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REVIEW

Quambalaria spp.: Emerging Tree Pathogens of Concern

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Abstract

Purpose of Review *Quambalaria* spp. are fungal pathogens originating from Australian eucalypt species that are becoming increasingly important globally. For example, *Quambalaria eucalypti,* which was once considered a minor pathogen, now poses a significant threat to plantation productivity and forest health across continents. Their spread has been made possible through the expansion of eucalypt plantation forestry outside Australia, the movement of plant material and the increase in travel and trade, which facilitates the intercontinental movement of pathogens. This review summarises current knowledge regarding the taxonomy, distribution, life cycle and ecological and commercial impacts of *Quambalaria* spp. and some considerations for their control.

Recent Findings Recent studies have discovered new species of *Quambalaria* and there is emerging evidence of host shifts to other genera in the Myrtaceae. Outbreaks in Brazil, China, Indonesia and South Africa illustrate the rapid spread of *Quambalaria* spp. to new plantation environments. These pathogens threaten eucalypt plantations worldwide because clonal propagation reduces tree diversity and the trade of plant material facilitates their spread. Evidence of sexual reproduction has been identified in one species, *Quambalaria pitereka*, alluding to a high evolutionary potential. Although new molecular diagnostics and resistance screening tools are emerging, their use in the control of these pathogens remains limited.

Summary *Quambalaria* spp. are important tree pathogens that are increasing their impact beyond their ancestral home in Australia. Their impact on plantation forestry and native forests underscores the broader vulnerabilities of forest ecosystems. Mitigating their impact will require integrated approaches that combine resistance breeding, enhanced surveillance, stringent quarantine measures, integrated disease management and changes to silviculture. Further research into host range, infection biology and pathogen evolution and the development of effective disease control is essential to safeguard global forests and ensure sustainable plantation forestry.

Keywords Basidiomycetes · Cankers · Eucalypts · Forest biosecurity · Microstromatales · Quambalariaceae · Shoot and bud blight

Introduction

Most commercially planted eucalypts, including species of *Eucalyptus* and *Corymbia*, are native to Australia and numerous species are cultivated globally due to their adaptation to many different environments, rapid growth, water use efficiency and valuable timber and pulp. Eucalypts form the backbone of the hardwood forestry industry in the Global South, contributing significantly to the economy and rural employment [1, 2]. However, the sustainability of eucalypt plantations is increasingly threatened by emerging pests and pathogens, including numerous genera of fungi [3]. One such genus is *Quambalaria,* which includes species responsible for diseases expressed as cankers, leaf spots, shoot and bud blight [4]. Currently, *Quambalaria* spp. are recognised as important pathogens due to their expanding distribution, host range and increasing impact on tree health in various environments (Fig. 1; Table 1).

The genus *Quambalaria* was established in 2000 [5] and encompasses several fungal species, including *Q. pitereka*, the type species originally described from shoot blight of *Corymbia eximia* (≡ *Eucalyptus eximia*) from New South

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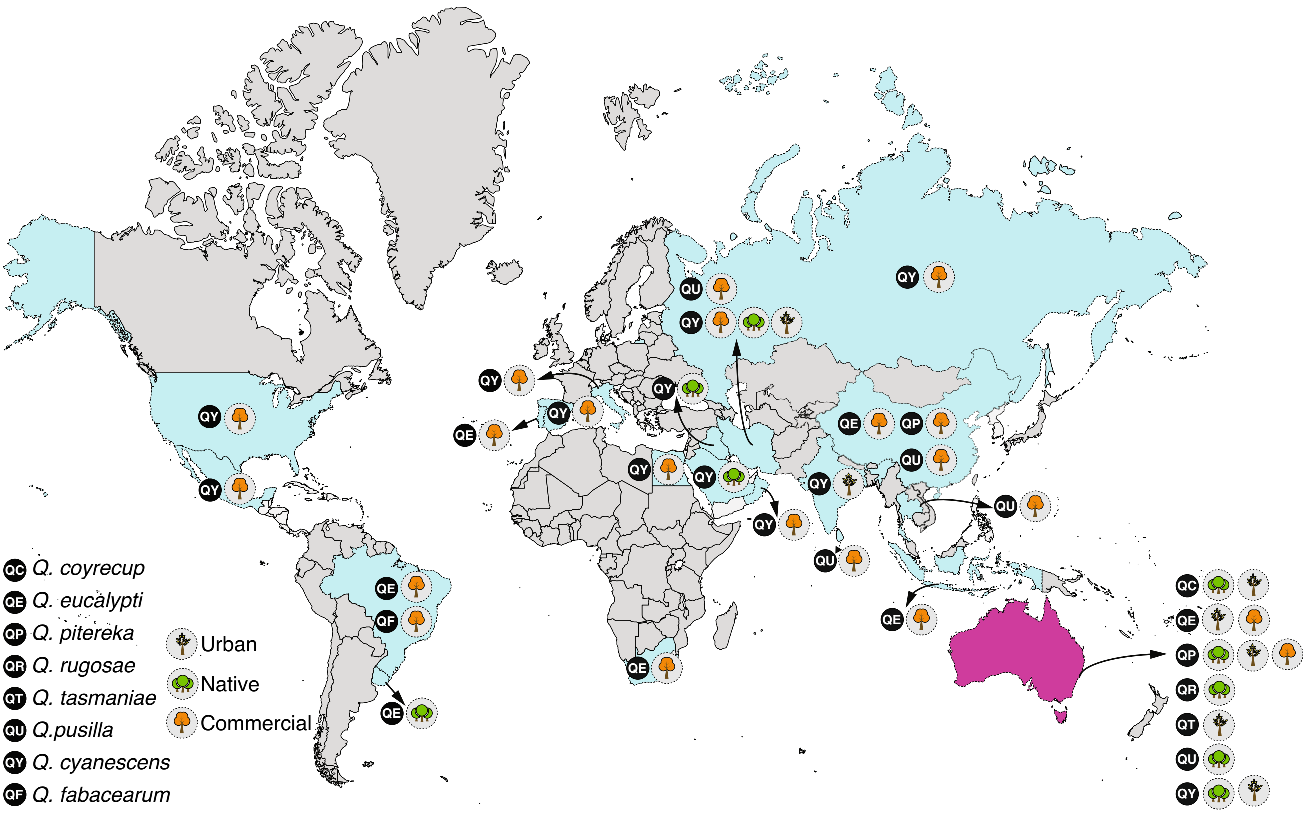


Fig. 1 Global distribution of the eight known *Quambalaria* species across commercial plantations, urban environments and native vegetation (for details see Table 1 and S1). Australia is the centre of diversity, highlighted in pink, with seven species recorded, while other countries show varying combinations of species and habitat types. Commercial plantations refer to managed forestry stands established for the production of forest-based products. Native vegetation refers to detections in natural ecosystems. Urban environments refer to ornamental or planted trees in cities and landscaped areas

Wales, Australia [6], as well as *Q. eucalypti* [7] and *Q. coyrecup* [8]. These species are associated with a variety of disease symptoms, such as shoot and bud blight, leaf spots, dieback and stem cankers. They can infect juvenile and mature trees and reduce the photosynthetic area, impair growth and in severe cases, contribute to tree mortality [9, 10].

The detection and increasing occurrence of *Q. eucalypti* infections in commercially managed *Eucalyptus* plantations have raised concerns within the plantation forestry sector [11–14]. Globally, *Eucalyptus* cultivation has historically responded rapidly to many pest and pathogen threats through effective management changes and breeding programmes [15, 16]. However, the increasing number of outbreaks of diseases caused by *Quambalaria* spp. justifies a more rigorous current pest and pathogen management strategy. This objective is complicated by the relatively limited knowledge of the biology, epidemiology and genetic diversity of *Quambalaria* spp.

