AUSTRALIAN FORESTRY
2019, VOL. 82, NO. S1, 80–93
https://doi.org/10.1080/00049158.2019.1595347

Taylor & Francis
Taylor & Francis Group

ARTICLE

OPEN ACCESS Check for updates

Ceratocystis wilt and canker – a disease that compromises the growing of commercial *Acacia*-based plantations in the tropics

A. Nasution[a,b], M. Glen[a], C. Beadle[a] and C. Mohammed[a]

[a]Tasmanian Institute of Agriculture, University of Tasmania, Hobart, Australia; [b]Plant Health Program - Research and Development, PT Riau Andalan Pulp and Paper, Pekanbaru, Indonesia

ABSTRACT

Ceratocystis wilt and canker disease has severely compromised the profitability of *Acacia mangium* plantations in Southeast Asia. The focus of this review is on *Ceratocystis* wilt and canker disease in Acacia trees. Its aim is to synthesise information about this fungal pathogen that can be used to inform development of suitable disease-control strategies in forest plantations. The last 20 years have seen many taxonomic changes in *Ceratocystis*, with some disagreement as to species boundaries. Therefore, an understanding of the origins and development of this disease requires reference to other species, particularly in the context of the biology and fungal taxonomy, disease symptoms and mechanisms of fungal dispersal. The risks and impacts of the disease on the sustainability of Acacia wood production are examined. Observing or surveying disease symptoms in plantations, selecting and planting tolerant or resistant Acacia trees, and the potential of endophytic bacteria as biological control agents are also included in this review.

ARTICLE HISTORY
Received 29 August 2018
Accepted 25 February 2019

KEYWORDS
Ceratocystis fimbriata; *Ceratocystis manginecans*; *Acacia mangium*; *Eucalyptus*

Introduction

Acacia species have been exploited for wood, including fuel wood, animal forage, human food, tannin and land rehabilitation (Midgley & Turnbull 2003). In southern and eastern Africa, they have been grown for more than 100 years for the control of sand drift, and for timber, plywood and pulp and paper industries (Roux et al. 2005; Griffin et al. 2011). In the Asia-Pacific region, tropical species have been grown for over 80 years. *Acacia auriculiformis* A. Cunn. ex Benth. was introduced from Australia to Malaysia in 1932, to Thailand in 1935 and subsequently to India and China (Midgley & Turnbull 2003). *Acacia mangium* Willd. and *Acacia crassicarpa* A. Cunn. ex Benth. have formed the basis of large plantation estates for pulpwood production in Southeast Asian countries for at least three decades (Griffin et al. 2011). *Acacia mangium* was first introduced from the humid tropical forests of north-eastern Australia to Malaysia in 1966 and subsequently to Papua New Guinea, Indonesia, Bangladesh, China, India, the Philippines, Sri Lanka, Thailand and Vietnam (Midgley & Turnbull 2003; Krisnawati et al. 2011). *Acacia crassicarpa* was first planted in the early 1980s in Thailand and soon after in Indonesia where it is very well-adapted to peatlands in Sumatra (Midgley & Turnbull 2003). *Acacia* hybrid (*A. mangium* × *A. auriculiformis*) was developed over 20 years ago (Kha 2000) and is now the most commonly planted *Acacia* 'species' in Vietnam (Griffin et al. 2011). All species are fast growing, have good wood quality, are able to grow up to 30 m in height and can adapt to many types of soil and environmental conditions (Griffin et al. 2011; Krisnawati et al. 2011).

The utilisation potential of these tropical *Acacia* species for pulp and paper led to the rapid expansion of their planting and use by large industrial companies in Southeast Asia; the total area planted in this region had reached over 2000 000 ha by 2014 (Harwood & Nambiar 2014), though by then, some estates reliant on *A. mangium* had already become non-commercial (Mohammed et al. 2014). This was because climate change and operational systems had combined to reinforce the threats of pathogens to forest production (Witzell et al. 2014). In particular, intensively managed short-rotation monoculture plantations based on seedlings or clonal stock of a single species had affected forest biodiversity and led to the emergence of 'new' diseases, often in the form of more aggressive strains of pathogens (Anderson et al. 2004; Martín-García et al. 2011).

