Crous *et al.* 1993a,b) and New Zealand (Dick and Gadgil 1983, Dick and Dobbie 2001). *Mycosphaerella heimioides* Crous & M.J. Wingf. and *M. gracilis* Crous & Alfenas (Crous and Alfenas 1995, Crous and Wingfield 1996) have been collected in Indonesia along with *M. parkii* Crous, M.J. Wingf., F.A. Ferreira & Alfenas and *M. suberosa* Crous, F.A. Ferreira, Alfenas & M.J.Wingf. Fungi in this group have shown a remarkable capacity for international movement. For example, *M. marksii* Carnegie & Keane, a species first described in Australia (Carnegie and Keane 1994), was found in 1995 causing a leaf spot on *E. camaldulensis* in Vietnam (Yuan unpublished). *Phaeophleospora epicoccoides*, the anamorph of *M. suttoniae*, occurs in most countries where *Eucalyptus* spp. are grown (Crous *et al.* 1998).

**Pathology**

Detailed information on the pathology and epidemiology of *Mycosphaerella* diseases has been largely derived from research carried out in Australia (Carnegie and Keane 1994, Carnegie *et al.* 1997, Park and Keane 1982, Park and Keane 1987, Park 1988) on *M. cryptica* and *M. nubilosa*. These fungi are major pathogens of temperate eucalypts, although *M. cryptica* can be found on species with distribution well into the tropics, e.g. *E. grandis*.

Epidemics in plantations are favoured by abundant moisture, especially prolonged periods of rain, during which spores are released from pseudothecia and disperse to infect susceptible foliage. As many species also produce conidia on or within asexual fruiting structures, plantations are subjected to very high levels of inoculum and virtually all foliage can be exposed to infection. There is no single pattern of disease development, etiology varying with host-pathogen combination. For example, Park (1988) found that *M. cryptica* completed several generations during each epidemic, through formation of conidia and secondary ascospores. *Mycosphaerella nubilosa*, on the other hand, showed a delay in symptom development of 4–5 months between infection of newly-formed leaves to the onset of epidemic disease levels in expanded foliage.

Many variations in symptom development are associated with *Mycosphaerella* infections (Figs 18, 21), resulting in different combinations of lesion size, colour and morphology. Fruiting bodies can form on one, or both surfaces of leaves. Infected leaves develop spots and blotches, their severity depending on the pathogen species and the susceptibility of the host. In highly susceptible interactions, large lesions develop, often with crinkling of the leaves. Affected trees suffer premature defoliation and severe disease can cause stunting of trees.
Impacts

Impacts of *Mycosphaerella* spp. on tree health can be severe or negligible, depending on the fungal species, host susceptibility, host physiology (including juvenility) and climate. Significant damage to plantations, associated with *Mycosphaerella* only, appears to be unusual in South-East Asia. This contrasts with the very severe epidemics caused by *M. nubilosa* and *M. cryptica* in temperate eucalypt plantations in parts of Australia and New Zealand and by a fungus, identified as *M. juvenis* Crous & Wingf., in South Africa (Crous and Wingfield 1996). Where significant epidemics have occurred in South-East Asia associated with these fungi, the anamorphs have been most prominent and several of these are covered elsewhere in this manual, e.g. *Phaeophleospora* spp. *Mycosphaerella* stages are often inconspicuous or have not been found. An exception is *M. marksii*, which has been found in several locations in Vietnam associated with necrosis of leaf margins and defoliation of susceptible clones of *E. camaldulensis* (Fig. 18).

Control and management

Despite the common occurrence of these fungi and their anamorphs, serious epidemics of mycosphaerella leaf disease in South-East Asia have not been reported. In contrast, temperate *Eucalyptus* spp. suffer significant damage in many parts of the southern hemisphere, including Australasia, South America and South Africa. In these countries, variation in susceptibility to *Mycosphaerella* has been identified between provenances of *E. globulus* (Carnegie et al. 1994) and between inter-provenance and inter-specific hybrids of *E. globulus* and *E. nitens* (Dungey et al. 1997). In the event of future epidemics of mycosphaerella leaf diseases occurring in South-East Asia, it can be expected that selection for resistant planting material at the species, family and clonal levels will be effective counter measures.

References


Figure 18. *Eucalyptus camaldulensis*, leaf spots caused by *Mycosphaerella marksii* infection

Figure 19. Detail of lesion with pseudothecia (small black structures embedded in the leaf)

Figure 20. Detail of pseudothecium of *Mycosphaerella* sp. shown in Figure 21, note asci located within central region, bar = 15 µm

Figure 21. Juvenile leaf of *E. alba* with *Mycosphaerella* lesions

Figure 22. Germinating ascospores of *Mycosphaerella cryptica*, bar = 26 µm
Figure 23. *Mycosphaerella* spp.: A. diseased leaf of *Eucalyptus camaldulensis* infected by *M. marksii*, Vietnam; B, C. ascospores and ascus of *M. marksii*; D. ascospores of *M. cryptica*; E. germinated ascospores of *M. cryptica* (24 hr incubation); F. conidia of the *M. cryptica* anamorph *Colletogloeopsis nubilosum*; G. longitudinal section of a peritheciun of *M. cryptica*; H. ascospores of *M. nubilosa*; I. germinated ascospores of *M. nubilosa* (24 hr incubation). Bar = 25 mm for A; 15 µm for B, C, D, E, H and I; 20 µm for F; and 30 µm for G.
Phaeophleospora leaf diseases

Diseases
Phaeophleospora leaf diseases

Causal organisms

*Phaeophleospora* spp.
Four species have been recognised so far on *Eucalyptus*:
*P. epicoccoides* (Cooke & Massee) Crous, F.A. Ferreira & B. Sutton;

There have been many changes in the nomenclature of *P. epicoccoides* and *P. eucalypti*, which can lead to confusion in identification and reporting of outbreaks. Taxonomic changes and new combinations are cited in detail in Walker *et al.* (1992), in Crous *et al.* (1997), who resurrected the name *Phaeophleospora* for *Kirramyces* spp., and by Crous and Wingfield (1997), in which the teleomorph of *K. epicoccoides* (*Phaeophleospora epicoccoides*) is described as *Mycosphaerella suttoniae* Crous & M.J. Wingf. Synonyms are listed below:

*Phaeophleospora epicoccoides*:
*Kirramyces epicoccoides* (Cooke & Massee) J. Walker, B. Sutton & I. Pascoe
*Cercospora epicoccoides* Cooke & Massee apud Cooke
*Hendersonia grandispora* McAlp.
*Phaeoseptoria eucalypti* (Hansf.) J. Walker
*Phaeoseptoria luzonensis* T. Kobayashi

*Phaeophleospora eucalypti*:
*Kirramyces eucalypti* (Cooke & Massee) J. Walker, B. Sutton & I. Pascoe
*Cercospora eucalypti* Cooke & Massee apud Cooke
*Pseudocercospora eucalypti* (Cooke & Massee) Guo & Liu
*Septoria pulcherrima* Gadgil & Dick
*Stagonospora pulcherrima* (Gadgil & Dick) Swart

*Phaeophleospora destructans*:
*Phaeophleospora destructans* (M.J. Wingf. & Crous) Crous, F.A. Ferreira & B. Sutton
*Kirramyces destructans* M.J. Wingf. & Crous

Host ranges

*P. epicoccoides*: 26 *Eucalyptus* and *Corymbia* species were listed by Walker *et al.* (1992), and 35 by Sankaran *et al.* (1995). It is likely that many more *Eucalyptus* species will be hosts of this pathogen.
P. eucalypti: 13 species were listed by Walker et al. (1992), in the Eucalyptus subgenus Symphyomyrtus only. Sankaran et al. (1995) listed 60 hosts encompassing Symphyomyrtus, Monocalyptus and Corymbia.

P. lilianiae: found so far only on Corymbia eximia (Walker et al. 1992).

P. destructans: found so far on E. grandis (Wingfield et al. 1996), E. camaldulensis and E. urophylla (Old et al. 2003).

Distribution

P. epicoccoides: is widely distributed in virtually all parts of the world where eucalypts are grown, including Africa, South America, Australia, India, South-East Asia, Japan, Indonesia, Philippines and New Zealand.

P. eucalypti: records exist mainly from Australia and New Zealand. There are records from South America, South Africa, India, Taiwan and Italy but these may be mistaken identifications, possibly with P. epicoccoides or Pseudocercospora spp. This fungus does not appear to have been recorded in South-East Asia.

P. destructans: was originally described by Wingfield et al. (1996) from Sumatra. This pathogen caused severe defoliation in clonal plantations of E. camaldulensis in eastern Thailand in 1999 (Pongpanich unpublished) and was found on native E. urophylla in East Timor 2002 (Old unpublished). In 2002 the pathogen was recorded in several locations in northern, central and southern Vietnam (Old et al. 2003).

Pathology

These fungi are anamorphs of Mycosphaerella, although the teleomorph may be unusual or not yet recognised. They are distinguished by their formation of pigmented columns (cirri) or irregular aggregations of conidia, often on the abaxial surfaces of leaves, exuding from substomatal pycnidia (Figs 27, 32, 34). These may be associated with discrete chlorotic or necrotic lesions, e.g. P. eucalypti (Figs 24, 25), P. destructans (Figs 30, 31), or widespread on leaves with little evidence of discrete lesion development or cell damage apart from general discoloration. This often gives the leaf a reddish or burgundy hue, e.g. P. epicoccoides (Fig. 26). In the less pathogenic species damage is often restricted to lower crowns but P. destructans can cause complete defoliation of susceptible trees and may invade shoots. In Sumatra, P. destructans infected young growing shoots of susceptible E. grandis trees, resulting in severe dieback and loss of apical growth. Such trees may die when they are out-competed by less susceptible individuals. Stands of susceptible clones would probably not survive epidemics of P. destructans leaf blight.

The ability of this group of pathogens to spread is great, as very large amounts of inoculum are produced on leaves. Survival on fallen leaves and subsequent splash dispersal of spores is also likely in humid environments. Phaeophleospora spp. are common nursery pathogens and can be spread with infected planting stock. The widespread occurrence of P. epicoccoides suggests that this species may also be seed-borne, possibly through surface contamination.

The most common species of Phaeophleospora are distinguished by their conidial morphology. Spores of P. epicoccoides are cylindrical or slightly club-shaped with rough walls and variable numbers of septa—often more than four (Figs 29, 34). Spores of P. eucalypti are variable in
size usually having one septum per spore (Figs 28, 34), and are produced on both upper and lower surfaces of leaves. Conidia of *P. destructans* have three septa, are slender, curved or sinuate (Figs 33, 34).

**Impacts**

*Phaeophleospora epicoccoides* is common in the lower crowns of trees and can cause significant defoliation of seedlings in nurseries (Sharma and Mohanan 1981). It caused severe infection of younger leaves of some *E. grandis* clones in South Africa, and stressed trees seemed to be somewhat more susceptible to disease (Crous et al. 1989; Knipscheer et al. 1990). Similarly an *E. grandis* x *E. camaldulensis* clone grown in New South Wales, Australia was severely defoliated by *P. epicoccoides* (Carnegie, personal communication). *Phaeophleospora destructans*, as implied by its name (Wingfield et al. 1996), has caused severe damage to young *E. grandis* and hybrids with *E. urophylla* in Sumatra, to *E. camaldulensis* in Thailand and to *E. camaldulensis* and *E. urophylla* in Vietnam. *Phaeophleospora destructans* appears to be far more damaging than *P. epicoccoides*, which can often be found on the same trees. In East Timor, *P. destructans* appeared to cause little damage to native *E. urophylla* suggesting that this fungus may be indigenous to that region. *Phaeophleospora lilianiae* and *P. eucalypti* have not been recorded in the South-East Asian region and will not be further considered here.