This review synthesises the current knowledge on *Quambalaria* spp., covering their taxonomy, biology and global distribution. The impact that these fungi have on natural forests, trees in urban environments, fragmented landscapes, as well as in intensively managed plantations and nurseries, is considered. A comprehensive database is also presented, compiling all known reports of *Quambalaria* spp. The review highlights important gaps in research on the ecology, diagnostics and management of these species, reflecting the limited attention these pathogens have received. By outlining current challenges and priorities, the overall aim is to promote more timely and effective responses to the growing threat of *Quambalaria* spp. to global tree health.

Taxonomy of *Quambalaria*

Taxonomic History

Fungal species in the genus *Quambalaria* have undergone major taxonomic changes over the past decades. In the 1930s, HAJ Pittman, the Western Australian Government plant pathologist, sent specimens of an unknown fungus from *Corymbia ficifolia* to the Imperial Mycological

Table 1 Summary of *Quambalaria* spp., their geographic distribution, associated host species (or substrate) and haplotypes (if available). If no publication record for a geographic location was available, the GenBank accession number is added as a reference. For details, see Table S1

Quambalaria species	Countries	Hosts	Haplotypes	Comments	References
Q. coyrecup	Australia	*Corymbia calophylla* *Corymbia ficifolia* *Corymbia polycarpa*	Unknown	Sporulation observed on canker lesions and symptomatic stem tissue	Paap et al. [8], Pegg et al. [4]
Q. cyanescens	Australia Egypt India Iran Italy Mexico Oman Russia Saudi Arabia Spain USA	*Betula pendula* *Corymbia calophylla* *Corymbia citriodora* *Corymbia ficifolia* *Corymbia variegata* *Citrus limon* *Calotropis procera* *Citrus* sp. *Eucalyptus pauciflora* *Ipomea carnea* *Juglans regia* *Prunus avium* *Prunus cerasus* *Phoenix dactylifera* *Punica granatum* *Pistacia vera* *Quercus brantii* *Vitis vinifera*	Unknown	Widespread and found on symptomatic and asymptomatic plant tissue, associated with shoot and leaf sporulation (often with *Q. pitereka*), canker lesions (with *Q. coyrecup*), discrete cambial lesions, grapevine and walnut diseases (with other microbes) and occurs as a low-abundance endophyte or phyllosphere component	Paap et al. [8], Pegg et al. [4], de Hoog, de Vries [90], Moubasher et al. [91], Padhi, Tayung [92], Meshram et al. [93], Narmani, Arzanlou [94], Vahedi-Darmiyan et al. [95], Mahdizadeh et al. [44], Kari Dolatabad et al. [96], Hagh-Doust et al. [97], Abdollahi Aghdam, Fotouhifar [98], Lorenzini et al. [99], Faddetta et al. [100], Argüelles-Moyao et al. [101], Al-Nabhani et al. [45], Antropova et al. [102], Najjar et al. [103], Mateo et al. [104], Travadon et al. [105]
Q. eucalypti	Australia Brazil China Indonesia Portugal South Africa Uruguay	*Corymbia torellina* × *C. variegata* *Eucalyptus dunnii* *Eucalyptus globulus* *Eucalyptus grandis* *Eucalyptus grandis* × *E. camaldulensis* *Eucalyptus grandis* × *E. pellita* *Eucalyptus longirostrata* *Eucalyptus microcorys* *Eucalyptus nitens* *Eucalyptus pellita* *Eucalyptus pellita* × *E. brassiana* *Eucalyptus pellita* × *E. grandis* *Eucalyptus saligna* × *E. maidenii* *Eucalyptus urophylla* × *E. grandis* *Myrceugenia glaucescens*	QE1 QE2 QE3 QE4	The pathogen is primarily associated with leaf and shoot blight (usually in nursery and plantation settings), alongside leaf spots linked to insect damage, stem girdling and trunk cankers, with occasional cases isolated from diseased clones or single native trees near plantations	Pegg et al. [4], Faedda, Silva [106], Alfenas et al. [46], Santos et al. [14], Chen et al. [11], Tarigan et al. [13], Bragança et al. [31], Wingfield et al. [7], Roux et al. [12], Pérez et al. [61], Faedda, Silva [106]
Q. fabacearum	Brazil	*Mimosa tenuiflora*	Unknown	The fungus was isolated from asymptomatic *M. tenuiflora* as an endophyte	Bezerra et al. [26]

Table 1 (continued)

Quambalaria species	Countries	Hosts	Haplotypes	Comments	References
Q. pitereka	Australia China	*Corymbia calophylla* *Corymbia citriodora* *Corymbia variegata* *Corymbia exima* *Corymbia ficifolia* *Corymbia henryi* *Corymbia maculata* *Corymbia torelliana* *Corymbia torelliana* × *C. citriodora* *Corymbia torelliana* × *C. variegata* *Corymbia torelliana* × *C. henryi*	QP1 QP2 QP3 QP4 QP5 QP6 QP7 QP8 QP9 QP10	The pathogen is primarily associated with shoot and leaf blight, causing shoot distortion, leaf spots, stem lesions and leaf lesions, particularly affecting seedlings, saplings, juvenile foliage of mature trees and mini-hedges, with sporulation observed on shoots and newly emerged leaves	Paap et al. [8], Pegg et al. [4], Walker, Bertus [6], Zhou et al. [40]
Q. pusilla	Australia China Iran Sri Lanka Thailand	*Eucalyptus tintinnans* *Eucalyptus urophylla* × *E. grandis* *Eucalyptus camaldulensis* *Cocos nucifera*	Unknown	The fungus has been isolated from stem cankers of *Eucalyptus*, although it is not consistently linked to disease development. It has also been recovered from king coconut water in Sri Lanka and from soil samples in Iran	Braun [18], Crous et al. [27], Chen et al. [11], Cheewangkoon et al. [107] Iran (JQ650238) Sri Lanka (MT879594)
Q. rugosae	Australia India	*Eucalyptus rugosa*	Unknown	Associated with leaf spot in Australia	Crous et al. [27]
Q. tasmaniae	Australia	*Eucalyptus* spp.	Unknown	Associated with leaf spots	Crous et al. [27]

Institute at Kew. This fungus was tentatively identified as "*Sporotrichum destructor*". However, the species was not formally described and a Latin diagnosis was not published [17]. For many years, this taxonomic uncertainty made the diagnosis and tracking of the pathogen difficult. Finally, the identity of this fungus was resolved by Paap et al. [8], who used molecular and morphological data, together with EW Mason's unpublished notes at Kew and Pittman's specimens, to describe the new species, *Quambalaria coyrecup.*

Following the first recognition of *S. destructor,* a new shoot disease was observed on *Corymbia maculata* (≡ *Eucalyptus maculata*) seedlings in the Australian state of New South Wales in the 1950s. Walker, Bertus [6] identified the causal agent as *Ramularia pitereka.* In 1987, a similar disease characterised by leaf infections and die-back of actively growing shoots appeared on *Eucalyptus grandis* clonal hedge banks in the KwaZulu-Natal province of South Africa. This led Wingfield et al. [7] to describe a new species, *Sporothrix eucalypti.* Later, Braun [18] re-evaluated the taxonomy of these fungi and transferred *R. pitereka* to the genus *Sporothrix* (Ophiostomatales) based on conidial scar morphology. He also described a third species, *Sporothrix pusilla,* from leaf spots on *Eucalyptus camaldulensis* in Thailand. These three species were distinguished from each other based on morphological characteristics and their specificity to different hosts. The placement of these pathogens with members of the Ophiostomatales was influenced entirely by morphology, but they were biologically different from others in the family that were typically insect-associated Ascomycetes [19].

Earlier research on *Sporothrix* showed clearly that this genus was highly diverse and that some of the species have basidiomycete affiliations [20–22]. For example, Weijman, de Hoog [22] and de Hoog [20] showed that species within *Sporothrix* exhibited varied phylogenetic relationships. Molecular evidence further complicated this situation when Berbee and Taylor [23] used 18S rDNA sequences to show that the type species of *Sporothrix*, *S. schenckii,* belonged to the sexual genus *Ophiostoma.* Other inconsistencies concerning the placement of *Quambalaria* spp. in *Sporothrix* also began to emerge. For example, Simpson [5] demonstrated that isolates of *R. pitereka* were unable to grow in the presence of the antibiotic cycloheximide, which is a defining characteristic of *Sporothrix* spp. residing in the Ophiostomatales [19, 24].