In particular, wood yields and the sustainability of production of *A. mangium* in Indonesia and Malaysia have been compromised by two diseases. The first was red root-rot disease caused by *Ganoderma philippii* (Bres. & Henn. ex Sacc.) Bres. (Lee 2004; Coetzee et al. 2011; Francis et al. 2014; Mohammed et al. 2014). Acacias became infected when planted in areas where the pathogen was already present, and losses in wood production increased with each rotation (Lee 2004; Francis et al. 2014). The second was wilt and canker disease caused by a species of *Ceratocystis* (Tarigan et al. 2011a; Fourie et al. 2015). The same pathogen is also a new disease threat in Vietnam (Thu et al. 2012). In Indonesia and Malaysia, this resulted in even greater mortality and loss of productivity in *A. mangium* than with root rot, and quickly led to the demise of this species as a source of pulpwood in Sumatra (Tarigan et al. 2011a).

The focus of this review is on *Ceratocystis* wilt and canker disease in Acacia trees. Its aim is to synthesise information about this fungal pathogen that can be used to inform development of suitable disease-control strategies in forest plantations. However, an understanding of the origins and development of this disease requires reference to other species, particularly in the context of the biology and fungal taxonomy, disease symptoms and mechanisms of fungal

CONTACT M. Glen Morag.Glen@utas.edu.au Tasmanian Institute of Agriculture, University of Tasmania, Hobart, Australia

© 2019 University of Tasmania. Published by Informa UK Limited, trading as Taylor & Francis Group
This is an Open Access article distributed under the terms of the Creative Commons Attribution-NonCommercial-NoDerivatives License (http://creativecommons.org/licenses/by-nc-nd/4.0/), which permits non-commercial re-use, distribution, and reproduction in any medium, provided the original work is properly cited, and is not altered, transformed, or built upon in any way.

dispersal. Some of the examples given below are no longer considered species of *Ceratocystis* due to recent taxonomic revisions but are still relevant in the discussion of disease transmission and control strategies. The risks and impacts of the disease on the sustainability of Acacia wood production are then examined. Observing or surveying disease symptoms in plantations, selecting and planting tolerant or resistant Acacia trees, and the potential of endophytic bacteria as biological control agents (BCA) also form part of this review.

Biology and disease dynamics

Ceratocystis is a fungal genus with several species that cause rot diseases of agricultural crops and vascular wilt and canker stain of woody plants (Van Wyk et al. 2009a; Harrington 2013), though application of the term vascular wilt to diseases caused by *Ceratocystis* and related species is controversial (Kile 1993). A typical vascular wilt pathogen such as *Fusarium* or *Verticillium* moves through the xylem but does not invade xylem parenchyma or ray cells until the host metabolism is disrupted and the tissues surrounding xylem vessels die (Talboys 1972). *Bretziella fagacearum* (Bretz) Z.W. de Beer, Marinc., T.A. Duong & M.J. Wingf. (syn. *Ceratocystis fagacearum* (Bretz) J. Hunt) is the only member of Ceratocystidaceae that fits this classical definition (Juzwik 2008). By contrast, species such as *Ceratocystis platani* (J.M. Walter) Engelbrecht & Harrington and *Ceratocystis albifundus* M.J. Wingf., de Beer & M.J. Morris kill parenchyma tissue and also kill cambium and bark tissue, resulting in cankers (Morris et al. 1993; Lehtijärvi et al. 2018). *Ceratocystis* species invade their hosts through wounds, which may be caused by human activity, other mammals such as monkeys, elephants and squirrels, wind or boring insects (Harrington 2007; Tarigan 2011b).

The emergence of *Ceratocystis* wilt and canker disease in commercial plantings of *Acacia* and also *Eucalyptus* spp. has been associated with a period of rapid growth of plantation estates based on these species following their introduction as non-native trees (Wingfield et al. 2001b; Roux & Wingfield 2009). Disease development is associated with discolouration of woody tissues, leaf yellowing, wilting and canker, and levels of mortality that affect the commercial viability of plantations (Barnes et al. 2003a; Roux & Wingfield 2009; Brawner et al. 2015). In Acacias, these symptoms have been reported for *Acacia mearnsii* De Wild. in South Africa and Uganda (Wingfield et al. 2001a), and for *A. mangium* in Indonesia (Tarigan et al. 2011a), Malaysia (Brawner et al. 2015) and Vietnam (Thu et al. 2012). These symptoms have also been reported for Eucalypts in Africa (Roux et al. 2000a; Roux et al. 2000b; Roux et al. 2001a; Roux et al., 2004b; Van Wyk et al. 2010a) and South America (Laia et al. 2000; Barnes et al. 2003b). The incidence of *Ceratocystis* disease in Eucalypts in Indonesia is confined to a small number of susceptible clones (Heru Indrayadi pers. comm.) and it has not yet been determined whether the pathogen on Eucalypts is identical to that on Acacias though this work is in progress (Istiana Prihatini pers. comm.).