**Control and management**

Until *P. destructans* emerged as a significant threat to eucalypt plantations in Indonesia, Thailand and Vietnam, these pathogens would not have been considered worthy of a management response. The very severe damage inflicted by *P. destructans* in Indonesia (recorded since 1996) and Thailand (since 2000) and in Vietnam during 2002, requires urgent action in terms of selection of resistant species, families or clones of preferred *Eucalyptus* species. A recent report (Old et al. 2003) indicates that the pathogen has spread throughout most of Vietnam. The pathogen has mainly been a plantation problem and the only viable management strategy is to select resistant germplasm. Substantial success has already been achieved in this regard in Sumatra where disease tolerant clones have been selected and deployed (Wingfield unpublished). Chemical control may be required in nurseries and clonal propagation facilities, but it is too early to recommend suitable control schedules.

**References**


Figure 24. Leaf of *Eucalyptus* sp., infected by *Phaeosphleospora eucalypti*, with chlorotic lesions

Figure 25. Older infection; lesions have become necrotic

Figure 26. Leaf of *E. camaldulensis* infected by *P. epicoccoides*; note burgundy hue of lesions

Figure 27. Pycnidium of *P. epicoccoides*, bar = 75 µm

Figure 28. Generally uniseptate conidia of *P. eucalypti*, bar = 25 µm

Figure 29. Multiseptate conidia of *P. epicoccoides*, bar = 25 µm
Figure 30. Juvenile leaf of *Eucalyptus camaldulensis*; note chlorosis and heavy sporulation of *Phaeophleospora destructans*

Figure 31. Older *P. destructans* infection of *E. urophylla*; note reddening of lesion margins with this *Eucalyptus* species, sporulation is mainly on underside of the leaf

Figure 32. Conidial masses of *P. destructans* on the underside of the leaf shown in Figure 31

Figure 33. Typically three-septate, sinuous conidia of *P. destructans*, bar = 25 µm
Figure 34. Phaeophleospora spp.: A. longitudinal section of *P. eucalypti* on *Eucalyptus nitens*; B. conidia of *P. epicoccoides* on *E. urophylla*; C. conidia of *P. eucalypti* on *E. nitens*; D. conidia of *P. destructans* on *E. camaldulensis*. Bar = 20 µm for B; 12 µm for C; and 18 µm for D.
Other eucalypt leaf spot diseases

Causal organisms
In addition to *Cylindrocladium* spp., *Cryptosporiopsis eucalypti*, and *Mycosphaerella* spp. and their anamorphs such as *Phaeophleospora* spp. (described elsewhere in this manual), many other foliar pathogens of *Eucalyptus* are present in plantations in South-East Asia. Most of these are described by Park *et al.* (2000) but there is generally little known regarding their pathogenicity or capacity to cause serious disease. In this section, several of these pathogens and their symptomology are briefly described, as they may be confused with more damaging organisms. Fungi included in this category are *Aulographina eucalypti* (Cooke & Massee) Arx & E. Mull., *Coniella* spp., *Microsphaeropsis* spp. and *Pseudocercospora eucalyptorum.* Crous, M.J. Wingf., Marasas & B. Sutton.

Host ranges and distribution
*Aulographina eucalypti* causes corky leaf spot and occurs only on *Eucalyptus* spp. (Figs 35, 36, 37). The fungus has been found in many temperate countries in which eucalypts are grown extensively (Sankaran *et al.* 1995) but there appear to be few records from the tropics. The authors have collected this fungus in Vietnam (Yuan unpublished) and Sri Lanka.

*Coniella* spp. appear to have wide host ranges including both tropical and temperate species (Figs 38–41, Fig. 48). *C. australiensis* Petr. and *C. fragariae* (Oudemans) B. Sutton have been found associated with leaf spots of *Eucalyptus* spp. in one or more of the following tropical countries: Australia, India, Indonesia, Sri Lanka and Vietnam.

*Microsphaeropsis* spp. (Figs 42–48) and *Pseudocercospora eucalyptorum* (Figs 49–52 are less well known fungi. Microsphaeropsis leaf spot has been recorded in Tasmania on *E. obliqua* as *M. callista* (H. Syd.) B. Sutton, (Yuan 1999). The same fungus has been recorded in Argentina and South Africa, suggesting a temperate range. Old (unpublished) found a *Microsphaeropsis*, closely resembling *M. globulosa* (Sousa da Camara) B. Sutton causing a severe leaf spot on *E. grandis* in Sri Lanka (Figs 42–45).

*Pseudocercospora eucalyptorum* has been found in many parts of the world (Crous *et al.* 1989, Sankaran *et al.* 1995). Records for the region include India, Sabah, Taiwan, Thailand and Vietnam. *Pseudocercospora* is one of the most common agents causing leaf spots of lower crowns of *E. camaldulensis* in the latter two countries (Old *et al.* 2002). Although the last authors retained *P. eucalyptorum* it seems likely that this pathogen may be an undescribed species (Yuan unpublished).

Symptoms
These fungi, which are most commonly found on the lower crowns of young trees, or on coppice shoots, cause leaf spots of various shapes and sizes as illustrated in Figs 35, 36, 38, 41, 42–45, 49 and 51. The size and shape of lesions and the numbers of fruiting bodies vary
with the host and environmental conditions. Correct diagnosis, therefore, may require the recognition of the spores through microscopical examination of infected leaves (Figs 37, 48, 52).

*Aulographina eucalypti* forms dark brown 'corky' leaf spots with elongate, or branched fruiting bodies called hysterothecia, opening by a longitudinal slit (Figs 35, 36), scattered on the surface of the lesions.

*Coniella fragariae* and *C. australiensis* cause medium to large reddish-brown lesions which often cause the leaf to partially curl as the tissue dessicates. Prominent black pycnidia are embedded in the lesions, sometimes concentrically arranged, and extrude vast numbers of dark brown to black spheroidal conidia onto the lesion surface (Figs 38, 41).

*Microsphaeropsis globulosa* on *E. grandis* forms large brown lesions with raised purple margins (Figs 44, 45); sporulation is sparse but the few pycnidia produced are easily seen with the naked eye (Fig. 43). These are embedded in the leaf surface and extrude small dark brown to black, thick-walled, ellipsoidal conidia (Figs 46, 47).

*Pseudocercospora eucalyptorum* forms profuse angular spots on infected leaves (Figs 49, 51). The central portions of these lesions bear dense tufts of conidiophores bearing needle-shaped, septate conidia (Fig. 50).

**Pathology and impacts**

These fungi vary greatly in their taxonomy, morphology and physiology. For example, *A. eucalypti* is an obligate pathogen on *Eucalyptus*, whereas *Coniella* spp. have wide host ranges and may require leaf damage or prior infection by other pathogens to invade leaves. However, they occur mostly on the lower crowns of *Eucalyptus*, are favoured by moist climates and are generally regarded as being of minor importance. Nevertheless as clonal forestry becomes more common in the South-East Asian region, some clones may be particularly susceptible to one or more of these pathogens, which could cause significant damage. For example, the *E. grandis* provenance shown in Figs 42–45 was very susceptible to *Microsphaeropsis* sp. and was eliminated from a provenance trial in Sri Lanka on this basis.

**Control**

No control measures are warranted with these fungi, except for elimination from provenance or clonal trials of any selection that shows unusual susceptibility. Their importance also lies in possible mis-diagnosis of these fungi for more damaging pathogens.

**References**


Figure 35. *Eucalyptus globulus* infected by *Aulographina eucalypti*

Figure 36. Detail of lesions, showing corky radiate appearance

Figure 37. *Aulographina eucalypti*: A. an ascus containing eight ascospores; B. ascospores. Bar = 5 µm for A and B.
Figure 38. *Coniella* leaf spot on *Eucalyptus camaldulensis*

Figure 39. Pycnidium of *Coniella fragariae*, bar = 40 µm

Figure 40. Conidia of *C. fragariae*, bar = 12 µm

Figure 41. Leaf spots associated with infection by *C. fragariae*; note concentric arrangement of pycnidia on the right-hand leaf
Figure 42. Leaf and stem lesions of *Eucalyptus grandis* caused by *Microsphaeropsis globulosa*

Figure 43. Detail of lesion showing scattered black pycnidia exuding spore masses

Figure 44. Detail of lesion showing raised reddened margins common to infection of *E. grandis* by this pathogen

Figure 45. Infected foliage of *E. grandis* with straw-coloured lesions and reddened margins

Figure 46. Pycnidium of *M. globulosa*, bar = 27 µm

Figure 47. Conidia of *M. globulosa*, bar = 7 µm
Figure 48. *Microsphaeropsis* spp. and *Coniella* spp.: A, B. conidia and conidiogenous cells of *M. callista* on *Eucalyptus obliqua*; C. conidia of *M. globulosa* on *E. grandis*; D, E. conidia and conidiogenous cells of *C. fragariae* on *E. urophylla*; F. conidia of *C. australiensis* on *E. camaldulensis*. Bar = 5 µm for A and C; 8 µm for D and F; 10 µm for B; and 12.5 µm for E.
Figure 49. *Eucalyptus camaldulensis* infected by *Pseudocercospora* sp.

Figure 50. Conidium of a *Pseudocercospora* sp., bar = 15 µm

Figure 51. Juvenile leaves of *E. camaldulensis* heavily infected by *Pseudocercospora* sp.
Figure 52. *Pseudocercospora* spp.: A, B. conidiophores and conidia of *Pseudocercospora* sp. on *Eucalyptus* sp.; C, D. conidiophores and conidia of *Pseudocercospora* sp. on *E. camaldulensis* from Vietnam. Bar = 25 µm for A, B, and C; and 15 µm for D.
Cryphonectria cankers

Diseases
Cryphonectria cankers

Causal organisms
Cryphonectria cubensis
Diaporthe cubensis Bruner
Cryphonectria cubensis (Bruner) Hodges

C. eucalypti
Endothia gyrosa (Schwein.:Fr.) Fr.
C. eucalypti M. Venter & M.J. Wingf.

C. gyrosa
E. tropicalis Shear & N.E. Stevens
E. havanensis Bruner
C. gyrosa (Berk.& Br.) Sacc.

Cryphonectria cubensis was first described as Diaporthe cubensis by Bruner (1917), who noted the pathogen on Eucalyptus cankers in Cuba. Hodges (1980) questioned the generic affinity of D. cubensis, placing it in the genus Cryphonectria on the basis of the stromatic tissue, the arrangement of the perithecia in the stromata and especially the presence of septate ascospores (Figs 57, 58). Walker et al. (1985) provided a summary of the taxonomy of Endothia and Cryphonectria spp. found on Eucalyptus. Currently, the taxonomy of these fungi is confused. In the near future, however, DNA-based studies are likely to resolve outstanding issues. For example, recent analyses of DNA-sequence data have shown that C. cubensis collections from Asia and South America represent discrete groups that may be distinct species (Myburg et al. 1999). Also, the fungus that has been known as C. cubensis in South Africa is distinctly different to that occurring elsewhere in the world (Myburg et al. 2002) and has recently been found on native Myrtaceae. Although very similar to C. cubensis, this fungus is a discrete species and is currently being described (Heath et al. 2002). There is also good evidence to suggest that the genus Cryphonectria is probably not appropriate for C. cubensis and related fungi.

Endothia gyrosa (syn. C. eucalypti) was identified as a widespread pathogen of Eucalyptus in Australia by Walker et al. (1985). It has subsequently been found in plantations in several parts of the world, including South Africa (van der Westhuizen et al. 1993). This fungus has recently been reclassified as a Cryphonectria species on the basis of stromatic features and DNA analysis (Venter et al. 2002). It is, however, quite distinct from other Cryphonectria spp. by virtue of its allantoid, aseptate ascospores (Fig. 59E).

Cryphonectria gyrosa is intermediate in morphology between C. cubensis and C. eucalypti, with septate ascospores, well developed orange stromata, and perithecia developing at
the base of the stromata. Perithecia bear long necks which grow upwards and form ostioles on the stromatic surface. Pycnidia, on the other hand, are of the *Dendrophoma* type with spheroidal bases and elongated necks (Fig. 60A). Conidia of *C. cubensis* and *C. gyrosa* are very small and spheroidal (Fig. 60B), whereas those of *C. eucalypti* are slightly curved in shape (Fig. 59B).