Following the emerging inconsistencies relating to their placement in *Sporothrix,* Simpson [5] established a new genus, *Quambalaria,* to accommodate *S. pitereka, S. eucalypti* and *S. pusilla.* This taxonomic reclassification was based on several distinguishing traits, such as their inability to tolerate cycloheximide, pathogenicity to *Eucalyptus* and *Corymbia,* the presence of dense white conidiophores and the absence of distinct denticles on the conidiogenous cells. Simpson [5] also noted that these fungi had dolipore septa and would more appropriately reside in the Basidiomycetes orders Exobasidiales or Ustilaginales. This was an important discovery and a significant departure from their prior placement in the Ophiostomatacetales and as Ascomycetes.

A breakthrough in the taxonomy of *Quambalaria* emerged from the work of de Beer et al. [25], who used LSU and ITS sequence data to show that *Quambalaria* spp., along with *Fugomyces cyanescens* (≡ *Q. cyanescens*), formed a distinct clade in the Microstromatales (Basidiomycetes). Their study also confirmed the presence of dolipore septa in *Quambalaria* spp., a feature absent in other families of the Microstromatales, such as Microstromataceae and Volvocisporiaceae. Consequently, the new family Quambalariaceae was established to accommodate these species [25].

The genus *Quambalaria* now comprises eight species (https://www.mycobank.org/). These include *Q. coyrecup* [8], *Q. cyanescens* [20], *Q. eucalypti* [7], *Q. fabacearum* [26], *Q. pitereka* [6], *Q. pusilla* [18], *Q. rugosae* and *Q. tasmaniae* [27]. Most of these species are associated with Myrtaceae, but their pathogenic status varies. For well-studied species, such as *Q. pitereka*, *Q. eucalypti* and *Q. coyrecup*, pathogenicity has been confirmed through infection trials. For less well-studied species, such as *Q. fabacearum*, *Q. pusilla*, *Q. rugosae* and *Q. tasmaniae*, association with disease is based on isolation from symptomatic tissues, but experimental proof of pathogenicity is lacking. In contrast, *Q. cyanescens* has been recovered from a wide range of environmental sources, including air, soil, insects and plants and is also recognised as an opportunistic human pathogen.

Taxonomic Re-Evaluation

As part of this review, we revisited the phylogeny of the genus *Quambalaria*. This was motivated because earlier studies, such as those by de Beer et al. [25] and Paap et al. [8], could not include all currently known species, while more recent studies, including Bezerra et al. [26] and Tarigan et al. [13], were constrained by limited taxon sampling. When Crous et al. [27] described *Q. rugosae* and *Q. tasmaniae*, a family-wide phylogeny was constructed. However, their analysis was based solely on ITS sequence data from 104 strains, using a parsimony approach. Since most species of *Quambalaria* have been analysed and reported on in isolation, we have, as part of this review, combined all available data to compile a single phylogeny using a concatenated data set of ITS and LSU rRNA gene sequences. This dataset contains 65 isolates, including multiple representative sequences per species (where available) across all known geographic ranges to capture intraspecific diversity (Table 1; Table S1). Phylogenetic analyses were conducted using the concatenated dataset with maximum likelihood and Bayesian inference approaches (see Supplementary Document for phylogenetic methods).

Our phylogeny (Fig. 2) supports the monophyly of most *Quambalaria* spp. and is consistent with those in previous studies where taxon sampling overlapped. The concatenated trees placed the single isolate of *Q. fabacearum* within the *Q. cyanescens* clade (Fig. 2). This finding is contrary to that of Bezerra et al. [26] but in agreement with Crous et al. [27]. The placement of *Q. rugosae* is not strong as it was based on only a single available isolate (Fig. 2).

Life Cycle of *Quambalaria*

The life cycle of *Quambalaria* spp. is poorly understood due to the ambiguous nature of their spore-producing structures and the fact that they were originally not recognised as Basidiomycetes. These fungi were originally classified based on the morphology of their reproductive structures, assumed to be conidia and conidiophores and thus indicative of asexual reproduction. As a result, the conidia were believed to be the primary infectious propagules [25]. The discovery that these fungi are Basidiomycetes raises questions about the biological nature of their spores, which could, in some cases, be basidiospores and consequently the result of meiosis. The putative sexual life cycle of Q. *pitereka* is thought to resemble that of other pathogenic smut fungi in the Ustilaginomycetes, such as *Ustilago* and *Mycosarcoma* spp., where plasmogamy between compatible haploid hyphae leads to the formation of a pathogenic dikaryon [28]. Although sexual fruiting structures have not been directly observed, *Q. pitereka* is hypothesised to produce basidia with basidiospores on infected host tissue [29]. These spores, visualised using electron microscopy, are thought to be the main reproductive and infectious units for *Q. pitereka,* as illustrated in the hypothesised life cycle (Fig. 3). After dispersal, compatible basidiospores germinate and fuse on young, susceptible tissues, such as expanding shoots, leaves and flower buds, where infection begins through dikaryotic hyphal growth (Fig. 3). Disease symptoms, including leaf and bud blight, shoot distortion and dieback, result from repeated infections throughout the host growing season.

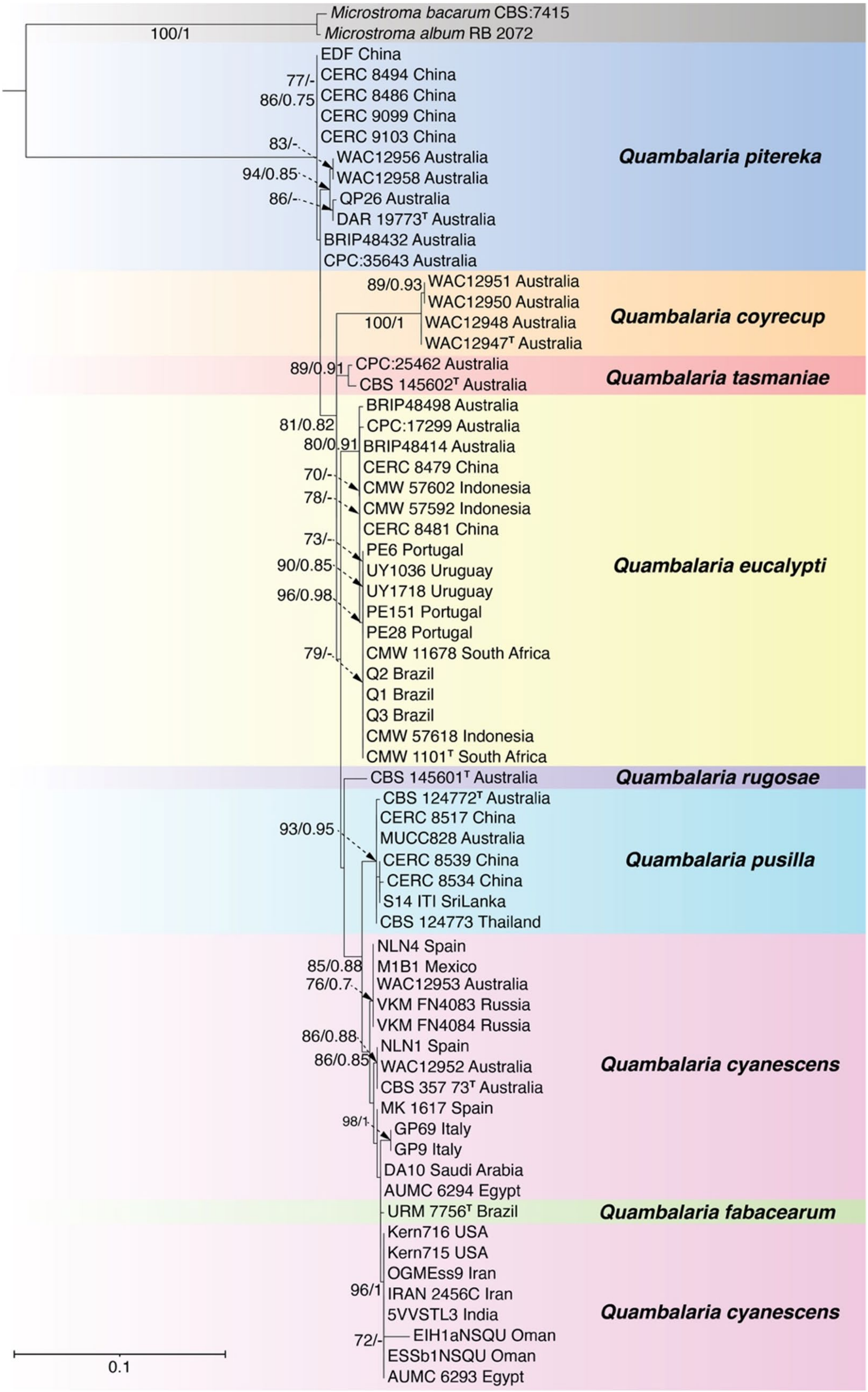
The possibility of a sexual cycle in *Quambalaria* spp. is supported by a recent population genetics study of *Q. pitereka* [29]. Using amplified fragment length polymorphism analysis of over 600 loci from 177 isolates, the study found high genotypic diversity across three study sites. The absence of a dominant genotype and the presence of linkage disequilibrium suggested frequent meiotic recombination, contradicting a clonal epidemic model and supporting a sexually reproducing life cycle. The lack of genetic structuring by host species or location, despite large geographic distances between the study sites, further supported this view and implies widespread dispersal of recombinant propagules, such as basidiospores. These findings are consistent with *Q. pitereka* being strictly outbreeding, as previously hypothesised by Begerow et al. [30]. Currently, the evidence for sexual reproduction relies solely on population genetic data. This hypothesis should be further tested using cytological studies and also investigated for other *Quambalaria* spp.