Taxonomy and plant hosts

The genus *Ceratocystis* and the genus *Ophiostoma* were previously placed in the same order, Ophiostomatales, based on the similarity of their conidial morphology. Ascocarps of both genera have a similar pattern of development and ecological niche; their necks are elongated and able to bear masses of sticky spores that easily stick to the legs and bodies of insects that feed on these fungi, facilitating spore dispersal to other trees (Malloch & Blackwell 1993). However, species in these genera can be distinguished through an examination of the anamorph (asexual) stage and their sensitivity to the antibiotic cycloheximide. *Ophiostoma* species are tolerant to cycloheximide, while *Ceratocystis* species are sensitive to cycloheximide (Samuels 1993). This difference and DNA sequence analyses have resulted in *Ceratocystis* being placed in the order Microascales (Spatafora & Blackwell 1994) and family Ceratocystidaceae (Réblová et al. 2011), distinct from Ophiostomatales.

A taxonomic revision of the family, supported by phylogenetic analyses (De Beer et al. 2014), resulted in the erection of two new genera, *Davidsoniella* and *Huntiella*, as well as emended descriptions for *Ambrosiella, Ceratocystis, Chalaropsis, Endoconidiophora* and *Thielaviopsis*. Species of *Ambrosiella* and *Endoconidiophora* are associates of ambrosia and bark beetles (Coleoptera: Scolytinae), with minimal direct impact on plant health. Most of the species pathogenic to dicotyledonous plants were placed into two of the emended genera, *Ceratocystis* and *Davidsoniella*, while pathogens of monocotyledonous plants fell into a single clade that corresponded to the emended description of *Thielaviopsis*. No sexual state is known for species retained or transferred to *Chalaropsis*, and the asexual state is indistinguishable from those of *Ceratocystis* spp. These species are found on woody substrates but are not known to have any ecological or economic significance (De Beer et al. 2014). *Huntiella* includes wound-colonising saprobes or mild pathogens that may be responsible for sapstain in timber. Some species, including the oak wilt pathogen *C. fagacearum*, did not fit into a well-defined clade. Subsequently, another new genus, *Bretziella*, was erected to accommodate *B. fagacearum*, syn. *C. fagacearum* (De Beer et al. 2017).

The species remaining in the redefined genus *Ceratocystis* consist of those conforming to the previous species concept of *Ceratocystis fimbriata* Ellis & Halst. (De Beer et al. 2014; Liu et al. 2018). Species delineations in this group are controversial and some authors maintain that many of the newly described species are conspecific with *C. fimbriata* (Oliveira et al. 2015).

The pathogenic association of a species of *Ceratocystis* with a cultivated crop was first reported in 1890 in New Jersey, USA, where it was associated with tuber black rot on *Ipomoea batatas* (L.) Lam. (sweet potato) (Halsted & Fairchild 1891). The causal fungus was described as the new species *C. fimbriata*. Since then, a large number of agricultural crops and woody trees, both gymnosperms and angiosperms, have been reported as hosts of *C. fimbriata* and related species (Roux & Wingfield 2009; Al Adawi et al. 2013). Baker et al. (2003) list 31 species of plants from 14 families as hosts of *Ceratocystis* spp. As well as many tree species, this includes root crops, edible aroids (*Araceae), Coffea arabica* L. and *Theobroma cacao* L. In *Colocasia esculenta* (L.) Schott (taro), *Ceratcystis fimbriata* causes a post-harvest rot, similar to that on sweet potato (Harrington et al. 2015).

Tree hosts include conifers such as *Picea abies* (L.) H. Karst. (Norway spruce), *Pinus* spp. and hardwoods: *Quercus* spp., *Ulmus* spp., *Platanus* spp., *Eucalyptus* spp. and *Acacia* spp.; *Hevea brasiliensis* Müll. Arg. and other cultivated

trees are also affected (Kile 1993; Harrington 2007). Economically important levels of damage can be caused, for instance, tree death of *Mangifera indica* L. (mango) caused by *Ceratocystis manginecans* M. van Wyk, Al Adawi & M.J. Wingf., and wilt disease of *Punica granatum* L. (pomegranate) by *C. fimbriata* (Huang et al. 2003). Environmental impacts of *Ceratocystis* species may also be dramatic; for example, rapid ohia death (ROD) in *Metrosideros polymorpha* Gauch. (Keith et al. 2015).