**Host range**

Most records of *C. cubensis* have been from *Eucalyptus* spp. on which the fungus is a major pathogen. Hodges et al. (1986), however, suggested that *C. cubensis* may also cause cankers on other Myrtaceae, and showed the fungus to be conspecific with *Endothia eugeniae* (Nutman & Roberts) Reid & Booth, a pathogen of cloves, *Syzygium aromatica*. Similarly Wingfield et al. (2001) have shown that *Tibouchina* spp. (Myrtales: Melastomataceae), which are native to South America and are grown as ornamentals in many parts of the world, are commonly infected by *C. cubensis*. This fungus was also recently found on water berry (*Syzygium cordatum*), a plant native to South Africa (Heath et al. 2002).

*Cryphonectria gyrosa* has been recorded from *Eucalyptus* in Japan (Kobayashi 1970), Florida (Barnard et al. 1987), India (Sharma et al. 1989), Vietnam (Old and Yuan unpublished) and as *Endothia havanensis* Bruner from several other woody hosts in Japan (Kobayashi 1970).

As *C. eucalypti* has not been recorded in South-East Asia, this fungus will not be further considered.

**Distribution**

*Cryphonectria cubensis* is a widespread and important pathogen of plantation eucalypts in the tropics and subtropics. It has been recorded from Surinam (Boerboom and Maas 1970), Brazil (Hodges and Reis 1974), Cuba (Bruner 1917), Puerto Rico, Florida and Hawaii (Hodges et al. 1979), Mexico (Wingfield unpublished), Colombia (van der Merwe et al. 2001), Trinidad and Western Samoa (Hodges 1980), India and Cameroon (Sharma et al. 1985), Congo (Roux et al. 2000), and Tanzania (Hodges 1980). Records from South-East Asia include Hong Kong (Sharma et al. 1985), Indonesia (Wingfield unpublished), Thailand (Wingfield et al. unpublished) and Vietnam (Thu unpublished). The only records from more temperate regions are from South Africa (Gibson 1981, Wingfield et al. 1989, Conradie et al. 1990), where the fungus probably represents a different species, and the south-west of Western Australia (Davison and Coates 1991).

The likely reclassification of *C. cubensis* found on *Eucalyptus* and other Myrtaceae into at least two distinct species will require the host ranges and distribution of these pathogens to be re-examined in the future.

*Cryphonectria gyrosa* is a less aggressive pathogen than *C. cubensis* and is infrequently recorded. Collections have been made in Japan (Kobayashi 1970), Florida (Barnard et al. 1987) and India (Sharma et al. 1989). Old found this fungus in Vietnam (Old and Yuan unpublished) on *E. camaldulensis* and in Indonesia on *Corymbia citriodora* (Old and Yuan unpublished).
Symptoms

Whole-tree symptoms associated with infection by *C. cubensis* include basal cankers, which can extend several metres up the stem (Fig. 53), and less severe cankers often associated with branch stubs. Where stems have been girdled, trees may wilt and die suddenly during hot dry weather. Older trees, which have survived initial infection, often develop basal swellings and severe bark cracking over brown necrotic sapwood (Fig. 55). Infected stems become discoloured, deep red or brown, through copious secretion of kino from cankers, which dries on the bark surface. Large numbers of sexual or asexual fruiting structures are produced either on the bark surface or in fissures and can be seen with the naked eye or using a hand lens (Fig. 54). Both perithecia (sexual) and pycnidia (asexual) fruiting bodies may be produced concurrently on necrotic tissue. Perithecia form globose bases under the bark surface from which the necks protrude, especially in humid weather, to discharge septate ascospores (Figs 56, 57). Conidia are produced within pycnidia, which are often borne superficially on the bark and bear long necks which ooze masses of yellow, elliptical conidia under humid conditions.

*Cryphonectria gyrosa* is a secondary pathogen of stressed trees, for example, trees which have been severely defoliated by *Cylindrocladium* shoot blight, forming elongate stripe cankers which often persist without killing the trees. Both perithecia and the *Dendrophoma* pycnidia are formed at the margins of healthy and diseased bark or on recently-dead branches.

Pathology

Infection by both of these fungi is through wounds. Natural growth cracks at the base of rapidly growing trees are a common means of entry. Other avenues include branch stubs where the pathogen gains entry through poorly occluded suppressed branches. If coppice rotations are practiced (Barnard *et al.* 1987) the pathogens can grow from basal cankers on stumps to attack newly developed coppice stems. For *C. cubensis*, the most common infection propagules appear to differ in different parts of the world. Ascospores and conidia are found in South America (Hodges 1980), India (Sharma *et al.* 1989), Indonesia, Thailand and Vietnam (Old and Wingfield, personal observation). In Africa, only conidia have been found (Wingfield *et al.* 1989). Spread of *C. cubensis* appears to be favoured by high rainfall (>2000 mm), high humidity throughout the year, temperatures which average 23ºC or higher, and the presence of susceptible host species. Unlike the more aggressive *C. cubensis*, information on the pathology of *C. gyrosa* is limited.

Impacts

The main damage to trees caused by *C. cubensis* is the development of large basal cankers which can kill trees during the first 2-3 years of growth. On older trees, extensive perennial cankers develop several metres in length up the bole of the tree. Under favourable climatic conditions with susceptible species or clones, up to 50% of stems in plantations have been killed (Alfenas *et al.* 1983). Cankers are characterised by death of phloem, cambium and sapwood with partial girdling of trees and copious flow of kino.

Economic effects of *C. cubensis* canker are reduced growth rate (Camargo *et al.* 1991), reduced coppicing (Hodges and Reis 1976, Sharma *et al.* 1985, Barnard *et al.* 1987), and increased mortality (Boerboom and Maas 1970, Hodges *et al.* 1979). Wood yield was significantly reduced when cankers extended to more than 25% of the commercially useful stem length (Ferrari *et al.* 1984). Compared with normal wood, cankered wood contains
more extractives and lignin, and is denser with shorter fibres and thinner cell walls (Foekkel et al. 1976). Although the main problem in processing wood from affected stands is the loss in pulp yield, an increase in extractives adversely affects bleaching. Pulp of a quality similar to that from healthy stands can be produced, provided infestation levels are below 34%. Stands with more than 50% of trees infected cannot be recommended for pulping (Foekkel et al. 1981).

Control

Some *Eucalyptus* spp. are very susceptible to *C. cubensis*, e.g. *E. saligna*, and the disease has been a major impediment to cultivation of this species. *Eucalyptus grandis* can also be highly susceptible. In Brazil, control has been highly successful through cultivation of *E. grandis* x *E. urophylla* hybrids and selection within these hybrids for resistant genotypes. Clonal propagation of resistant genotypes has provided an effective control of the disease in Brazil, and in South Africa selection for resistant hybrid clones based on *E. grandis* and many other pure species has been in progress for the last decade (Van Zyl and Wingfield 1999, Van Heerden and Wingfield 2002).

References


Figure 53. *Eucalyptus urophylla* clone severely damaged by *Cryphonectria cubensis*, northern Vietnam.

Figure 54. Perithecial necks of *C. cubensis* protruding from infected bark.

Figure 55. *E. grandis* hybrid with recent infection by *C. cubensis*; note necrotic sapwood under the cracked bark.

Figure 56. Section through a peritheciun with a dense mass of asci and ascospores, bar = 82 µm.

Figure 57. Asci of *C. cubensis* showing eight septate ascospores within the asci, bar = 7 µm.
Figure 58. Cryphonectria cubensis: A. longitudinal section of a perithecium; B. asci; C. ascospores. Bar = 70 µm for A; and 8 µm for B and C.
Figure 59. Cryphonectria eucalypti on Eucalyptus nitens: A, B. conidia and conidiogenous cells of Endothiella anamorph; C. longitudinal section of conidiomata; D, E. asci and ascospores of C. eucalypti; F. longitudinal section of ascomata. Bar = 12.5 µm for A and D; 5 µm for B; 250 µm for C; 15 µm for E; and 300 µm for F.
Figure 60. Cryphonectria gyrosa on Eucalyptus camaldulensis:  A. Portion of stem showing superficial pycnidia of Dendrophoma anamorph and immersed perithecia of C. gyrosa in brightly coloured stromata; B, C. conidia and conidiogenous cells of Dendrophoma sp.; D. asci of C. gyrosa; E. ascospores of C. gyrosa.  Bar = 10 µm for B-E; A not scaled.
Coniothyrium canker

Disease
Coniothyrium canker

Causal organism
Coniothyrium zuluense M.J. Wingf., Crous & T.A. Coutinho

Host range and distribution
This disease, known only on Eucalyptus spp., was first detected on clones of E. grandis in the KwaZulu Natal province of South Africa in 1991 (Wingfield et al. 1997). Infections were initially limited to a small locality but the disease spread rapidly throughout all subtropical areas of South Africa (Van Zyl et al. 2002a). In 2002, coniothyrium canker was reported on E. camaldulensis in Thailand (Van Zyl et al. 2002b) and was recently found in Mexico (Roux et al. 2002), Vietnam (Old and Wingfield unpublished), Argentina and Uruguay (Wingfield unpublished). There is circumstantial evidence to suggest that the disease in South and Central America was introduced from South Africa, possibly with seed. Thus far, all areas where the disease is found are in the tropics and subtropics.

Symptoms
Initial symptoms of infection appear as discrete black, or dark brown, sunken lesions on the young green tissue at the tops of trees (Fig. 61). Trees appear not to be infected prior to the second year of growth. On highly susceptible trees, lesions are numerous and merge to form large necrotic patches on the stems. Infections penetrate the cambium and result in obvious kino pockets in the wood (Figs 62, 63). Copious exudation of kino is common from these lesions. Because kino is water soluble, severely infected trees tend to have stems stained from red to dark black. This is particularly obvious in clonal stands where resistant trees are planted alongside those that are highly susceptible.

New infections generally occur each year on new growth. Thus, on highly susceptible trees discrete cankered areas can be seen sequentially up the stems of trees and on young shoots (Fig. 64). Severely cankered stems tend to produce epicormic shoots around the cankers. As these shoots grow, tops of trees commonly die. In severely infected clonal stands, height growth ceases and trees assume brush-like flattened crowns. In seedling stands or stands comprised of mixed clones of varying susceptibility, severely infected trees are suppressed by trees that grow more vigorously, and large numbers of trees can die.

Pathology
Little is known regarding the pathology of C. zuluense. The pathogen appears to be favoured by hot humid conditions and to infect young green stem tissue in the spring. The pathogen produces large numbers of small slightly darkened asexual spores (conidia) in small flask shaped pycnidia on the surface of lesions (Figs 65-68). These spores exude from the pycnidia
in wet weather and are apparently distributed by rain splash. Infections are thought to occur directly through the green bark in the absence of wounds.

Isolations from pycnidia commonly result in mixed cultures of *C. zuluense* and two bacteria belonging to the genus *Pantoea*. There may be a synergistic relationship between the fungus and the bacteria as co-inoculation with the two organisms induced lesions significantly larger than those associated with *C. zuluense* alone (Van Zyl 1999).

**Impact**

Coniothyrium canker is one of the most serious diseases affecting plantation development in South Africa. When the disease first appeared in that country, many clones were highly susceptible to infection and had to be replaced. In severe cases, the disease leads to death of leading stems and in some cases trees die. Infected trees have many lesions on the stems, severely hindering bark removal and reducing log value at pulp mills that require timber free of bark. Timber intended for solid wood products contains kino pockets that greatly down-grade its value.

**Control and management**

Coniothyrium canker is a relatively recently recognised disease of *Eucalyptus* and it appears to be rapidly spreading to new areas. In South Africa, where the disease has been known longest, substantial success has been achieved in reducing its impact. This has generally been through the selection of disease-tolerant clones. *Eucalyptus grandis* appears to be highly susceptible although some clones of this species relatively resistant to the disease have been identified. In South Africa, hybrids of *E. grandis* with *E. camaldulensis* and *E. urophylla* have been selected for tolerance to infection by *C. zuluense*. Thus, selection of resistant planting stock offers many opportunities to manage coniothyrium canker. There is good evidence, however, that clones lose resistance with time, presumably due to changes in the pathogen. Thus avoidance through breeding and selection is likely to be an ongoing and costly process.