Fig. 2 Maximum Likelihood phylogeny of *Quambalaria* based on concatenated dataset (ITS and LSU). The tree includes representative isolates from all eight currently accepted species in *Quambalaria,* including types (T). The analysis was conducted using the maximum likelihood method with bootstrap support values (≥70%) and Bayesian posterior probabilities (≥0.70) shown at the nodes as ML/BI. The tree was rooted using *Microstroma bacarum* and *Microstroma album* as outgroups ▶

While the hypothesised life cycle of *Q. pitereka* is sexual and outcrossing, it remains unclear whether other *Quambalaria* spp. produce basidiospores or rely primarily on conidia as their main infectious propagules (Fig. 3). In support of a sexual cycle Paap et al. [8], when describing *Q. coyrecup*, illustrated denticulate conidiogenous cells that could represent sterigmata on basidia. However, the primary and secondary conidia of *Q. coyrecup* bear no morphological resemblance to basidia or basidiospores. These same morphological features were observed by Paap et al. [8] when examining the original specimen of this fungus (PERTH 00791962) collected by CT Brocket in 1927. Similar morphological structures can also be seen in the description of *Q. eucalypti* by Wingfield et al. [7]. Collectively, these observations suggest that *Quambalaria* spp. likely possess both sexual and asexual reproductive modes (Fig. 3), with the dominant strategy influenced by factors such as host species and environmental conditions.

Disease Symptoms

The majority of *Quambalaria* spp. have been reported from Myrtaceae, although records from other hosts exist (Table 1, S1). However, the strength of evidence supporting their role as primary pathogens varies among taxa. For well-known species, such as *Q. eucalypti* [12, 31], *Q. pitereka* [32] and *Q. coyrecup* [33], pathogenicity has been shown in inoculation experiments, confirming their ability to cause disease on susceptible hosts. *Quambalaria cyanescens* has been isolated from numerous plant species on various continents but is not known to cause disease symptoms (Fig. 1, Table 1, S1). This suggests that it is not a plant pathogen but an opportunist occurring on plant tissues and medically compromised patients [34–36]. The remaining species, such


Microstroma bacarum CBS:7415
Microstroma album RB 2072
100/1
EDF China
77/-
CERC 8494 China
86/0.75
CERC 8486 China
CERC 9099 China
CERC 9103 China
83/-
WAC12956 Australia
94/0.85
WAC12958 Australia
86/-
QP26 Australia
DAR 19773T Australia
BRIP48432 Australia
CPC:35643 Australia
Quambalaria pitereka
89/0.93
WAC12951 Australia
WAC12950 Australia
100/1
WAC12948 Australia
WAC12947T Australia
Quambalaria coyrecup
89/0.91
CPC:25462 Australia
CBS 145602T Australia
Quambalaria tasmaniae
BRIP48498 Australia
81/0.82
CPC:17299 Australia
80/0.91
BRIP48414 Australia
CERC 8479 China
70/-
CMW 57602 Indonesia
78/-
CMW 57592 Indonesia
CERC 8481 China
73/-
PE6 Portugal
90/0.85
UY1036 Uruguay
96/0.98
UY1718 Uruguay
PE151 Portugal
PE28 Portugal
CMW 11678 South Africa
79/-
Q2 Brazil
Q1 Brazil
Q3 Brazil
CMW 57618 Indonesia
CMW 1101T South Africa
Quambalaria eucalypti
CBS 145601T Australia
Quambalaria rugosae
CBS 124772T Australia
CERC 8517 China
93/0.95
MUCC828 Australia
CERC 8539 China
CERC 8534 China
S14 ITI SriLanka
CBS 124773 Thailand
Quambalaria pusilla
NLN4 Spain
85/0.88
M1B1 Mexico
76/0.7
WAC12953 Australia
VKM FN4083 Russia
VKM FN4084 Russia
86/0.88
NLN1 Spain
86/0.85
WAC12952 Australia
CBS 357 73T Australia
MK 1617 Spain
98/1
GP69 Italy
GP9 Italy
DA10 Saudi Arabia
AUMC 6294 Egypt
Quambalaria cyanescens
URM 7756T Brazil
Quambalaria fabacearum
Kern716 USA
Kern715 USA
OGMEss9 Iran
96/1
IRAN 2456C Iran
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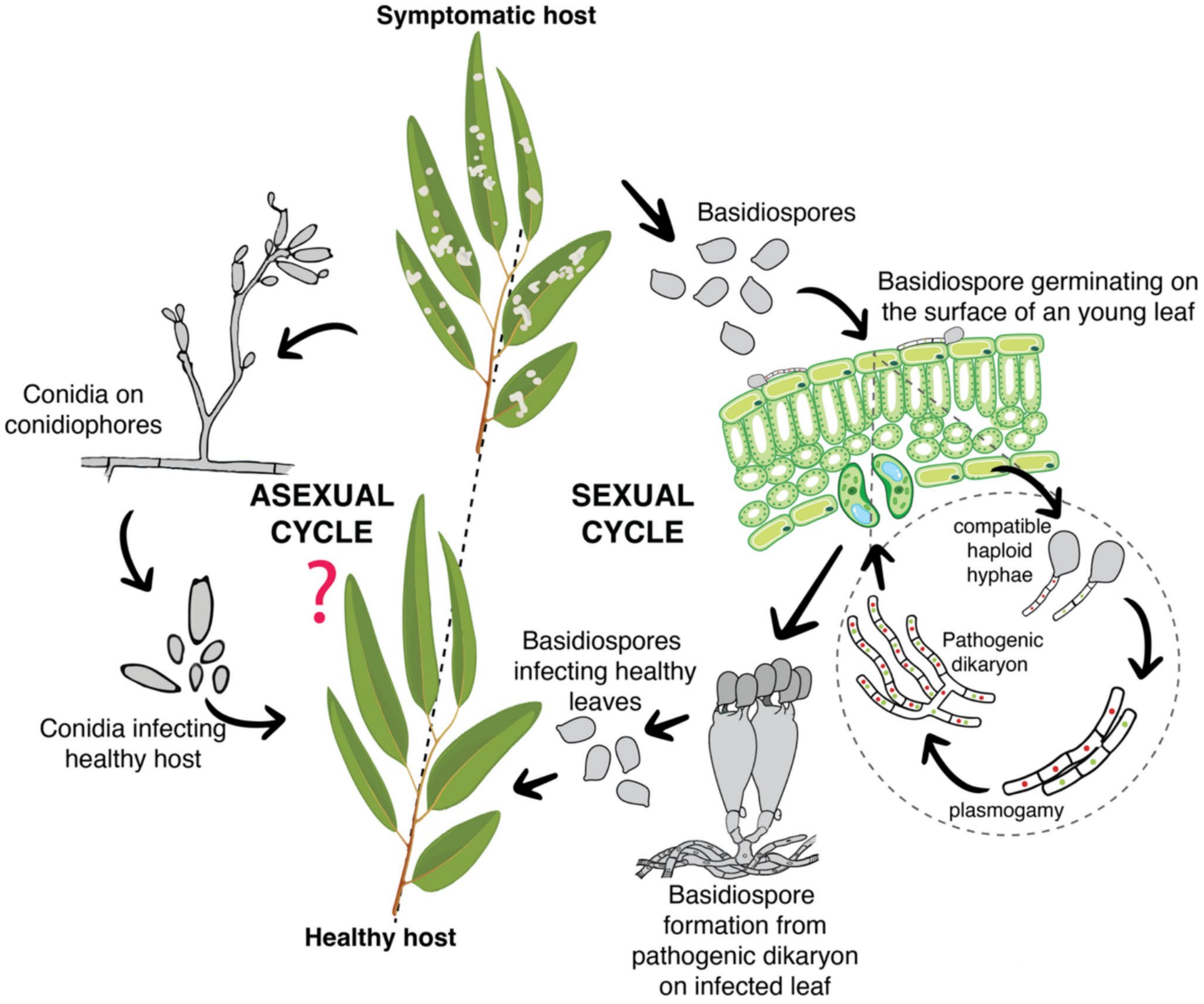