Initially identified as *C. fimbriata* (Morris et al. 1993; Roux & Wingfield 2009) several new *Ceratocystis* species have been described as causing serious damage on *Acacia* spp., for example, *C. albifundus* wilt and canker disease in *A. mearnsii* (Barnes et al. 2005). The *Ceratocystis* species infecting *Acacia* spp. in Indonesia was initially identified as belonging to two species, *C. manginecans*, originally described from *M. indica* in Oman (van Wyk et al. 2007a), and a new species, *Ceratocystis acaciivora* Tarigan & M. van Wyk (Tarigan et al. 2010), the two being differentiated by slight differences in their rDNA internal transcribed spacer (ITS) sequence. Subsequent phylogenetic analyses showed that the ITS marker was unreliable (Al Adawi et al. 2013; Naidoo et al. 2013) and phylogenetic analyses based on four informative gene regions and single nucleotide polymorphism (SNP) markers, could not distinguish these two species (Fourie et al. 2015). As a result, *C. acaciivora* was considered synonymous with *C. manginecans* (Fourie et al. 2015). *Ceratocystis manginecans* infects *Lansium parasiticum* (Osbeck) K.C. Sahni & Bennet (duku) as well as *Acacia* spp. in Indonesia (Irsan et al. 2016), though has not been reported from mango in Indonesia. In Oman and Pakistan, it infects native legume trees (Al Adawi et al. 2013) as well as mango (Van Wyk et al. 2007a; Table 1). Two new species, *Ceratocystis mangicola* M. van Wyk & M.J. Wingf. and *Ceratocystis mangivora*, M. van Wyk & M.J. Wingf. were described from mango in Brazil (Van Wyk et al. 2011a), however, pathologists in Brazil continue to regard the pathogens on mango, *Eucalyptus, Hevea, Tectona* and many other host species as members of a single species, *C. fimbriata*, (Firmino et al. 2012; Harrington et al. 2014; Valdetaro et al. 2015; Oliveira et al. 2016; Zhang et al. 2017). Recent population genetic studies provide some evidence to support this view; isolates from kiwifruit (*Actinidia* spp.) have been separated into three distinct groups, with one group closely related to isolates from Eucalypts and another closely related to isolates from mango and taro (Ferreira et al. 2017). The majority of isolates belonged to a third group, labelled PM, that was linked to the nursery that supplied kiwifruit plants to the other sampled farms. The high level of clonal replication indicated a strong likelihood of vegetative reproduction, either in infected scion material or by contaminated tools. The relative pathogenic aggressiveness of isolates from the three groups was not tested, though all groups were isolated from multiple diseased kiwifruit vines. Phylogenetic analysis of the mating type genes provided additional support for groupings based on microsatellite data (Ferreira et al. 2017), with the PM population the most divergent.

Morphology and reproduction

Ceratocystis spp. have teleomorph (sexual) and anamorph (asexual) stages of reproduction. The teleomorph stage can be recognised through the presence of small, dark- through to light-coloured, sub-globose, globose or spherical fruiting bodies known as ascocarps (or ascomata) which are typically ostiolate or perithecial. The base of the ascocarps is enlarged and they have long necks with ostiolar hyphae on the tip (see fig. 2 in De Beer et al. 2014). Deliquescent asci emerge from the centrum of the ascocarp and produce sticky, hat-shaped ascospores (sexual spores; Upadhyay 1993). The ascospores are hyaline and lack germ-pores. The ascospores exude from the ascocarp as a sticky droplet through the ostiolar hyphae at the top of the ascomatal necks. These spores adhere easily to insects (Upadhyay 1993; De Beer et al. 2014).

The anamorph stage of *Ceratocystis* is morphologically like that of *Chalara* species. All *Ceratocystis* species have a *Chalara*-like anamorph (Paulin-Mahady et al. 2002; Harrington 2013). Simple tubular conidiogenous cells called phialides typically taper towards their apices, and produce either chains of rectangular conidia or dark barrel-shaped secondary conidia as asexual spores (De Beer et al. 2014). Production of chlamydospores or aleuroconidia by some species of *Ceratocystis* facilitates survival in and transmission through soil. Aleurioconidia are pigmented, thick-walled, chlamydospore-like spores that are produced in specialised conidiophores (see fig. 3 in Harrington 2013; De Beer et al. 2014).