**References**


Figure 61. Stem of *Eucalyptus grandis* showing small cankers associated with *Coniothyrium zuluense* infection

Figure 62. Stem shown in Fig. 61 with bark removed showing kino pockets in the sapwood

Figure 63. Cross-section of stem with included kino pockets; the cankers make bark removal difficult and reduce stem quality for pulping

Figure 64. Girdling canker on young shoot of *E. grandis*
Figure 65. Stem canker caused by *Coniothyrium zuluense* showing small pycnidia embedded in the lesion

Figure 66. Section through a pycnidium showing conidia of *C. zuluense*, bar = 24 µm

Figure 67. Detail of conidia, bar = 10 µm
Figure 68. *Coniothyrium zuluense*: A. longitudinal section of a conidioma; B. conidia and conidiogenous cells; C. conidia. Bar = 15 µm for A; 5 µm for B; and 7.5 µm for C.
Pink disease

Disease
Pink disease

Causal organism
_Erythricium salmonicolor_ (Berk. & Br.) Burds.
As _Corticium salmonicolor_, this fungus is described in IMI Description 511 (Mordue and Gibson 1976). Other synonyms include _Thanatephorus, Phanerochaete_ and _Pellicularia_ spp.

Host range
The fungus has a very wide host range, including _Eucalyptus, Acacia_ and other genera important in forestry plantations. _Erythricium salmonicolor_ is also a pathogen of fruit trees in orchards and in home gardens. It has caused serious damage to many tropical crops such as cacao, citrus coffee, tea, and rubber (Hilton 1958, Browne 1968).

Known distribution
Worldwide in the tropics and subtropics, e.g. Australia (on fruit trees, not recorded on _Eucalyptus_) (Penrose 1987); New Zealand; India on many hosts (Seth _et al._ 1978); Japan on citrus (Oniki _et al._ 1985); Vietnam, Laos and Sri Lanka on rubber and acacias (Old personal observation); Malaysia on rubber and acacias (Hilton 1958, Chin 1990, Lee 1993), Papua New Guinea (Muthappa 1987), Indonesia on rubber and acacias including Kalimantan (Hadi and Nuhumara 1997) and Sumatra (Zulfiyah and Gales 1997). Records of damage on _Eucalyptus_ include India (Seth _et al._ 1978, Sharma _et al._ 1999); Philippines (Soriano personal communication); Indonesia (Sumatra) (Wingfield unpublished); Zambia (Shakacite personal communication); South Africa (Roux _et al._ 2002); Costa Rica (de Segura 1970); Brazil (Ferreira and Alfenas 1977, Ferreira 1989) and Vietnam (Old unpublished).

Symptoms
_Erythricium salmonicolor_ attacks trees of all ages. The fungus forms four distinct types of growth on stems and branches, namely ‘cobweb’, ‘pustule’, ‘necator’ and ‘pink incrustation’. The cobweb stage is the first sign of infection with white, sparse mycelium growing rapidly across stem surfaces (Figs 69, 70). As the fungus invades the bark and cambium, a diffuse canker develops and can be seen by stripping off the bark (Fig. 71). By this stage, pink aggregates or pustules of sterile mycelium form on the bark surface and these may later develop into the necator stage which is conidial, orange-red in colour and borne mainly on the upper side of branches. The necator stage is less frequently seen than the pustule phase and is formed late in the disease cycle. The salmon pink encrusting hymenia of the _Erythricium_ teleomorph stage is the characteristic symptom of this disease and occurs mostly on the underside of dead and dying branches (Fig. 72).
Whole-tree symptoms include crown dieback and stem breakage, especially during high winds. Dieback is due to elongated diffuse cankers, which eventually girdle stems. *Eucalyptus* and acacias respond to infection by production of clusters of epicormic shoots in the vicinity of cankers and these are useful indicators of disease. Crown dieback, stem breakage and tree death lead to gaps in the plantation, further increasing susceptibility to wind damage.

**Pathology**

Pink disease is prevalent in high-rainfall areas or locations where mists persist. The disease is spread between trees by windborne basidiospores and conidia and within the crowns of affected trees by the rapid growth of the cobweb stage. All these aspects of disease spread are favoured by high rainfall. The fungus is a primary pathogen, able to invade healthy intact bark and attack the cambium (Seth *et al.* 1978). The role of surrounding vegetation, especially susceptible crops such as rubber (Fig. 69) and acacias, as sources of inoculum for the fungus has been documented for eucalypt plantations in India (Seth *et al.* 1978).

**Impact**

Most reports of *E. salmonicolor* causing significant damage to eucalypt plantations come from southern India (Seth *et al.* 1978, Sharma *et al.* 1984). The pathogen causes economic damage on many species of *Eucalyptus* in Brazil (Ferreira 1989) and has limited the use of some clones of *E. grandis* and hybrids in some regions of that country. Similarly, *E. grandis* x *E. urophylla* hybrids have been severely damaged by the pathogen in northern Sumatra (Wingfield unpublished). In India, Seth *et al.* (1978) estimated losses to mortality in 5-11-year-old plantations of *E. tereticornis* at 55-96%. In south-eastern Vietnam, pink disease is more commonly a problem on acacias with significant damage being recorded on *Acacia mangium* and hybrids between *A. mangium* and *A. auriculiformis* (Thu unpublished) but the disease has been found on *E. camaldulensis* in high-rainfall locations in central Vietnam (Fig. 73). The impact of the disease may not be immediately apparent as diseased trees may be most common within stands, where the humidity is higher than at the edge of plantations.

**Control and management**

As indicated by Old *et al.* (2000), in rubber plantations and fruit tree orchards, the disease can be successfully controlled through early recognition of the symptoms followed by prompt application of suitable fungicides. Bordeaux mixture, which is an aqueous suspension of CuSO$_4$: CaO: H$_2$O (1:2:10), and a brush-on formulation of tridemorph, applied at regular intervals have been shown to be effective in controlling the disease in rubber plantations and mango orchards in Malaysia (Lim and Khoo 1985). These measures are unlikely to be economical, nor practicable, in forest tree plantations. The strategy with most promise is use of species, provenances or clones which are resistant to pink disease, especially in high-risk regions with high rainfall. Selection of such trees is most advanced in southern India, where clonal propagation of *E. tereticornis* and selection of clones resistant to both leaf blight and pink disease has been successful (Sharma *et al.* 1999).

**References**


Figure 69. Rubber tree showing spreading mycelium of *Erythricium salmonicolor*

Figure 70. Infected stem covered with mycelium of pink disease

Figure 71. Stem shown in Fig. 70 with bark stripped off to show a diffuse canker which has girdled the stem

Figure 72. Dead branch encrusted with pink mycelium of *E. salmonicolor*

Figure 73. *Eucalyptus camaldulensis* with girdled main stem and poor form due to pink disease
Figure 74. *Corticium salmonicolor*: A. basidium and basidiospores; B. conidiogenous cells and conidia; C. hypha with a ‘clamp connection’ commonly found in basidiomycete mycelium; D. longitudinal section of fruiting structure producing conidia. Bar = 15 µm for A; 20 µm for B and C; and 120 µm for D.
Stem canker diseases

Disease
Stem and branch cankers

Causal organisms

Botryosphaeria spp. and their anamorphs, Lasiodiplodia theobromae (Pat.) Griff. & Maubl., Dothiorella and Fusicoccum. Most collections on Eucalyptus have been placed in the B. dothidea–B. ribis complex. These species have often been regarded as synonyms but the taxa have recently been separated on the basis of DNA analyses (Zhou and Stanosz 2001, Slippers et al. In press). Dothiorella is regarded by Crous and Palm (1999) as a synonym of the earlier described genus Diplodia.

Valsa., anamorphs Cytospora spp.

Host range
A wide range of trees and woody shrubs, in plantations and native vegetation throughout temperate and tropical regions, including eucalypts, acacias and other forestry species and fruit, shade and amenity trees.

Known distribution
Botryosphaeria spp. and their anamorphs are associated with stem cankers of Eucalyptus and other plantation species in tropical and temperate regions worldwide (Roux and Wingfield 1997, Old and Davison 2000). Cytospora eucalypticola van der Westhuizen is common in native forests and plantations in Australia and has been found in South Africa, where it was first described (van der Westhuizen 1965). Old et al. (1991) found a teleomorph for this fungus that could not be separated from Valsa ceratosperma (Tode: Fr.) Maire, a fungus with a world-wide distribution on many woody hosts. Both anamorphs and teleomorphs of this fungus have been found by the authors associated with stem cankers of Eucalyptus in Sri Lanka, Vietnam, Indonesia and Thailand.

Symptoms
Stem cankers are dead areas of bark which sometimes extend into the underlying sapwood. They vary in size from localised lesions confined by callus tissue to sunken lesions which may extend more than a metre along the branch or stem axis. Such ‘diffuse’ cankers may be darkly discoloured and cracked especially toward the centre. Eucalyptus stems usually secrete kino from these lesions, discolouring the bark with reddish to dark brown pigments (Figs 77, 82). Branches and stems may be partially or completely girdled, causing crown dieback and possibly tree death. Fruiting bodies of the causal fungi can usually be found on the cankers themselves, especially at the margins between diseased and healthy bark or on newly dead branches. The fruiting bodies (Fig. 76) are typically partially submerged in the outer bark but can be readily seen through a hand lens. Cankers are often associated with wounds or borer damage, or with branch stubs.
Identification of the causal agents of cankers is often quite difficult, requiring detailed examination of fruiting structures (Figs 78–81). In this section several species commonly found in eucalypt plantations in the tropics are illustrated, with drawings of the structures most useful in identification (Figs 81, 83). Fungi associated with cankers span a range of pathogenicities. *Botryosphaeria* spp. and their anamorphs cause cankers and dieback on many woody species (Figs 75–80) whereas *Valsa* spp. (Figs 82, 83), usually found as the anamorphic state, *Cytospora*, are opportunistic pathogens with little capacity to invade healthy trees. Although no attempt is made here to ascribe pathogenic roles to individual species of *Botryosphaeria*, advances are being made in separating members of the *B. dothidea* complex, including those most frequently found on *Eucalyptus* (Smith et al. 2001, Slippers et al. In press). Doubtless many other fungal genera are able to invade stressed trees or live as saprophytes or endophytes in woody tissues of eucalypts but there is little information available on their occurrence and pathogenic status in South-East Asia.

**Pathology**

Trees planted in unsuitable environments, for example infertile soils and climates to which they are poorly adapted (drought-prone areas), are more susceptible to canker diseases (Crist and Schoeneweiss 1975). Trees in dense stands, especially if suppressed, subject to insect attack (either defoliation or stem borers), are also more readily invaded. Poor silvicultural operations resulting in stem wounds also predispose trees to infection. These fungi are likely to be present at low frequency in all stands, and vigorous trees can be infected but show no symptoms in the absence of environmental stress.

Some canker fungi can gain access to stem tissue through wounds, either naturally caused or resulting from pruning of branches. Other species, including *Botryosphaeria* spp. and their anamorphs and probably *Valsa* spp., can infect healthy shoots and remain as endophytes in healthy tissue until trees are stressed by drought, temperature extremes or defoliation (Smith et al. 1996). Both groups of canker pathogens kill cambial tissue and sapwood but do not cause decay. Open lesions exposed by death of bark and cambium, however, provide avenues for infection by decay fungi.

**Impacts**

Impacts of these pathogens are usually small in vigorous, well-managed stands, and occurrence may be limited to suppressed trees. Where stands are stressed, for example where species or provenances are poorly adapted to climatic and edaphic factors (Shearer et al. 1987), incidence of cankers can be significant. In Australia, where eucalypts are commonly exposed to chronic insect attack, Old et al. (1990) demonstrated that defoliated trees were more susceptible to invasion by *B. ribis* Grossenb. & Duggar and *Cryphonectria eucalypti* (syn. *Endothia gyrosa*). Defoliation of eucalypt plantations by insects is relatively uncommon in South-East Asia but trees are often grown in suboptimal conditions and can suffer extreme defoliation by leaf blight pathogens (Fig. 75). Cankers caused by *B. ribis* were the most common and serious symptoms of disease of *Eucalyptus* in central Japan, where the climate is generally unsuitable for their cultivation (Old and Kobayashi unpublished).