Fig. 3 Putative life cycle of *Quambalaria*. The life cycle is hypothesised based on current morphological and population genetic evidence. In *Quambalaria pitereka*, a sexual cycle has been observed in which basidiospores germinate on susceptible leaf surfaces and compatible haploid hyphae undergo plasmogamy, producing pathogenic dikaryotic hyphae. These hyphae infect young leaves and shoots, leading to symptoms such as leaf blight, shoot distortion and dieback. Spore-producing structures resembling conidia (mitospores) have been reported in several *Quambalaria* spp., including *Q. eucalypti* and *Q. coyrecup*, but their biological nature remains uncertain; they may represent asexual spores or basidiospores resulting from meiosis (indicated with a question mark)

as *Q. fabacearum* [26], *Q. pusilla* [18], *Q. rugosae* and *Q. tasmaniae* [27] have been reported from symptomatic plant tissues or as endophytes, but their role as pathogens remains to be determined (Table 1, S1).

Quambalaria spp. with confirmed pathogenicity infect hosts through wounds or natural openings such as stomata, with hyphae colonizing intercellular spaces without direct penetration of parenchyma cells [37, 38]. These fungi colonise actively growing tissues, including leaves, shoots and floral structures, where they produce sporulating lesions upon entering a necrotrophic phase [11, 12, 31, 37, 39]. However, this infection strategy differs in *Q. coyrecup*, which infects woody tissues [8]. Disease development is often favoured by warm, humid conditions that promote conidial production [37] and dispersal, enabling rapid spread within susceptible host populations, particularly during periods of active host growth [39].

Quambalaria eucalypti is a pathogen of *Eucalyptus* trees, where it commonly causes leaf and shoot blight. Initial symptoms appear as leaf spots bearing white, powdery spore masses (Fig. 4A), which progress to leaf blight accompanied by leaf distortion and buckling [4, 12] (Fig. 4B, C). Under conducive, moist conditions, such as in nurseries, the infection may also extend to young stems, producing sunken, black cankers that sometimes contain visible white spores (Fig. 4B, C). As the disease intensifies, affected trees