Geographic distribution of *Acacia* and *Eucalyptus* pathogens

The genus *Ceratocystis* has a wide geographic distribution and can be found causing serious disease to a range of species in tropical, subtropical and temperate climates and in all continents (Table 1). In the previous century, many reports of damage to woody hosts were ascribed to *C. fimbriata*, but these pathogens have subsequently been described as new species, e.g. wilt disease in *A. mearnsii* in South Africa was initially ascribed to *C. fimbriata* (Morris et al. 1993) and later described as *C. albifundus* (Wingfield et al. 1996). The same pathogen was found in *A. mearnsii* in Uganda (Roux et al. 2001b), Malawi, Zambia (Roux et al. 2004a), Kenya and Tanzania (Roux et al. 2005). It also affects *Acacia caffra* (Thunb.) Willd., *Acacia decurrens* Willd., and *Acacia nigra* Clos, syn. *Calliandra chilensis* Benth. (Roux et al. 2007; Nkuekam et al. 2008; Roux & Wingfield 2009) as well as *Protea* spp. and *Terminalia sericea* Burch. ex DC. in South Africa (Crous et al. 2013; Pornsuriya & Sunpapao 2015). *Ceratocystis obpyriformis* R.N. Heath & Jol. Roux, *Ceratocystis pirilliformis* I. Barnes & M.J. Wingf. and *Ceratocystis polyconidia* R.N. Heath & Jol. Roux, are also found on *A. mearnsii* in South Africa (Heath et al. 2009b; Lee et al. 2016). Unfortunately, fewer publications focus on the relative damage caused by these different species or methods of disease control than on the fungal taxonomy.

Of these *Acacia*-infecting species, only *C. pirilliformis* has been recorded from Australia, where it causes sap-stain in *Eucalyptus nitens* (H. Deane & Maiden) Maiden (Barnes et al. 2003a), but has not been reported in association with *Acacia* species. A molecular and phylogenetic study using polymorphic simple sequence repeat (SSR) markers on *C. pirilliformis* indicated that this pathogen is more diverse in Australia than in South Africa and therefore presumed to be native to Australia (Nkuekam et al. 2009). In South Africa, it causes vascular stain in *Eucalyptus grandis* W. Hill (Barnes et al. 2003a; Nkuekam et al. 2009) in addition to *A. mearnsii*. Two other species, *Ceratocystis atrox* M. van Wyk & M.J. Wingf. and *Ceratocystis corymbiicola* Kamgan-Nkuek., occur on Eucalypts in Australia, while another six have been

Table 1. *Ceratocystis* pathogens of woody plants, their hosts and geographic locations