Pongpanich (unpublished) showed that *B. ribis* is a significant pathogen in plantations of *E. camaldulensis* in western Thailand where clonal trees are grown on a large scale. Some eucalypt clones were found to be very susceptible to stem invasion and have been discarded in favour of more resistant clones (Fig. 77). Also in Thailand, large clonal plantations of *E. camaldulensis* have been found susceptible to *Cryptosporiopsis eucalypti* and
Phaeophleospora destructans. Trees defoliated by these pathogens were rendered extremely susceptible to invasion by *L. theobromae* and many hectares of young trees were killed by this combination of defoliation and invasion by stem pathogens (Fig. 75). Similar damage, though on a less extensive scale, has been recorded in trials of *E. camaldulensis* in southeastern Vietnam, where provenances defoliated by *Cylindrocladium reteaudii* and *C. eucalypti* were killed by stem invasion by *L. theobromae*, *C. gyrosa* and *Cytospora eucalypticola*.

**Control and management**

The only feasible control is through good matching of species and provenance to climatic and edaphic factors and avoidance of stress through good silviculture (spacing, thinning). Where clonal forestry is practised there is a need for extensive clonal trials, prior to widespread release of clones to farmers, to ensure that trees are relatively resistant to foliar and stem pathogens.

**References**


Figure 75. Defoliation of this *Eucalyptus camaldulensis* clonal plantation in Thailand was followed by rapid invasion of main stems by *Lasiodiplodia theobromae*, causing widespread tree death. The tree on the left was resistant to defoliation and did not suffer from cankers.

Figure 76. Detail of fruiting bodies of *Botryosphaeria ribis*. Photo K. Pongpanich

Figure 77. *E. camaldulensis* stem lesion associated with *Botryosphaeria* sp.

Figure 78. Perithecium of *Botryosphaeria* showing asci, bar = 15 µm

Figure 79. Conidia of *Fusicoccum*, an anamorphic state of *Botryosphaeria*, bar = 13 µm

Figure 80. Ascospores of *B. appendiculata*, bar = 12.5 µm
Figure 81. *Botryosphaeria* and anamorphs on *Eucalyptus* sp.: A. longitudinal section of ascoma of *B. appendiculata* from Vietnam; B. ascus and ascospores of *B. appendiculata*; C. conidia of *Lasiodiplodia theobromae*; D. conidiophores of *Fusicoccum* sp.; E. conidia of *Fusicoccum* sp.; F. longitudinal section of conidioma of *Fusicoccum* sp.. Bar = 25 µm for B; and 70 µm for C.
Figure 82. *Eucalyptus grandis* with stem infections by *Cytospora eucalypticola*
Figure 83. *Cytospora eucalypticola* and *Valsa ceratosperma*: A. longitudinal section of conidiomata of *C. eucalypticola*; B. conidiophores of *C. eucalypticola*; C. conidia of *C. eucalypticola*; D. asci of *V. ceratosperma*; E. ascospores of *V. ceratosperma*; F. longitudinal section of ascomata of *V. ceratosperma*. Bar = 160 µm for A; 8 µm for B and E; 6 µm for C; 12 µm for D; and 250 µm for F.
Bacterial Wilt

Causal organism

*Ralstonia solanacearum* (Yabuuchi *et al.* 1995) Smith

Synonyms: *Bacillus solanacearum* (Smith), *Pseudomonas solanacearum* (Smith) Smith, *Burkholderia solanacearum* (Smith) Yabuuchi *et al.*

Host range

Eucalypt-infecting strains are all Race 1 (the race with a broadest host range) and either biovar 1 (in South America) or biovar 3 (Asia and Australia) (Gillings and Fahy 1993). These biovars also attack a wide range of hosts including many woody species, e.g. casuarina, olive, teak, neem, cassava and cashew (Hayward 1993). Eucalypt hosts recorded so far include *E. camaldulensis*, *C. citriodora*, *E. grandis*, *E. ‘leizhou’, E. mellita*, *E. propinqua*, *E. saligna, E. urophylla* and *E. grandis* (Ciesla *et al.* 1996).

Distribution


Symptoms

These pathogens are soil-borne, and disease symptoms develop shortly after planting (Figs 84, 85). Affected trees are often scattered through the stand and show wilting, leaf drop, stem death and reduced growth rate (Fig. 85). Vascular discoloration commonly occurs, roots die and basal cankers may be found on affected trees (Figs 86, 87). Symptomatic trees usually wilt and die. Stem sections, cut through discoloured vascular tissue (Fig. 87) exude bacterial masses if incubated moist in plastic bags for 24 hours or suspended in water (Fig. 88). Selective media, serological and molecular methods including DNA analyses are routinely used to detect and type bacterial isolates (Seal and Elphinstone 1994).

Pathology

There is a large body of physiological, biochemical and ecological information indicating that *R. solanacearum* is a complex and heterogeneous species, causing disease on an extremely wide range of crops. A full description, based on standard morphological and biochemical characteristics, is given in Saddler (1994) and a summary of the subspecific classification system is described in Hayward (1991) and Gillings and Fahy (1993). The latter authors also describe rapid DNA-based methods for detecting the bacteria and identifying isolates to subspecific levels. The biology and epidemiology of *R. solanacearum* are discussed by Hayward (1991).
Bacteria occur in soil but the relationship between the epidemiology of disease on other hosts and the disease on *Eucalyptus* is not known. On other hosts, spread can be by movement of cuttings, storage organs and seed. There is also some evidence for insect vectors. Local splash-dispersal within plantations from cankers on infected eucalypt stems seems likely. Sequences and lengths of crop rotations may also be a factor in the incidence of bacterial wilt in *Eucalyptus*. For example, experience in Vietnam and eastern Thailand suggested that wilt of clonal plantings of *E. camaldulensis* occurred more commonly when eucalypts were planted in fields formerly growing cassava.

**Impacts**

In severely affected plantations of *E. urophylla* in northern Vietnam, tree mortality was as high as 30% one year after planting. In a plantation of 13-month-old *E. pellita* examined in northern Queensland by the authors in 1996, significant numbers of one-year-old trees suffered bacterial wilt. Eighteen months later, no newly diseased trees were found and some recovery of trees was evident through healing of basal cankers. In older trees, such cankers may be invaded by secondary pathogens causing stem and butt rot with increased incidence of wind-throw.

**Control and management**

Bacterial wilt can originate from infected nursery stock, although for *Eucalyptus*, *R. solanacearum* is rarely reported as a nursery problem. Different *Eucalyptus* species undoubtedly vary in their susceptibility to disease, with *E. pellita* and *E. urophylla* often being susceptible. Variation in resistance to bacterial wilt has been identified for a range of eucalypt species in Brazil (Dianese and Dristig 1993). Clonal variation in susceptibility also occurs, with evidence from South Africa that the disease is restricted to some inter-specific hybrid clones. The circumstantial evidence for enhanced disease incidence following cassava is of interest, and other crop sequences may influence pathogen populations. No control practices for bacterial wilt of eucalypts have been evaluated, nor can any be recommended. For example, culling of diseased trees would not be effective as the pathogen would remain in infected roots and infested soil.

**References**


Figure 84. *Eucalyptus urophylla* clonal planting affected by bacterial wilt (*Ralstonia solanacearum*) in northern Vietnam  
Figure 85. Clonal plantation showing scattered bacterial wilt disease  
Figure 86. Stem with sapwood exposed showing dark staining  
Figure 87. Transverse section of affected stem  
Figure 88. Stem section incubated 24 h within a plastic bag to promote bacterial oozing
Woody root and stem rots of living trees

Causal organisms
Several species within the Class Basidiomycotina (Basidiomycetes), which encompasses fungi with sexually-produced spores borne on macroscopic fruiting structures bearing gills, pores or smooth hymenial surfaces. Important pathogens include species of Armillaria, Ganoderma and Phellinus, especially P. noxius (Corner) G.H. Cunn. These fungi invade woody roots and can gain access to butts and stems of trees via this route, in addition to infecting above-ground stems and branches. Several other genera can invade stems through wounds and cause heart rots of standing trees. Their importance in eucalypt plantations in South-East Asia, however, is probably slight as rotations are usually less than ten years and heart rots are unlikely to be extensive in such young trees.

Host range
These fungi have very wide host ranges including eucalypts, acacias and other species grown in forestry plantations. Ganoderma species are also important pathogens of rubber, oil palm and other plantation species, and P. noxius causes brown root rot of many woody hosts throughout the tropics (Ivory 1996).

Known distribution
Armillaria luteobubalina Watling and Kile is the most damaging of the four Armillaria species recorded on Eucalyptus and Corymbia spp. in Australasia (Kile 2000), but this fungus is indigenous to southern and eastern Australia and has not been found on other continents. For the region of interest of this manual, there are records of Armillaria spp. causing disease of Eucalyptus in Papua New Guinea (Arentz and Simpson 1989) on E. grandis, E. globulus ssp. bicostata and E. robusta, and causing death of E. grandis on high altitude sites in northern Sumatra (Wingfield unpublished).

Ganoderma species are commonly recorded as root and stem rot pathogens of plantations in South-East Asia (Figs 89–90) and are particularly important in acacia plantations (Old et al. 2000). Ganoderma spp. have been reported as root rot pathogens of Eucalyptus in southern Africa (Masuka and Nyoka 1995), India (Bakshi 1974, 1976), and more widely as agents of heart rot. Kile (2000), however, does not list any such disease records for South-East Asia. Ganoderma philippii (Bresad. & P.Henn) Bresad is regarded as a cause of root rot of acacias in Sumatra, Indonesia, where large areas are planted with A. mangium. Ganoderma root rot is also important on a wide range of crops in Malaysia including rubber, tea cocoa and oil palms (Varghese and Chew 1984). The observation of root rot patches in a plantation of E. pellita near Pekanbaru, Sumatra, with Ganoderma fruiting on recently-dead trees suggested that this fungus was the cause of Eucalyptus deaths (Old personal observation). Phellinus noxius is pan-tropical in distribution (Figs 92, 93) and Ivory (1996) lists Corymbia citriodora and six Eucalyptus species as hosts of this pathogen but these observations were made in Oceania and not South-East Asia.
Symptoms

Root rot is regarded as a major problem for the cultivation of exotic acacia plantations, especially in Indonesia and Malaysia (Lee 1997, 2001) and information on symptoms, pathology, impact and control are provided in Old et al. (2000). Outbreaks of rot of woody roots and invasion of eucalypt stems by basidiomycete fungi in South-East Asia are few. Etiology at the plantation and individual tree levels, however, are similar for these pathogens, which often have wide host ranges. The brief description provided below is mainly based on experience with other host/pathogen combinations.

Root rot disease is characterised by expanding patches of dead and dying trees with the most severely affected trees at the centre and trees with early symptoms at the periphery. The foliage of affected trees usually becomes paler green and dull in appearance with reduced leaf size and eventual leaf shedding. Crown dieback commences with flattening of the normally conical apex characteristic of rapidly growing Eucalyptus, and death of fine branches. In common with other root diseases, partial crown recovery through the formation of epicormic shoots may be observed, but this will be short-lived and death eventually follows. Gaps are created in the stand and the combination of gaps and root rot predisposes trees to wind-throw.

Root rots of forest trees and plantations can be partly distinguished based on the appearance, especially colour, of infected roots and the fungal structures associated with them. These are: red root disease (associated with Ganoderma infection), brown root rot (caused by P. noxius) and the less common white root rot.