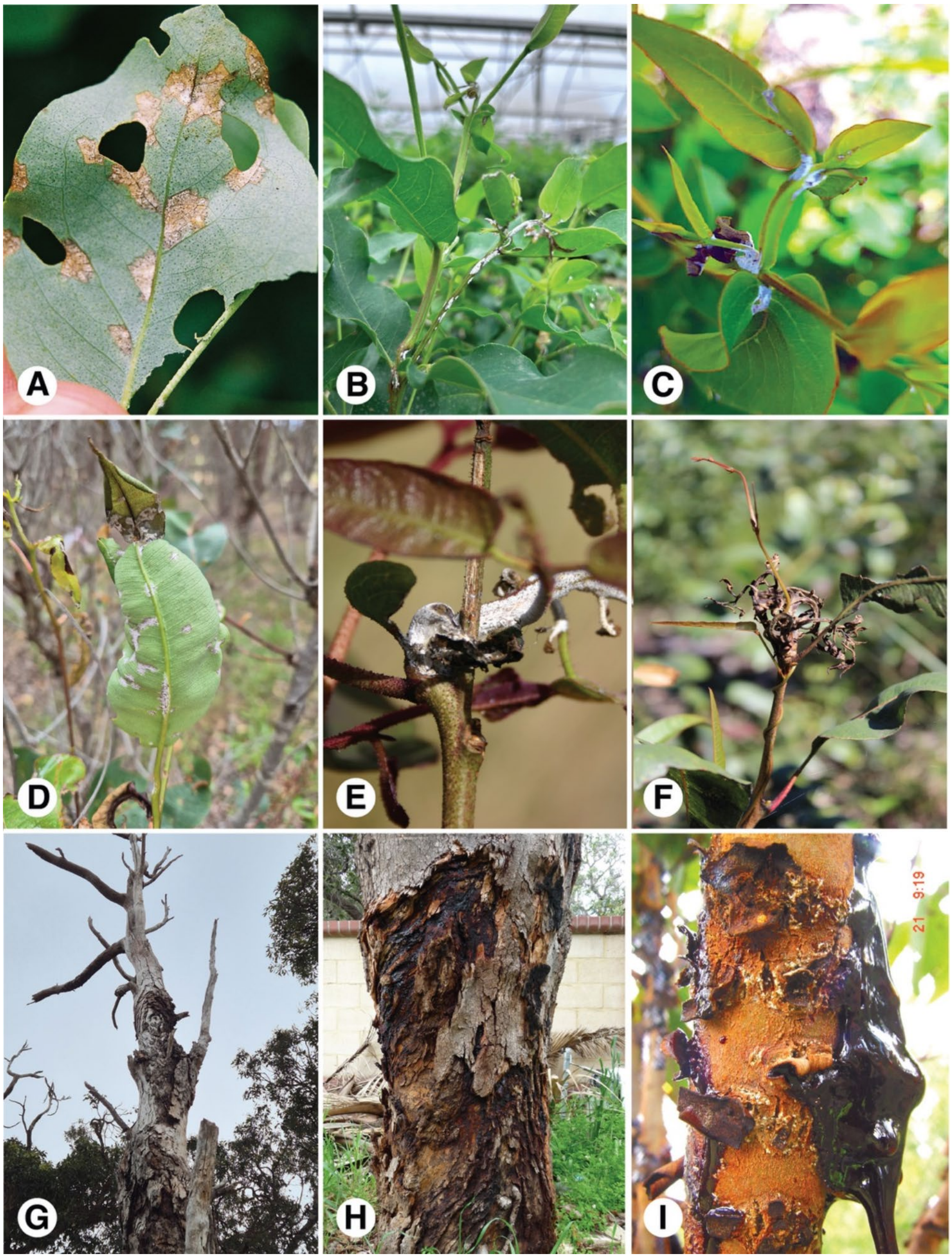

Fig. 4 Disease symptoms caused by *Quambalaria* spp. on eucalypt hosts. *Quambalaria eucalypti*: (**A**) leaf spots on *Eucalyptus globulus* from Queensland, Australia; (**B**) sporulating blight and shoot dieback on mini-hedges used for cutting propagation in a South African forestry nursery; (**C**) blight and shoot dieback in a South African *Eucalyptus* plantation. *Quambalaria pitereka*: (**D**) shoot blight of *Corymbia calophylla* in Western Australia; (**E**) shoot blight on plantation-grown *Corymbia variegata* from Kingaroy, Queensland; (**F**) shoot dieback on regenerating *C. calophylla* following wildfire in Western Australia. *Quambalaria coyrecup*: (**G**) mortality of *C. calophylla* in Western Australia; (**H**) perennial trunk canker on an urban tree of *C. calophylla* from Western Australia. *Quambalaria cyanescens*: (**I**) stem cracking and gummosis on *Eucalyptus* sp. infested with stem-boring insects in Queensland, Australia

experience severe dieback of leaves and shoots, along with distorted new growth in susceptible *Eucalyptus* genotypes. [12].

Leaves of *Corymbia* spp. infected by *Q. pitereka* are characterised by creamy to light brown pustules on both surfaces, often accompanied by leaf distortion, buckling and necrotic lesions [37, 40] (Fig. 4D, E). An in vitro study showed that disease development is favoured by temperatures ranging between 20–25 °C and humidity above 90% [37]. Young shoots are particularly susceptible, with infections leading to shoot blight and dieback of actively growing tips [4] (Fig. 4F). Stem distortion and small cankers can develop at leaf bases or on young stems, particularly with repeated infections. Chronic shoot dieback may stimulate epicormic shoot production, resulting in a bushy, malformed tree structure [37]. These symptoms collectively reduce tree growth and form and under conditions of high disease pressure, particularly in monoculture plantations, can lead to tree mortality [10, 11]. Data from field trials identified serious impacts of *Q. pitereka* on tree form and stem architecture, decreasing the quality and value of trees, as well as impacting tree growth rates [41].

Corymbia calophylla trees infected with *Q. coyrecup* develop elongated perennial stem cankers that exude gum and gradually girdle branches or stems, leading to progressive decline and eventual tree death [8] (Fig. 4G, H). Early symptoms begin as sunken, resinous lesions that enlarge into target-like cankers containing a distinctive white fungal sporulation layer beneath the bark. The pathogen produces similar cankers on *C. ficifolia*, with disease observed on planted trees in urban and disturbed environments. However, this canker disease has not been observed in undisturbed populations within this species' natural range.

Interactions with insects appear to be an important factor influencing the infection of some *Quambalaria* spp. in various host plants. Insects such as bark beetles and weevils (Curculionidae) may also vector spores or facilitate infection, such as in the cases of *Q. cyanescens* [42] and *Q. eucalypti* [4], while honey bees and other insects have been shown to transmit *Q. pitereka* [43]. In Australia, *Q. eucalypti* is frequently associated with insect damage, especially with weevils from the Curculionidae [4]. Based on these observations, it has been hypothesised that the fungus might exploit wounds caused by insect feeding as entry points into host tissue [4]. Wounding by insects may facilitate colonisation by *Q. cyanescens*; however, it is not a pathogen but rather an opportunistic coloniser of plant tissues (Fig. 4I). For example, the decline of walnut (*Juglans regia*) caused by *Q. cyanescens* in Iran coincided with infestations by codling moths, *Cydia pomonella*, a recognised pest of walnuts [44]. Similarly, *Q. cyanescens* has been isolated from leaf spots on date palms, *Phoenix dactylifera*, in Oman infested with the dubas bug, *Ommatissus lybicus*, a phloem-feeding insect [45]. These co-occurrences of the fungus and insects support the hypothesis that *Q. cyanescens* depends on predisposing factors, such as mechanical or insect damage, to successfully colonise host tissues and initiate disease.

Effects of *Quambalaria* Diseases

Nursery Diseases

Quambalaria eucalypti and *Q. pitereka* thrive in nursery environments where biological, environmental and operational factors favour infection and spread. Warm, humid and sheltered conditions, combined with overhead irrigation and dense planting, promote fungal growth and dispersal. High plant density and genetic uniformity in vegetative propagation further accelerate disease development. Routine practices such as shoot harvesting for cuttings create wounds that facilitate infection [13, 38], while repeated wounding and the regeneration of young, susceptible tissue enable these pathogens to persist and spread through successive propagation cycles.

Quambalaria eucalypti was first recorded in South Africa in 1987, infecting propagation hedges of two *Eucalyptus grandis* clones in the KwaZulu-Natal province [7]. Subsequent reports confirmed its identity and pathogenicity in several other countries (Table 1; Fig. 1). In 2000, *Q. eucalypti* was reported infecting *E. saligna* × *E. maedenii* and *E. saligna* mini-hedges in a plantation nursery in the state of Rio Grande do Sul in Brazil [46]. More recently, outbreaks have been documented in nurseries in Indonesia in North Sumatra, Riau and North Kalimantan [13].

Quambalaria pitereka was first identified from nurseries in eastern New South Wales, Australia, in 1971, where it caused substantial damage to *C. maculata* and *C. eximia* seedlings [6]. However, evidence suggests that the disease had been present in nurseries for at least 15 years before this report, resulting in mortality rates exceeding 50% in the case of *C. maculata* [47]. A personal correspondence from J de Bavay in 1955 to Walker and Bertus described a similar disease affecting *C. maculata* under glasshouse conditions in Sydney. The symptoms she recorded matched closely those later detailed by Walker, Bertus [6]. Despite its significant impact in nurseries, *Q. pitereka* has not been reported in similar settings outside of Australia (Table 1, Fig. 1). This limited distribution may be due to its host specificity, as *Q. pitereka* infects only *Corymbia* species, which are less commonly planted outside of Australia.

Plantation Forestry

Quambalaria pitereka is an important pathogen of spotted gums, such as *Corymbia variegata*, *Corymbia citriodora*, *Corymbia henryi* and *Corymbia maculata*. The pathogen has caused extensive shoot blight in plantations across Australia, affecting trees at all developmental stages, from seedlings and saplings to mature trees [4, 11, 40]. These plantations are established in areas adjacent to native forests of *Corymbia* species, which are believed to be the initial source of inoculum. Forest health surveys in New South Wales between 1996 and 2005 consistently identified *Q. pitereka* as the main disease in *Corymbia* plantations, contributing to widespread shoot dieback and reduced growth and form [48]. Little disease was observed in the first few years of plantation expansion in New South Wales, which began in the mid-1990s to offset reduced access to native forest harvesting. However, once the number of plantations and the area planted to *Corymbia* increased, the proportion of plantations with damage from *Q. pitereka* substantially rose. By 1999, 95% of *Corymbia* plantations had damage from *Q. pitereka*, two-thirds at significant levels [48]. Similarly, in Queensland, severe damage from *Q. pitereka* occurred to *Corymbia* plantations following extensive expansion. This led to a brief cessation of planting of *Corymbia* in New South Wales until more tolerant species (*C. variegata*) and provenances were identified [32] and the removal of the species as a priority for future plantation establishment in

Queensland [49–51]. Infection in mature plantations occurs at very low levels in a small number of highly susceptible trees, resulting in stunted, multi-stemmed trees (*pers. obs.* A. J. Carnegie). Today, plantation expansion using eucalypts in subtropical and tropical regions of Australia remains limited, partly due to persistent pest and disease pressures. Spotted gum is no longer a preferred species in plantations and the importance of the disease has diminished due to a lack of young plantations. Although spotted gum remains a valuable timber species, its utilisation now relies largely on harvesting from native forests rather than plantations.