Ceratocystis species	Host	Geographic location	References
C. albifundus	*Acacia caffra*	South Africa	Roux et al. 2007
	A. decurrens	South Africa	Roux & Wingfield 2009
	A. mearnsii	Kenya, Malawi, South Africa, Tanzania, Uganda, Zambia	Roux & Wingfield 2009
	A. nigrescens	South Africa	Nkuekam et al. 2008
	Dalbergia nitidula	Zambia	Roux & Wingfield 2013
	Protea spp.	South Africa	Crous et al. 2013
	Terminalia sericea	South Africa	Pornsuriya & Sunpapao 2015
C. atrox	*Eucalyptus grandis*	Australia	Van Wyk et al. 2007b
C. cacaofunesta	*Theobroma cacao*	Brazil, Colombia, Costa Rica, Ecuador, Guatemala, Trinidad & Tobago, Venezuela	Engelbrecht et al. 2007a
	Herrania sp.	Costa Rica	Engelbrecht et al. 2007b
C. caryae	*Carya* spp.	USA	Johnson et al. 2005
C. cercfabiensis	*Eucalyptus* spp. (stumps)	China	Liu et al. 2015
C. collisensis	*Cunninghamia lanceolata*	China	Liu et al. 2015
C. colombiana	*Citrus* spp., *Coffea* sp.	Colombia	Van Wyk et al. 2010b
	Coffea arabica	Colombia	Van Wyk et al. 2010b
	Schizolobium parahybum	Colombia	Van Wyk et al. 2010b
C. corymbiicola	*Corymbia variegata*	Australia	Nkuekam et al. 2012
	Eucalyptus spp.	Australia	Nkuekam et al. 2012
C. curvata	*Eucalyptus deglupta*	Colombia, Ecuador	Van Wyk et al. 2011b
C. diversiconidia	*Terminalia ivorensis*	Colombia, Ecuador	Van Wyk et al. 2011b
C. ecuadoriana	*E. deglupta*	Colombia, Ecuador	Van Wyk et al. 2011b
C. eucalypticola	*Eucalyptus* spp.	South Africa	Nkuekam et al. 2013
	Eucalyptus sp.	Brazil, Republic of the Congo, Uganda, Uruguay	Lee et al. 2016
C. ficicola	*Ficus carica*	Japan	Kajitani & Masuya 2011
C. fimbriata	*Ipomoea batatas**	Brazil, China, Cost Rica, Haiti, Japan, Kenya, Korea, Mexico, New Zealand, Japan, Kenya, Korea, Papua New Guinea, Puerto Rico, Taiwan, USA, Virgin Is., West Indies	USDA ARS
C. fimbriatomima	*Eucalyptus* sp.	Venezuela	Van Wyk et al. 2009a
C. harringtonii	*Populus* spp.	Canada, USA, Poland	Johnson et al. 2005
C. huliohia	*Metrosideros poymorpha*	USA (Hawaii)	Barnes et al. 2018
C. larium	*Styrax benzoin*	Indonesia	Van Wyk et al. 2009b
C. lukuohia	*M. polymorpha*	USA (Hawaii)	Barnes et al. 2018
C. mangicola	*Mangifera indica*	Brazil	Van Wyk et al. 2011a
C. manginecans	*A. crassicarpa*	Indonesia (Sumatra)	Al Adawi et al. 2013
	A. mangium	Indonesia Malaysia Vietnam	
	D. sissoo	Pakistan	Al Adawi et al. 2013
	Lansium parasiticum	Indonesia	Irsan et al. 2016
	M. indica	Oman, Pakistan	van Wyk et al. 2007a
	Mimusops elengi	Thailand	Pornsuriya & Sunpapao 2015
	Prosopis cineraria	Oman	Al Adawi et al. 2013
C. mangivora	*Mangifera indica*	Brazil	Van Wyk et al. 2011a
C. neglecta	*E. grandis*	Colombia	Rodas et al. 2008
C. obpyriformis	*A. mearnsii*	South Africa	Heath et al. 2009b
C. papillata	*Annona muricata*	Colombia	Van Wyk et al. 2010b
	Citrus spp.	Colombia	Van Wyk et al. 2010b
	Coffea arabica	Colombia	Van Wyk et al. 2010b
	Schizolobium parahybum	Colombia	Van Wyk et al. 2010b
	Theobroma cacao	Colombia	Van Wyk et al. 2010b
C. pirilliformis	*Acacia mearnsii*	South Africa	Lee et al. 2016
	Eucalyptus spp.	South Africa, Malawi	Nkuekam et al. 2013
	Eucalyptus spp.	Australia	Van Wyk et al. 2013
	Rapanea sp.	South Africa	Lee et al. 2016
C. platani	*Platanus* spp.	Albania, France, Italy, Spain, USA	Tsopelas et al. 2017
	Platanus spp.	Greece, Italy, Switzerland, Turkey	Lehtijarvi et al. 2018
	Syngonium podophyllum	USA (Hawaii)	Li et al. 2017
C. polychroma	*Syzygium aromaticum*	Indonesia (Sulawesi)	Van Wyk et al. 2004
C. polyconidia	*A. mearnsii*	South Africa	Heath et al. 2009b
C. smalleyi	*Carya* spp.	USA	Johnson et al. 2005
C. tanganyicensis	*A. mearnsii*	Tanzania	Heath et al. 2009b
C. thulamalensis	*Colophospermum mopane*	South Africa	Mbenoun et al. 2014
	Combretum zeyheri	South Africa	Mbenoun et al. 2014
C. tsitsikammensis	*A. melanoxylon*	South Africa	Misse et al. 2017
	Eucalyptus sp.	South Africa	Misse et al. 2017
	Ocotea bullata	South Africa	Nkuekam et al. 2008
	Rapanea melanophloeos	South Africa	Nkuekam et al. 2008
	Terminalia sericea	South Africa	Nkuekam et al. 2008
C. variospora	*Betula platyphylla*	Japan	Linnakoski et al. 2008
	Prunus sp.	USA	Li et al. 2017
	Quercus spp.	USA	Johnson et al. 2005
C. zambeziensis	*A. nigrescens*	South Africa	Mbenoun et al. 2014
	Combretum imberbe	South Africa	Mbenoun et al. 2014
	Schotia brachypetala	South Africa	Mbenoun et al. 2014

**Ceratocystis fimbriata s.l.* has been reported from nearly 100 host species, including many woody plants, so would require a complete table of its own. Determining an accurate host range for *C. fimbriata s.s.* is likely to require additional work and the description of several new species.

described from *Eucalyptus* spp. in South Africa and South America (Table 1).