Roots affected by red root rot are covered by a reddish-brown fungal crust, visible after washing clean of adhering soil. A white mottling pattern is evident on the underside of the infected bark. Although many Ganoderma spp., especially when associated with heart rot of large standing live or dead trees form conspicuous fruiting bodies (Figs 89–91), these may be absent from disease foci in fast-grown short-rotation plantations. In other instances fruiting bodies can be found on dead or dying trees near the centre of patches of diseased trees.

Brown root rot is described in detail by Ivory (1996), and this account applies to pathogenic infections of all hosts of P. noxius. The most characteristic sign is a thick brown mycelial mantle on the surface of woody roots that often extends to the above-ground stem (Fig. 92). These hyphal mats exude sticky fluid which causes soil particles to adhere. The fungus invades the main stem where it causes a white pocket rot with a characteristic honeycomb appearance (Fig. 93). Although fruiting bodies of Phellinus spp. are common on large standing or fallen trees in forests, they are rarely found in exotic plantations.

White root rot caused by Rigidoporus lignosus (Klotzsch) Imazeki is a very important disease of plantation crops in tropical South-East Asia, including rubber, oil palm and other crops (Nandris et al. 1987). Although a Rigidoporus species has been found to be an important decay fungus of mining timbers including E. tereticornis (Sharma et al. 1988), white root rot does not appear to have been formally reported in eucalypts plantations.
Pathology

Root rot problems in exotic plantations typically occur on sites recently cleared of natural or secondary native forest. The causal fungi rarely cause widespread damage in native forests, disease patches remaining small. Population studies of infected stands have shown that each patch is colonised by a unique clonal genotype (Ivory 1996). Vegetative spread appears to be limited by natural equilibria that are not well understood. Clearing of sites for plantation establishment leaves large stumps and woody root masses that subsequently act as inoculum sources for many years. From these food bases, the root pathogens spread into the newly established plantations and initiate disease foci, which can continue to enlarge throughout the first rotation and possibly into successive rotations. The degree of damage will depend on the susceptibility of the exotic plantation species and the build-up and persistence of the inoculum base in the woody residues and infected trees.

Impacts

The apparently low incidence and minor importance of these pathogens of woody roots in eucalypt plantations in South-East Asia is probably related to the common practice of establishing such plantations on sites which were cleared of native forest many years ago. This situation could change, for example if eucalypts are planted on former rubber, tea, coffee or cacao plantation sites, in response to market trends or to diversify income of rural populations. Under such circumstances red, brown and white root rots could emerge as significant problems for Eucalyptus cultivation. These fungi, in common with many related basidiomycetes, have the capacity to invade the heartwood of standing trees and cause decay. As eucalypts are primarily grown, in South-East Asia, on very short rotations, heart rot is not currently a significant issue. If in future Eucalyptus is grown for structural uses, then wood decay in standing trees could become important. Measures would then be necessary to achieve the necessary product quality by minimising access of decay fungi to stems through wounds.

Control

No control measures are appropriate as these diseases are seldom reported in Eucalyptus plantations. If problems emerge in the future responses should be modelled on experience gained with exotic acacia plantations (Old et al. 2000).

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Figure 89. *Ganoderma* sp. growing on infected stem of *Acacia auriculiformis*, Sri Lanka

Figure 90. *Ganoderma* fruiting bodies on rainforest tree species, Indonesia

Figure 91. *Ganoderma* sp. on rainforest tree species, Australia

Figure 92. *Phellinus noxius* with spreading brown mycelium which gives the disease the name 'brown root rot'. Photo S.S. Lee.

Figure 93. Stem shown in Fig. 92 with bark removed to show honeycomb appearance of white pocket stem rot. Photo S.S. Lee.
Nursery diseases

Healthy planting stock is a necessary requirement for the success of forest plantations, regardless of the species being grown. The design, construction, operation and maintenance of *Eucalyptus* nurseries in South-East Asia is rapidly changing in pace with the increase in plantation area and the adoption of new technologies, especially clonal forestry. Nursery techniques include simple operations using plastic bags containing seedlings in non-sterile soil, placed on the ground (Fig. 94); facilities with selected trees providing large numbers of cuttings propagated on a large scale (Fig. 95); and enterprises where plantlets, grown under sterile conditions from tissue-cultured mother plants, are propagated. It is expected that companies in South-East Asia will invest in large-scale, highly sophisticated propagation facilities with rigorous control of growth conditions such as are found in Brazil and South Africa (Fig. 96).

It is beyond the scope of this manual to provide detailed recommendations for maintaining *Eucalyptus* seedlings or clonal plantlets in a healthy condition prior to planting out. Comprehensive information on nursery practice in developing countries can be found in Quayle and Gunn (1998) and chapters on nursery diseases and their management can be found in Keane *et al.* (2000). Instead a few general principles will be stated and the main characteristics of commonly-encountered problems, namely damping-off, seedling blights, powdery mildew and their causal pathogens, will be discussed. Although some diseases are primarily associated with propagation of eucalypts, other pathogens span boundaries between nurseries and plantations. Several of these, including *Cylindrocladium* spp., *Phaeophleospora* spp. and *Puccinia psidii*, are dealt with elsewhere in this manual.

Nurseries, from the most basic to the most sophisticated, grow eucalypt seedlings in containers including polythene bags, single plastic tubes or arrays of tubes, which can be moved as single units. The key feature is that the growing medium surrounding the plant roots is separated from that of neighbouring plants. Provided that the growth medium is initially free from soil-borne pathogens, such containers make it possible to ensure that the impact of any chance contamination can be limited to one or a few individual plants. It is essential to have effective hygiene such as sterilisation of tubes, trays or other equipment that may have been contaminated during prior use.

Incubation of plastic bag containers or tubes on bare ground invites contamination with soil-borne organisms and may impede drainage, which inhibits root growth and favours some pathogens. Raising containers above the ground on benches, preferably constructed of sturdy mesh, is good practice and will greatly reduce these risks. If benches are unavailable, containers should be set on beds of freely draining stone chips or gravel, and opportunities taken to replace or partially sterilise this matrix on a systematic basis to reduce the build-up of pathogens.

Airborne contamination of seedlings by pathogenic fungi occurs in even the best-managed nursery. The regular watering and high humidity required for rapid early growth of seedlings, cuttings or tissue-cultured plantlets provide ideal conditions for fungal proliferation. The nursery
manager’s skills and experience are needed to maintain the balance between these conflicting influences, and to identify the time when any application of fungicide is warranted. Other hygiene measures include ensuring that water supplies are free from water-borne pathogens, and reduction of sources of airborne spores, for example by efficient disposal of diseased or otherwise discarded plant material.

*Eucalyptus* cuttings are subject to attack by many of the same pathogens that attack seedlings, with the additional problem of maintaining the clonal hedges or other forms of foundation stock in a healthy condition. As propagation inevitably includes wounding of mother plants, strict hygiene is essential when collecting shoots, including cleaning and surface sterilisation of cutting tools. The maintenance of clonal hedges in a juvenile condition can prolong their susceptibility to pathogens, e.g. *Puccinia psidii*. Infection of clone banks by pathogens can reduce their viability and also provides a means for disseminating pathogens to non-infested areas via contaminated planting stock.

**References**


Figure 94. Nursery south of Hue City, central Vietnam. Seedlings being grown in polythene bags placed on the ground.

Figure 95. Clonal *Eucalyptus camaldulensis* being raised on a large scale in western Thailand.

Figure 96. Advanced tree propagation facility in Sao Paulo State, Brazil. Clonal cuttings being grown under conditions of controlled temperature and humidity.
Damping-off

Disease
Damping-off

Causal organisms
The organisms most commonly associated with damping-off in eucalypt nurseries are *Botrytis cinerea* Pers., *Cylindrocladium* spp., *Pythium* spp., *Phytophthora* spp., and *Rhizoctonia solani* Kühn.

Host range
Damping-off affects many host species including *Eucalyptus* spp. and is caused by a number of different pathogens sharing the capacity to exist in soils or other media in which seedlings and cuttings are grown. The pathogens can attack seedlings pre- or post-emergence and their impact is greatly heightened by poor nursery practice, especially lack of hygiene and over-watering. All *Eucalyptus* species are potential hosts to damping-off fungi, although susceptibility undoubtedly varies. For example, Podger and Batini (1971), testing seedlings for susceptibility to *P. cinnamomi*, demonstrated that members of the subgenus *Monocalyptus* were generally more susceptible to disease than those classified in the subgenus *Symphyomyrtus*.

Known distribution
Damping-off probably occurs wherever eucalypts are nursery-grown on a large scale. Published information from tropical and subtropical South-East Asia on causal organisms and their etiology is limited, with the exception of the Philippines (Kobayashi and De Guzman 1988, De Guzman et al. 1991). Most reports from these latitudes emanate from India (Sharma et al. 1984, Sharma and Mohanan 1991), Australia (Brown and Wylie 1991) and Brazil (Ferreira and Muchovej 1991). The few published reports, however, indicate that the pathogens listed above are also responsible for damping-off of eucalypts in Indonesia, Papua New Guinea (Arentz 1991), Thailand (Pongpanich 2002) and Vietnam (Mao 1996).

Symptoms
Damping-off is often separated into pre- and post-emergence phases although the same groups of fungi are usually responsible. The pre-emergence phase results in poor seedling emergence due to killing of the seed before germination or by invasion of seedling radicles and hypocotyls. The post-emergence phase occurs soon after seedling emergence, when succulent hypocotyls and stem tissues are not lignified and are especially vulnerable. Stems become water soaked due to invasion of the tissues and cell maceration, and the plants fall over. This sequence of events typifies infection by *Pythium* species, which are commonly but not exclusively responsible for the disease. Damping-off in its broadest sense includes disease of very young, non-lignified seedlings associated with invasion by any of the above-listed pathogens. *Rhizoctonia* and *Cylindrocladium* and *Botrytis* species are especially notable,
due in part to their ready identification to genus by non-specialists, compared to the more difficult *Pythium* and *Phytophthora* species.

**Pathology**

Damping-off is the most common disease of forest nurseries (Brown and Ferreira 2000). The causal pathogens are soil-borne and are able to grow or survive as dormant propagules in soil, compost and other nursery potting media. These fungi are commonly endemic in nurseries and management is aimed to minimise their effects as far as possible. Seedlings rapidly become resistant to damping-off because of secondary thickening and lignification of stem tissue, but these same fungi can attack either roots or aerial parts and cause seedling blights. *Cylindrocladium* spp. are of particular importance in the tropics and subtropics as seedling blight and leaf blight pathogens. Plants remain susceptible to these latter fungi throughout their time in the nursery and even after out-planting is completed.

Excessive moisture in growing media due to over-watering, high humidity, high seedling density and high organic content of growth media are the main factors contributing to initiation and spread of damping-off by *Pythium* spp. *Rhizoctonia* spp. are less demanding with regard to soil moisture conditions and can cause disease under a broader range of environmental regimes (Vaartaja and Morgan 1961). *Phytophthora* spp., especially *P. cryptogea* and *P. cinnamomi*, are major nursery pathogens in Australasia (Figs 97, 98) and are also associated with tree deaths in plantations and native forests. Old and Dudzinski (2000), however, found few significant reports of nursery diseases attributed to this genus in Asia. An exception is Papua New Guinea, where Arentz (1991) considered *Phytophthora* spp. to be the most serious pathogens in nurseries.

**Impacts**

Damping-off becomes apparent as irregular patches of dead and dying seedlings, either in seedling trays or arrays of plants pricked out or sown directly into containers. In well-managed, hygienic nurseries, the disease is almost absent. Under conducive environmental conditions, however, the disease can be very serious resulting in high mortality, economic loss and disruption to planting programmes. Once the infection starts, it can spread very quickly and kill a large number of seedlings within a few days.

Vegetative propagation from cuttings of tissue-cultured plantlets is increasingly important in plantation forestry. The term ‘damping-off’ is not strictly applicable to cuttings, as the stem tissue is somewhat lignified. Nevertheless, similar suites of fungal pathogens thrive in clonal nurseries, and shoot blights caused by *Botrytis cinerea*, *Cylindrocladium* spp. and *Rhizoctonia solani* are major problems.