Quambalaria eucalypti has been recorded in Australia (New South Wales and Queensland) on plantation species such as *E. grandis*, *E. dunnii* and *E. longirostrata*, where it causes restricted necrotic leaf lesions, leaf distortion, primarily on juvenile foliage and occasional stem infections [4]. It is not considered a significant pathogen in Australia and has not been reported from the extensive *E. globulus* or *E. nitens* plantations in temperate regions of the country. This species has a broad host range and its presence across subtropical and tropical plantation regions highlight the capacity of the pathogen to influence plantation productivity in diverse climatic zones [4]. In South Africa, *Q. eucalypti* was initially restricted to forestry nurseries. Later, Roux et al. [12] reported extensive leaf and shoot dieback, stem cankers and mortality of young shoots on one-year-old *E. nitens* in a plantation setting. Similarly, in Brazil, where the pathogen was previously known only from nursery outbreaks, it has now been identified in plantation trees [14]. In Portugal, widespread infection of *E. globulus* in plantations was detected during national monitoring surveys [31]. These reports underscore the expanding geographic range of *Q. eucalypti* and the increasing threat it poses to global *Eucalyptus* plantation health.

In plantations, *Q. cyanescens* is typically detected only in the presence of other *Quambalaria* spp., such as *Q. coyrecup*, *Q. eucalypti* and *Q. pitereka*, raising questions about its ecological role in disease development [4, 8]. Although it has been isolated from *E. pauciflora* leaves in Armidale, New South Wales [25], it remains unclear whether other *Quambalaria* spp. were also present in those samples.

Urban and Native Forests

Quambalaria coyrecup is endemic to southwestern Australia, where it causes cankers on *C. calophylla*, a widespread and ecologically important tree. In intact forests, co-evolution with its host likely moderates disease expression, but in fragmented and disturbed landscapes, *Q. coyrecup* has become a major driver of *C. calophylla* decline [52]. In these environments, infection results in kino-exuding stem and branch cankers that progressively girdle tissues, leading to dieback and tree mortality. Disease incidence is strongly linked to anthropogenic disturbance, including fragmentation, land-use change and edge effects, which increase tree stress and disrupt beneficial mycorrhizal associations [9, 53]. Climate stressors further weaken host defences and increase susceptibility to pathogen impacts [54, 55]. The fungus was first observed on a widely planted amenity tree, *C. ficifolia*, a range-restricted species naturally occurring in a small area of the south coast of Western Australia; however, as far as is known, *C. ficifolia* remains unaffected by canker within its natural distribution (*pers. obs*. T. Paap).

Quambalaria pitereka was introduced into southwestern Australia from eastern states of the country during the 1990s, likely through human-mediated movement of infected plant material [8]. In this region, *C. calophylla* is susceptible, with infection resulting in bud blight, leaf spots, shoot dieback and reduced growth [8, 56]. However, disease development depends on synchrony between high humidity and flushes of new growth [37]. Thus, severe outbreaks are typically sporadic and confined to periods or localities where these conditions coincide. The capacity of *C. calophylla* to resprout following fire is a key resilience mechanism for the species, yet the resulting juvenile shoots are highly susceptible to *Q. pitereka* and disease can be particularly severe after fire [56] (*pers. obs.* G. Hardy). Unlike *Q. coyrecup*, *Q. pitereka* does not cause widespread mortality of mature trees in this region. However, its establishment remains a concern given the lack of co-evolutionary history of *C. calophylla* with the pathogen and the limited inherent resistance evident in some populations [56].

Quambalaria rugosae, described from *Eucalyptus rugosa* on Kangaroo Island, South Australia, is closely related to *Q. pitereka* [27]. The species is currently known only from a localised area on Kangaroo Island, a biodiversity hotspot with many endemic species [57]. To date, *Q. rugosae* has not been associated with significant disease or host decline. However, given the restricted distribution of *E. rugosa* and the close relationship of *Q. rugosae* to known foliar pathogens, ongoing surveillance is warranted. The potential for introduction to the Australian mainland through the movement of horticultural plant material also represents a biosecurity consideration [58–60].

Beyond Australia, *Quambalaria* spp. may also pose risks to native vegetation in regions where these fungi have been accidentally introduced. For example, *Q. eucalypti* was detected during a routine survey on a single native *Myrceugenia glaucescens* tree in Tacuarembó, Uruguay. [61]. Although this appears to be an isolated case, the infected tree was located near a commercial *Eucalyptus* plantation, suggesting possible spillover from plantation forestry into adjacent native vegetation. Genetic analysis revealed that the isolate from *M. glaucescens* was identical to one from

Eucalyptus in nearby Durazno Province, where plantations are widespread [61]. The proximity of plantations to native Myrtaceae-rich ecosystems in South America, Asia and Africa, such as those from the genera *Eugenia*, *Syzygium*, *Myrsine* and *Myrtus,* increases the risk of cross-infection. For example, in South Africa, refugia of native forests often occur within or near *Eucalyptus* plantations, creating greater opportunities for intro-generic host jumps, as has occurred with the Cryphonectriaceae [62]. This underscores the importance of systematic surveillance in native ecosystems, as pathogen introductions may otherwise go undetected in the absence of targeted monitoring.

Management

Fungi in the genus *Quambalaria* are endemic to Australia, with host specificity providing strong evidence of their evolutionary origins (Fig. 1, Table 1, S1) [4, 25], especially in a region that is also the centre of eucalypt diversity [63, 64]. The long-standing association between *Quambalaria* spp. and their Australian hosts suggests a co-evolutionary relationship in which natural selection has driven the development of host resistance mechanisms. Consequently, eucalypts have evolved defence responses against *Quambalaria* infections, typically controlled by oligogenic resistance, a pattern consistent with other coevolved host–pathogen systems [65–67].

While all *Corymbia* spp. used for commercial plantations are susceptible to infection by *Q. pitereka*, there is high variability in resistance between and within species, provenances and families, offering the potential for disease-resistance breeding [32, 41, 49–51, 68, 69]. The importance of such variation was highlighted during the rapid expansion of eucalypt plantations in New South Wales in the mid-1990s, where both the incidence and severity of *Q. pitereka* increased significantly between 1996 and 1999 as large areas of *C. maculata* were established [48]. This led to a shift in planting strategies, with *C. maculata* largely replaced by more tolerant provenances of *C. variegata*. Subsequently, extensive efforts were undertaken in New South Wales and Queensland to identify and deploy resistant genotypes, including the development of hybrids and clonal material [32, 41, 49–51, 68, 69].

Effective resistance deployment must also account for pathogen variability. *Quambalaria pitereka* exhibits considerable genetic and phenotypic diversity, with isolates differing in virulence on the same host [70]. For example, Pegg et al. [70] demonstrated significant differences in aggressiveness among isolates on spotted gum species and provenances. Similarly, in Western Australia, *C. calophylla* provenances originating from wetter regions show greater resistance to *Q. pitereka* [56]. These findings highlight the need to align host selection not only with site conditions but also with local pathogen populations, an aspect that has received limited attention to date.

At the landscape scale, disease dynamics differ between regions. In Australia, native forests act as a continuous reservoir of inoculum for nurseries and plantations. In contrast, in regions where *Quambalaria* spp. occur outside their native range, there is greater scope to manage disease through landscape-level interventions. These include risk-site mapping, coordinated management across neighbouring plantations and maintaining buffers between plantations and native vegetation to reduce inoculum pressure [4, 48].

Nurseries represent particularly high-risk environments for infection due to high plant densities, monoculture systems and frequent irrigation. In South Africa, *Q. eucalypti* is primarily a nursery pathogen affecting mini-hedges and vegetative cuttings [7, 12]. Management in these systems, therefore, relies on the use of clean planting material, strict hygiene practices and environmental control, especially reducing humidity, which favours conidial germination [37]. Where necessary and permitted, systemic fungicides may be applied, although their use must be balanced against cost and regulatory constraints [71]. However, fungicides were widely used in Australian nurseries during the expansion of spotted gum plantations in the 1990 s (*pers. obs.* A.J. Carnegie).