The most severe outbreaks of *Ceratocystis* wilt and canker of *Acacia* species have been in Southeast Asia where *C. manginecans* infects *Acacia* species, including *A. auriculiformis, A. crassicarpa* and *A. mangium* (Tarigan et al. 2010; Thu et al. 2012; Brawner et al. 2015). This species was first described from mango trees in Oman and Pakistan (Van Wyk et al. 2007a). Other hosts include *Dalbergia sissoo* DC. in Pakistan (Al Adawi et al. 2013), *L. parasiticum* in Indonesia (Irsan et al. 2016), *Mimusops elengi* L. in Thailand (Pornsuriya & Sunpapao 2015) and *Prosopis cineraria* (L.) Druce in Oman (Al Adawi et al. 2013). Infections of *Eucalyptus* spp. also occur in Indonesia but the species identification has not yet been confirmed (Indrayadi pers. comm.). *Ceratocystis fimbriata* has recently been identified on *Eucalyptus* in Pakistan (Alam et al. 2017), but this identification did not rely on DNA sequencing.

Many of the recently described *Ceratocystis* species lived in equilibrium with indigenous hosts in their native environment and attracted little scientific attention until they moved onto a susceptible, exotic host species (Baker et al. 2003). Subsequent isolation and DNA sequencing enabled identification as novel species (Barnes et al. 2003a), for example, *Ceratocystis fimbriotomima* M. van Wyk & M.J. Wingf. in Venezuela that infected introduced *Eucalyptus* spp. as well as native species (van Wyk et al. 2009a). Similarly, *Ceratocystis colombiana* M. van Wyk & M.J. Wingf. that infects native trees in Colombia was identified as a new pathogen infecting introduced coffee, cacao and citrus (van Wyk et al. 2010b), and *Ceratocystis ficicola* Kajitani & Masuya that infects native trees in Japan, as a new pathogen that infects *Ficus drupacea* Thunb. (syn. *Ficus indica* L.) (Kajitani & Masuya 2011). Morphologically these three species are very close to *C. fimbriata*; support for their separation into distinct taxa depends on comparison of carefully selected DNA sequence data (van Wyk et al. 2010b; Kajitani & Masuya 2011).

Discrimination among many *Ceratocystis* species is highly dependent on an enthusiastic application of phylogenetic species recognition concepts where every grouping that forms a well-supported clade is deemed to be a separate species. There is a risk that this approach may identify geographically or environmentally isolated sub-populations as distinct species (Harrington et al. 2014; Fourie et al. 2015). While these new 'species' are shown to be distinct, host-adapted lineages, it is not clear whether they fit the classical biological species concept (Oliveira et al. 2015); some may be in the process of speciation (Baker et al. 2003). Fungi with both sexual and asexual methods of reproduction can have complicated population genetics that may lead to confusion over species boundaries, e.g. *C. acaciivora*. The ITS region is commonly used for phylogenetic analyses, but this region can produce misleading results in *Ceratocystis* species; a single haploid isolate has been shown to contain two widely divergent ITS sequences (Harrington et al. 2014).

Mating studies have confirmed species boundaries among *C. fimbriata s.s., C. cacaofunesta* and *C. platani* (Engelbrecht & Harrington 2005), with interspecific pairings mainly infertile, though some produced perithecia either lacking ascospore masses or with transparent or milky ascospore masses and the few, if any, ascospores observed were misshapen. Few 'interspecific' mating tests have been conducted in *Ceratocystis*, but success in mating *C. manginecans* with *C. fimbriata s.s.* has been demonstrated (Fourie et al. 2018). Of the 70 hybrid offspring produced, 11 were pathogenic to sweet potato, seven were pathogenic to *A. mangium* and three were pathogenic to both. The authors concluded that host specificity was governed by a small number of genes.

Symptoms

Rot in roots or stems, vascular wilt, sapwood discolouration and cankers are symptomatic of plants infected by *Ceratocystis* species (Kile 1993). Vascular wilt, wood stain and stem cankers are the most characteristic symptoms of infection by *Ceratocystis* species in woody trees (Harrington 2013). Tuber crops such as *I. batatas* and *Colocasia esculenta* commonly exhibit black rot symptoms (Muramoto et al. 2012; Harrington et al. 2015).