**Control and management**

Damping-off can be managed effectively by following appropriate nursery practices (Brown 2000). Ferreira and Muchovej (1991) provide a detailed account of a seedling management system developed in Brazil which reliably controls damping-off and shoot blight. The main features are:

1. use of pathogen-free seed;
2. direct sowing into conical plastic tubes, containing pathogen-free growing media, which are suspended in racks about 1 m above the ground;
3. thinning seedlings to one per tube as soon as possible (before 7 cm in height);
4. use of pathogen-free water;
5. culling seedlings for uniform height with removal of diseased or dead seedlings;
6. when such cultural controls fail to prevent disease, fungicide sprays are applied at
intervals of 3-4 days over 14 days, using paired combinations of thiabendazole, cap-
tan, benomyl and thiram.

When chemical treatment becomes necessary to control outbreaks of damping-off, drenching
with fungicide in place of normal watering has been found to be very effective. An alternative
is the use of foliar sprays, the choice of application method being determined by the
selected chemical and available facilities. Sharma and Mohanan (1991) investigated a wide
range of fungicide combinations for nursery diseases including damping-off and seedling
blights. Recommended treatments included combinations of captan, carbendazim, copper
oxychloride and quintozene. As the optimum fungicide treatment depends partly on the
causal agent, Brown (2000) has listed chemicals suitable for the control of a wide range of
fungal pathogens. After fungicidal treatment, control of watering to prevent excessive soil
moisture helps to check further spread of the disease.

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Figure 97. Asexual fruiting structure (sporangium) of *Phytophthora cinnamomi* which discharges many small motile spores with the ability to spread in irrigation water or in well-watered soil, bar = 20 µm

Figure 98. Sexual spore of *P. cinnamomi*, thick-walled for prolonged survival and with the potential for sexual recombination, although this may be a rare event, bar = 13 µm
Web blight

Disease
Web blight

Causal organism
*Rhizoctonia solani* Kühn, teleomorph *Thanatephorus cucumeris* (Frank) Donk. (Mordue 1974) is the cause of a particularly damaging seedling blight occurring primarily in humid regions of the tropics. The genus *Rhizoctonia* represents a morphological group, being the mycelial state of several basiomycete fungal genera such as *Thanatephorus* and *Ceratobasidium*. *Rhizoctonia* spp. are characterised by sterile mycelia with rather wide hyphae (Fig. 99) and wide angled branching. The lateral branches are narrowed and septa occur near the junctions with the main axis of the hyphae (Fig. 100). Isolates identified as *R. solani* are pathogenic to plants, have brown or yellow-pigmented hyphae and often form discrete rounded aggregations of hyphae known as sclerotia. The teleomorphs of individual isolates are often not known and are difficult to produce on artificial media.

Host range
Web blight occurs on a wide range of woody and non-woody hosts including *Acacia* spp., *Albizia lebbek*, *Azadirachta indica*, *Eucalyptus* spp., *Paraserianthes falcataria*, *Melia azedarach*, *Ceiba pentandra*, *Lagerstroemia speciosa*, *Cupaniopsis anacardioides* and species of bamboo.

Known distribution
Although *Rhizoctonia* is distributed worldwide in temperate and tropical regions and is a damaging nursery pathogen, the disease known as web blight is less commonly reported than damping-off and seedling root rot. Most reports of web blight over the last few decades are from southern India (Sharma *et al.* 1984, Sharma and Sankaran 1991, Mohanan 1996) on *Eucalyptus*, *P. falcataria*, *A. indica* and bamboo. The geographical distribution may relate to the high temperatures and humidity of the humid tropics being especially conducive to the extensive proliferation of aerial mycelium characteristic of web blight.

There are reports of severe damage to *Melia azedarach*, *A. indica* and *A. lebbek* and seedlings of several other tree species in Assam in northern India (Mehrotra 1989 a,b). Other reports of web blight are from Florida (McMillan *et al.* 1994) on *C. anacardioides*, from Virginia on woody ornamental plants (Lambe 1982) and from Japan on *Cupressus macrocarpa* (Hoshi *et al.* 1995). Kobayashi and Oniki (1993) reported *R. solani* causing web blight in Indonesia on chrysanthemum, geranium and *Mentha* spp.

Symptoms
The disease is characterised by the growth of aerial mycelium of *R. solani* which proliferates from infested soil or other nursery growing media, to attack the stems, cotyledons and
young leaves of densely spaced seedlings. The strands of mycelium are visible to the naked eye or through a hand lens as webs of hyphae, giving the disease its name. Light brown irregularly-shaped sclerotia form on the mycelial web. Infected seedlings develop water-soaked lesions, wilt and die. If appropriate control measures are not adopted the disease spreads rapidly to adjacent healthy seedlings.

**Pathology and impacts**

*Rhizoctonia solani* is able to grow as a saprophyte in soil or compost. The sclerotia described above are resistant to biodegradation and allow the fungus to survive in the absence of host plants. The pathogen will commonly be present in non-sterile soil or nursery media without causing significant disease. Excessive moisture due to over-watering and shade, however, coupled with high seedling density and high organic content of growth media, create an environment conducive to web blight and epidemics can occur. Re-use of plastic pots or tubes without sterilisation is not recommended as the fungus can survive on contaminated containers. Impacts on seedling production can be severe, as the disease spreads rapidly.

**Control and management**

Web blight is more likely to be a problem where seed is sown directly into mother beds or germination trays rather than in nursery operations using plastic tubes or poly-pots. In the event of an outbreak, chemical treatment can become necessary for control. Drenching with carbendazim, or Terraclor® (quintozene) applied in place of normal watering, is reported to be effective (Mohanan 1996, Sankaran *et al*. 1996) After treatment, control of watering to prevent excessive soil moisture helps to check further spread of the disease.

**References**


Figure 99. Stout hyphae of *Rhizoctonia solani*, the cause of web blight, showing characteristic branching and pigmentation, bar = 45 µm

Figure 100. Detail of hypha showing constriction of the branch at the point of right-angle branching, bar = 25 µm
Powdery mildew

Disease
Powdery mildew

Causal organisms
Several species of genera *Erysiphe* and *Sphaerotheca* belonging to the family Erysiphaceae of the order Erysiphales are reported to cause powdery mildews of various eucalypt species.

Due to the absence of the teleomorph stage, powdery mildews on *Eucalyptus* are commonly recorded in various parts of the world as *Oidium* spp., the asexual stages of *Erysiphe* and *Sphaerotheca*. This problem of identification may be resolved in future by DNA sequencing of anamorph collections. Records in which teleomorphs have been associated with powdery mildew on *Eucalyptus* spp. are listed below.

*Erysiphe cichoracearum* DC. and *Erysiphe polyphaga* Hammarl [= *Erysiphe orontii* Castagne] were found in New Zealand (Boesewinkel 1979, 1981). The former species was also recorded in UK (Gibson 1975) and USA (Gardner and Yarwood 1974, Matheron and Matejka 1992).

*Sphaerotheca aphanis* (Wallr.) U.Braun [= *S. alchemillae* (Grev.) L.Junell; *Erysiphe alchemillae* Grev.] was reported in New Zealand (Boesewinkel 1979, 1981), *S. macularis* (Wallr.) Jacz. in Germany (Brandenburger 1961) and *S. pannosa* (Wallr.) Lév., the teleomorph of *Oidium eucalypti* Rostr., in Argentina, Australia, Brazil, Denmark, Italy, New Zealand, Poland, Portugal, South Africa and United Kingdom (Grasso 1948, Glasscock and Rosser 1957, Spaulding 1961, Yarwood and Gardner 1972, Gibson 1975, Boesewinkel 1981, Crous et al. 1989).

Host range
Powdery mildews have been commonly found on many species of *Eucalyptus*:

*Erysiphe cichoracearum* on *E. calycogona*, *E. camaldulensis*, *E. campaspe*, *E. cladocalyx*, *E. globulus*, *E. leucoxylon*, *E. leucoxylon var. rosea*, *E. microcarpa*, *E. pauciflora*, *E. pauciflora* ssp. niphophila, *E. platypus*, *E. polyanthemos*, *E. populnea*, *E. porosa*, *E. pulverulenta*, *E. tetragona*, *E. viminalis* and *E. viridis*;

*Erysiphe polyphaga* on *E. crebra* and *E. moluccana*;

*Sphaerotheca aphanis* on *E. albens*, *E. cinerea*, *E. crebra*, *E. diversicolor*, *E. grossa*, *E. megacarpa*, *E. nutans*, *E. paniculata*, *E. tereticornis* and *E. torquata*;

*Sphaerotheca macularis* on *E. algeriensis*, *C. citriodora*, *E. cornuta*, *E. diversicolor* and *E. gomphocephala*;

*Sphaerotheca pannosa* on *E. albens*, *E. camaldulensis*, *E. gunnii*, *E. moluccana*, *E. nitens*, *E. perriniana* and *E. tereticornis*;

**Known distribution**

The disease has been reported on *Eucalyptus* in Argentina, Australia, Brazil, Denmark, Germany, India, Italy, New Zealand, Poland, Portugal, South Africa, UK and USA (Sankaran *et al.* 1995). Formal reports of powdery mildew on *Eucalyptus* spp. in nurseries in South-East Asia are few although Kobayashi (2001) reported that *Oidium* is often prevalent on *Eucalyptus* seedlings in nurseries in the region.

**Symptoms**

*Sphaerotheca pannosa*, known as the ‘rose mildew’ is one of the most common and destructive species causing powdery mildews of a variety of plant hosts. It attacks leaves and young shoots of *Eucalyptus*, producing a thick layer of densely inter-woven white mycelium (Fig. 101) on the surface of leaves and shoots (Crous *et al.* 1989), sometimes causing spotting and malformation of older growth (Gibson 1975, Boesewinkel 1981).

**Pathology**

Spores of *Oidium* spp. germinate on the surfaces of the leaves producing germ tubes that penetrate the walls of the leaf epidermal cells. The fungus forms absorbing structures known as haustoria through which it obtains nourishment from the host cells. The fungus proliferates over the leaf surfaces, producing abundant conidia (Fig. 102), which result in the powdery white appearance from which the name of the disease is derived. The spores, which are produced successively on specialised hyphae arising from the superficial mycelium (Figs 102, 103), are dispersed by wind to other susceptible hosts, initiating new infections. When the perfect stages are present, small black dots (cleistothecia) are observed immersed in white mycelium layers. The cleistothecia of *Erysiphe* and *Sphaerotheca* have hairlike, unbranched flexuous appendages and contain 1–several asci with eight ascospores.

**Impacts**

Powdery mildews of *Eucalyptus* rarely occur in plantations, although are commonly encountered in glasshouses and nurseries where they primarily cause leaf distortion and poor growth of seedlings. In Arizona, USA, *Erysiphe cichoracearum* was observed only on young plants of *E. camaldulensis*, *E. cladocalyx*, *E. leucoxylon*, *E. polyanthemos* and *E. viminalis* in greenhouse conditions (Matheron and Matejka 1992). In Tasmania, *Oidium* sp. causes severe nursery problems of susceptible species, such as *E. nitens* and *E. globulus* ssp. *globulus* (Wardlaw and Phillips 1990). In India, powdery mildew causes distortion, necrosis and ultimately leaf fall on eucalypt hybrid seedlings (Sehgal *et al.* 1975).

**Control and management**

Early recognition and prompt removal of infected plants are important in preventing disease spread and fallen leaves should be destroyed to reduce inoculum potential.

Chemical treatments are seldom necessary, but sometimes the control of powdery mildew relies primarily on the use of fungicides (Wardlaw and Phillips 1990) and can be effected by spraying with a fungicide, such as benomyl, chlorothalonil, triadimefon, maneb or zineb (Sehgal *et al.* 1975).
Biological control is an alternative means of management of foliar diseases including powdery mildews, especially in greenhouses. Commercial biocontrol products containing *Trichoderma harzianum* T39, *Ampelomyces quisqualis*, *Bacillus* and *Ulocladium* have been developed for greenhouse crops (Paulitz and Belanger 2001). Foliar sprays with other substances such as JMS Stylet-oil for cucurbit powdery mildew (McGrath and Shishkoff 2000) and nonswelling chlorite mica clay for cucumber powdery mildew (Ehret *et al.* 2001) have been proved to significantly reduce the severity of the disease in greenhouses.