Overall, these observations highlight that effective management of *Quambalaria* diseases depends on integrating host resistance, pathogen variability and inoculum dynamics within a site-specific epidemiological framework.

Knowledge Gaps and Future Prospects

Much of the current understanding of *Quambalaria* spp. is limited to their occurrence on eucalypts, predominantly *Eucalyptus* and *Corymbia*. This narrow focus limits our knowledge of their broader host range and potential ecological roles beyond their function as plant pathogens.

Quambalaria spp. infect hosts through wounds or natural openings [37, 38]. This suggests a relatively non-invasive strategy, but it is unclear whether infections remain localised or spread systemically. Systemic colonisation would have major implications for management and could explain symptomless persistence. *Quambalaria* can also persist as an endophyte in *C. calophylla* in undisturbed forests, only expressing pathogenicity under stress resulting from anthropogenic disturbance [72, 73]. This opportunistic behaviour resembles that of the tree pathogens *Cytospora* [74] and *Botryosphaeria* spp. [75]. Understanding whether *Quambalaria* survives primarily inside host tissues or in external reservoirs and how host or environmental stress influences disease expression remains a critical knowledge gap.

Insects [4, 43, 45, 76] and artificial wounds [13, 38] often serve as the infection points for various *Quambalaria* spp. [4, 42, 43]. Wounding of host tissues can facilitate pathogen entry by bypassing natural defence barriers, while insects may contribute both by creating feeding wounds and by acting as potential vectors, carrying fungal propagules between hosts [77, 78]. Although the role of specific insects as vectors of *Quambalaria* spp. remains poorly resolved, their involvement suggests that disease development may be closely linked to interactions among host susceptibility, wounding events and pathogen dispersal. Consequently, studies are needed to better understand the role of insects in the epidemiology of *Quambalaria* spp.

A clear understanding of the life cycle of *Quambalaria* spp. and the sources of infectious propagules remains a major knowledge gap. Such information is essential for effective disease management, as control strategies depend on identifying the primary source of inoculum and modes of dispersal. A confirmed sexual state has not been observed for all *Quambalaria* spp. However, population genetic evidence suggests that *Q. pitereka* may undergo sexual recombination [29]. If sexual reproduction does occur, it could introduce an additional infective spore type and generate novel genotypes with increased virulence. This would have important implications for biosecurity and breeding programs focused on plantation monocultures. Determining whether sexual reproduction occurs in *Quambalaria* and whether it is restricted to *Q. pitereka* or more widespread across the genus, will require integrated population genetic, cytological and pathological studies. Clarifying which sexual or asexual spores are produced and under what conditions they form and disperse, remains a critical step towards understanding the life cycle of these pathogens.

As reported for *Q. pitereka*, host susceptibility to *Quambalaria* spp. varies widely [70] and this variability is further complicated by differences in virulence among pathogen isolates. Due to this host–pathogen complexity, as seen with various other tree pathogens, reliable predictions of resistance are challenging, underscoring the need for broader, multi-site and multi-genotype trials to assess how resistance holds up across diverse environments and pathogen populations. Thus, breeding remains the cornerstone of *Quambalaria* disease management, as substantial genetic variation in resistance has been documented among eucalypt provenances and families [32], providing opportunities for selective improvement. However, any resistance identified must be tested against diverse pathogen populations, potential co-infections and variable environmental conditions to ensure durability [56]. Advances in tissue culture, genomics and genetic engineering offer new tools for the development and deployment of disease-resistant trees [79, 80]. Resistant trees must also meet commercial standards for growth, wood properties and climate adaptability. Ultimately, integrating resistance breeding with genomic and silvicultural strategies will be key to achieving durable *Quambalaria* disease management.

A key research priority is mapping the genetic diversity of pathogenic *Quambalaria* spp. in both native and introduced regions. Identifying additional gene markers will improve species delineation, as demonstrated in other pathogens such as *Cercospora* [81], *Ceratocystis* [82] and *Phytophthora* [83]. Expanding genomic resources will clarify taxonomy and enable more precise tracking of pathogen spread and evolution.

Climate change could intensify the impacts of *Quambalaria* spp. on eucalypt forests. Warmer, wetter conditions will promote infection and dispersal, while extreme weather will reduce host resilience [37, 84, 85]. Future research should focus on integrating climate models with epidemiological data to predict outbreak risk under various climate scenarios, as has been done for other important tree pathogens, such as various *Phytophthora* spp. [86–88]. Long-term field trials and controlled-environment experiments are also needed to test how elevated temperature and rainfall interact with host resistance and pathogen virulence.

Some *Quambalaria* spp. have been associated with increased susceptibility of trees to other opportunistic pathogens, such as *Cytospora*, *Teratosphaeria* and *Biscogniauxia mediterranea* [31]. In other cases, *Quambalaria* spp. occur as part of broader disease complexes involving multiple pathogens. For example, *Q. coyrecup* contributes to lethal cankers in fragmented and highly disturbed *C. calophylla* forests of Western Australia, especially under stress or when co-occurring with pathogens such as *Phytophthora cinnamomi* and *P. multivora* [55, 72, 89]. Their expanding range and role in disease complexes further threaten the sustainability of plantations. Future research should investigate the mechanistic basis of these interactions. Experimental inoculation studies could clarify whether managing one pathogen indirectly reduces *Quambalaria* impacts and landscape-level surveys could identify hotspots where disease complexes are most severe.

Overall, effective management of *Quambalaria* diseases will require an integrated approach that links pathogen ecology, host resistance, biotic interactions, reproductive biology, pathogen characterisation and effective phenotyping for host resistance and molecular diagnostics. Coordinated research and adaptive breeding programs are essential as eucalypt plantations expand into new environments and confront evolving pathogen pressures.

Conclusions

The rise of some *Quambalaria* spp. as globally significant tree pathogens reflects broader trends in forestry, exposing the risk of monoculture planting and international plant trade, resulting in global dissemination of plant pathogens. Once considered obscure, *Quambalaria* spp. are increasingly recognised as a threat to both commercial forestry and native ecosystems. Their capacity to persist as endophytes, interact with insect vectors and facilitate secondary infections underscores the complexity of their pathology. The strong evidence for sexual reproduction and genetic recombination further raises concern about their evolutionary potential and the emergence of more aggressive or adaptable strains.

Despite growing recognition of the importance of *Quambalaria* spp., major research gaps remain. The mechanisms underpinning host specificity, latent infections, systemic movement and vector facilitation are poorly understood. The case of *Quambalaria* highlights the importance of adopting a proactive, interdisciplinary approach that combines pathology, ecology and silviculture. Resistance breeding offers a promising avenue but must be supported by ongoing field validation, multi-isolate testing and integration with existing disease management strategies.

Looking forward, a coordinated global research agenda is essential to adequately manage threats due to tree pathogens [3], including *Quambalaria* spp. This includes mapping genetic diversity, refining species boundaries, identifying virulence markers and developing accessible tools for pathogen identification and characterisation. Equally important is the need to monitor emerging hosts and landscapes beyond eucalypt plantations. As the threat from *Quambalaria* increases, strong and adaptable forest management will be essential to keep plantations productive, safeguard biodiversity and ensure forestry remains sustainable.

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Author Contributions B.A.J. led the literature synthesis and drafting of the manuscript. T.P., G.S.P., A.J.C., M.J.W., J.R., G.E.St.J.H., A.D., and A.H. contributed to the development of the concept, provided critical input on content, and revised the manuscript for important intellectual content. T.B. conceptualised the review, assisted B.A.J with writing the review, conducted the phylogenetic analyses, coordinated the manuscript preparation and led the final editing.

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Data Availability The alignments and phylogenetic trees are available through Mendeley Data (https://doi.org/10.17632/4gdvdfssx6.1).

Compliance with Ethical Standards

Conflict of Interest All the authors declare that they have no conflict of interest.

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