**References**


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Figure 101. Powdery mildew on a *Eucalyptus* seedling grown in the greenhouse; the mealy-white appearance is due to production of vast numbers of conidia

Figure 102. Barrel-shaped conidia of powdery mildew, bar = 10 µm
Figure 103. *Oidium* sp.: A. conidia in chain; B. conidia. Bar = 15 µm for A and B.
Eucalyptus rust

Disease
Eucalyptus rust, guava rust

Causal organism
*Puccinia psidii* Winter

Known distribution
South America, mainly east of the Andes, including northern-most Argentina, Uruguay, Paraguay, Brazil, Venezuela, Ecuador and Columbia. Also present in Central America and the Caribbean, including Cuba, Dominican Republic, Jamaica, Puerto Rico, Trinidad and Florida (Coutinho *et al.* 1998). This fungus is not known to occur in Africa, Australasia, Oceania or Asia and must be regarded as a dangerous exotic pathogen in countries where *Eucalyptus* and other *Myrtaceae* are grown or constitute native vegetation.

Host range
All genera within the family *Myrtaceae* are potentially susceptible to this rust, but information on the host range is incomplete. In addition to *Eucalyptus*, 9 genera and more than 30 species of *Myrtaceae* are recorded hosts of *P. psidii* (Laundon and Waterston 1965, Burnett and Schubert 1985, Ferreira 1989, Coutinho *et al.* 1998). Recent unpublished research by Zauza *et al.* has revealed further hosts of *P. psidii* and has identified resistant species within most genera. Susceptible genera include several which are well represented in Australian native vegetation, e.g. *Angophora*, *Callistemon*, *Corymbia*, *Eucalyptus*, *Kunzea*, *Melaleuca*, *Syzygium* and *Syncarpia*. Of particular importance are plantation and fruit trees including guava, cloves and other *Syzygium* species, *Melaleuca* spp., *Myrciaria jaboticaba* and *Pimenta dioica* (allspice). Evidence of host specialisation exists within the pathogen, so isolates from one host genus may or may not infect other genera within *Myrtaceae* (Ferreira 1989). Old *et al.* (2003) described a disease severity scale for artificially inoculated eucalypt seedlings, developed by Alfenas and coworkers (unpublished). Almost 100 seedlots comprising 30 species of *Eucalyptus* and *Corymbia* have been screened for rust susceptibility. *Eucalyptus grandis* (Queensland), *E. camaldulensis* var. *obtusa* (Northern Territory) and *E. cloeziana* were among the most susceptible, whereas seedlots of *E. alba*, *E. paniculata*, *E. pellita*, *E. tereticornis*, *E. resinifera*, *E. brassiana* and *C. tessellaris* had high proportions (> 70%) of resistant plants.

Symptoms
Egg-yellow uredinia develop on juvenile leaves of host plants (Fig. 104). Older pustules contain both single celled urediniospores and two-celled, stalked teliospores. The rust attacks both foliage and young green shoots on which cankers are formed, with rupture of the epidermis and phloem. The presence of these cankers can cause stem distortion and crowns of badly affected trees become stunted and multi-stemmed (Figs 105, 106). The fungus also
attacks fruits of susceptible species such as guava (Fig. 107), *Myrciaria jaboticaba* (Fig. 108), and *Syzygium jambos*, which are often found in Brazil in gardens in the vicinity of *Eucalyptus* plantations and act as reservoirs of inoculum. On these hosts the fungus fruits prolifically.

**Pathology**

*Puccinia psidii* is an autoecious rust, both sexual and vegetative sporulation occurring on the same host. Urediniospores and teliospores form on the same lesions, but teliospores are rarely found on *Eucalyptus* hosts. The pathology of the rust is well described in Coutinho *et al.* (1998) along with other aspects of the biology of the fungus and epidemiology of disease. Disease spread results from airborne urediniospores alighting on leaf surfaces and germinating during darkness and conditions of leaf wetness at temperatures of 15–25°C. Rust lesions then become visible 2–4 days later. Unusually, for a rust pathogen, penetration of the host epidermis occurs directly rather than through stomata. Penetration of mature leaves is generally unsuccessful and this is consistent with the observation that, on *Eucalyptus*, trees less than two years old are susceptible to disease. Coppice shoots of older trees are, however, vulnerable to infection due to their juvenile foliage.

**Disease impacts**

Disease impacts are difficult to predict across the range of crop species and natural vegetation that is potentially susceptible to attack by this pathogen. For example, in the 1930s an industry in Jamaica based on extracting essential oils from pimento was forced to close only two years after *P. psidii* was first reported (Maclachlan 1938). For *Eucalyptus* in Brazil, impacts are generally small where resistant clones are selected from the *E. grandis* × *E. urophylla* hybrids that are extensively grown as short-rotation crops for pulp and paper mills. A significant proportion of the plantation estate, however, is grown from seed and outbreaks of rust may occur on a scale that needs to be managed. The impacts of rust are particularly important during propagation of cuttings in clonal nurseries, as juvenile foliage can be very susceptible to infection.

**Control and management**

Control can be readily achieved by use of species with a high or moderate level of resistance to rust and by selection within hybrid progeny for resistant clones. This approach has been highly successful in Brazil where rust-resistant selections within pure *E. grandis* and *E. grandis* × *E. urophylla* hybrids have been readily identified. If, for commercial reasons, seedlings are used on a significant scale to establish plantations, clonal seed orchards containing resistant trees have been found to be a viable option (Alfenas personal communication). Seed for planting in known high rust-hazard areas can be collected from trees homozygous for dominant resistant genes, thus ensuring a rust-resistant plantation. Experience from agriculture suggests that it is unwise to rely on a single, or few major genes for resistance to rust diseases. Fungicides are available that will control rust effectively (Coutinho *et al.* 1998). These include the protectant fungicide mancozeb and the systemic fungicides triadimenol and triforine. Fungicide application is most appropriate, however, in nurseries and clonal hedge plantings, and is not generally practised in plantations. In most instances susceptible planting stock is best discarded in favour of resistant trees.
Incursion pathways

*Puccinia psidii* is recognised as an extremely dangerous pathogen as it has originated in a continent where *Eucalyptus* is grown widely but is not indigenous (Ciesla et al. 1996). *Eucalyptus* spp. are widely planted as exotics in countries, including South-East Asia, where the climates should be suitable for rust epidemics to occur. Australia is at particular risk as *Eucalyptus* and other Myrtaceae dominate much of the native vegetation. In several regions of South-East Asia, in addition to Myrtaceae in native vegetation, there are many important fruit, oil and spice crops which would be susceptible to disease, e.g. *Psidium guajava*, *Melaleuca cajuputi* and *Syzygium aromaticum*. The most dangerous pathway for spread of the pathogen to new regions of the world is by germplasm, including rooted cuttings and other vegetatively propagated trees, seed and pollen. Recent research using a highly sensitive PCR-based DNA diagnostic method (Langrell et al. 2003) has detected rust spores in many seed and pollen samples from commercial sources in Brazil. Spores were also detected on clothing, footwear, camera bags and other field equipment exposed to contamination from rust-affected plantations.

References


Figure 104. Egg-yellow spores of *Puccinia psidii* produced on a juvenile leaf of *Eucalyptus grandis*

Figure 105. *E. grandis* heavily infected by rust; note stunting of foliage

Figure 106. Detail of infected stems showing stem lesions and shrivelled leaves

Figure 107. Guava fruit infected by *P. psidii*

Figure 108. *Myrciaria jaboticaba* infected by *P. psidii*
Glossary of terms

**Acervulus** (plural acervuli), a saucer-shaped fruiting structure embedded in host tissue and bearing conidia

**Allantoid**, slightly curved with rounded ends (sausage shaped)

**Anamorph**, the asexual (imperfect) form of a fungus

**Annular**, ring-like

**Anthracnose**, a group of plant diseases characterised by the formation of discrete necrotic spots on young shoots and foliage

**Ascospore**, a spore produced inside a sac-shaped **ascus** resulting from meiotic cell division, characteristic of fungi classified in the **Ascomycetes**

**Autoecious** (applied to rusts), completing life cycle on one host

**Basidiospore**, a propagative spore formed after meiosis by a basidiomycete

**Capitate**, having a well-formed head

**Chlamydospore**, an asexual spore produced primarily as a survival structure (rather than for dissemination), originating from a pre-existing cell by the formation of a thickened inner cell wall layer

**Clamp connection**, a hyphal swelling that is found between adjacent cells in many Basidiomycete fungi. The connection forms during cell division and is a way of maintaining the ratio of nuclei of differing genetic origin within fungal mycelia

**Cleistothecium**, a closed fruiting body without a defined opening, containing ascospores formed by powdery mildews

**Conidium** (plural conidia), a specialised non motile asexual spore produced on a **conidiophore**, typically for dissemination purposes

**Conidioma**, (plural conidiomata), a specialised conidium-bearing structure now used widely for all such fruiting structures, e.g. acervulus, pycnidium

**Etiology**, the science of the causes of diseases

**Hymenium**, the spore-bearing layer of a basidiomycete fruiting body

**Kino**, polyphenolic substances produced by eucalypts in response to cell damage
Hyphopodium, a short hyphal branch of one or two cells in extent

Microconidium, the smaller conidium produced by a fungus that also forms macroconidia

Necrosis, the death of plant cells, often resulting in tissue becoming dark in colour

Perithecium, subglobose or flask-shaped fruiting structure, containing asci and ascospores, formed by many ascomycete fungi

Pseudothecium, a stromatic fruiting body formed by some ascomycete fungi. Asci are formed within chambers lacking distinct walls (locules)

Phialide, a specialised cell, often borne on a conidiophore, which generates a succession of conidia in basipetal succession without any increase in its own length

Pycnidium (plural pycnidia), a flask-shaped fruiting structure embedded in host tissue containing conidiophores and conidia

Rhizomorph, a root-like aggregation of fungal hyphae with a well defined apical meristem, formed by some basidiomycete tree pathogens and decomposer fungi for spreading through soils and along root surfaces

Sclerotium (plural sclerotia), a firm mass of fungal hyphae, often round in shape, not containing spores but often able to survive in the absence of host tissue and germinate when conditions are favourable for infection

Septum (plural septa), cross walls present in fungal hyphae and spores of many species

Seta (plural setae), sterile hair-like hyphae which often project from spore bearing structures and are useful in classification

Sporangium (plural sporangia), a structure formed by many algae and fungi within which spores are formed asexually by nuclear division and protoplasmic cleavage of the contents. The spores may be motile (zoospores) or non-motile. Sporangia may be borne on sporangiophores

Stroma (plural stromata), a mass of vegetative stromatic hyphae within or on which spores of fruiting bodies are formed

Teleomorph, the sexual (perfect) form of a fungus

Teliospore, a resting or over-wintering rust spore

Thallus, the vegetative body of fungi

Urediniospore, repeating, vegetative rust spores formed rapidly during epidemic disease

Vesicle, a sac-shaped structure which may be the swollen apex of a conidiophore or a sterile hypha as in Cylindrocladium
The Center for International Forestry Research (CIFOR) was established in 1993 as part of the Consultative Group on International Agricultural Research (CGIAR) in response to global concerns about the social, environmental and economic consequences of forest loss and degradation. CIFOR research produces knowledge and methods needed to improve the well-being of forest-dependent people and to help tropical countries manage their forests wisely for sustained benefits. This research is done in more than two dozen countries, in partnership with numerous partners. Since it was founded, CIFOR has also played a central role in influencing global and national forestry policies.

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This manual represents an outcome of 10 years of work by the authors in South-East Asia. It focuses on the main diseases and pathogens of eucalypts found in the region and will assist in disease diagnosis. Summaries of published work on the pathogens and recommendations for their control are provided.