The sudden wilting of leaves is the earliest visible symptom associated with 'true' vascular wilt. In this case, the pathogen travels through the non-living water-conducting vessels and tracheids, colonising the host away from the wound. An example is *B. fagacearum* that attacks *Quercus* species (Kile 1993). This wilting develops as the hyphae grow through and then plug the vessels, blocking the conducting system above the site of infection, thereby desiccating the plant (Mace et al. 1981).

In *Ceratocystis* infections, sapwood discolouration is induced after the pathogen attacks living parenchyma cells (Harrington 2007). Staining or sap streaks can start from where spores have invaded freshly damaged tissue; they rapidly germinate and colonise the xylem and phloem (Johnson et al. 2005), absorbing nutrients from the xylem parenchyma (Mace et al. 1981). The discolouration is caused by a combination of host response chemicals and the pigmentation of the spores and hyphae of the *Ceratocystis* (Harrington 2013). Once present in the vascular cylinder, the hyphae of *Ceratocystis* species can then move systematically into the cambium and inner bark; killing these tissues causes a canker (Kile 1993; Harrington 2013).

In Acacias, *Ceratocystis* species cause vascular stain, canker on the stem and eventually wilting. Once infected, the symptoms are first expressed as black or red lesions on the bark, blackened streaks within vascular tissue or sapwood discolouration (Fig. 1). Cankers on the stem and cracked or sunken bark above cankers emerge as further symptoms. This is followed with yellowing of the leaves, wilting, and death of the tree due to lack of nutrient supply (Roux et al. 2001b; Tarigan et al. 2011a; Brawner et al. 2015). The foam or fermentation exudate of yeasts or bacteria also often emerge from the lesions or from entrance holes made by stem borer or fungal feeding insects near to stem cankers (Fig. 1). In particular, this exudate attracts fungivorous nitidulid beetles (Coleoptera: Nitidulidae) which are associated with fungal dispersal (Brawner et al. 2015).

Epidemiology

Disease spread depends upon inoculum production and dispersal, the presence of susceptible hosts and suitable environmental conditions. *Ceratocystis* species produce several different kinds of spore inoculum and exploit a range of

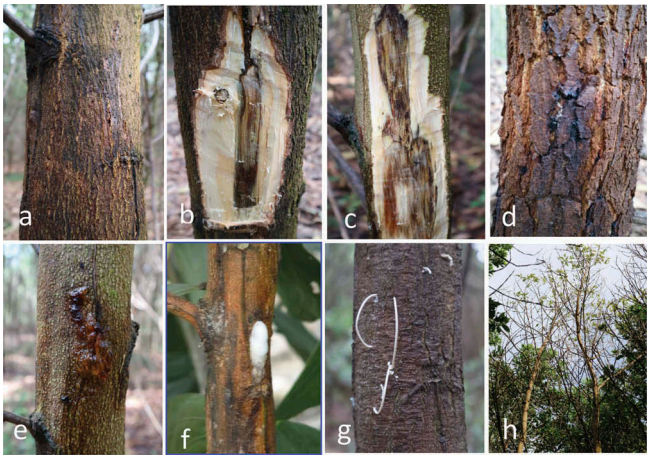

Figure 1. *Ceratocystis* wilt and canker disease symptoms on *Acacia mangium*. External discolouration (a); internal discolouration (b, c); canker on stem (d); gummosis (e); fermented exudate with fruity odours (f); frass indicating activity of boring insects (g); and yellowing leaves and dieback of tree (h) (Photos: Aswardi Nasution 2017)

dispersal mechanisms which may vary according to the type of spores produced. Like many filamentous fungi, the spores of *Ceratocystis* species infect their hosts through an infection court, often a wound, where the spores germinate after deposition (Kile 1993; Harrington 2013); wounding is therefore critical for infection to occur. Root grafting may also contribute to disease development (Harrington 2013). For woody trees, the most common mechanism of disease spread is through spore dispersal, vector activity and mechanical transmission. Spore types may include endoconidia, aleurioconidia, chlamydospores (formed by thickening of the walls of endoconidia) and ascospores. The first three are asexual spores while ascospores are the products of sexual recombination. Sporulating mats on exposed wood or in the bark of infected trees produce all spore types (Friday et al. 2016) while aleurioconidia are particularly abundant in the infected wood (Araujo et al. 2014a). Conidia and ascospores have a short period of viability whereas aleurioconidia have thicker, more durable walls and can likely survive for years, especially inside wood, and increase the likelihood of the disease being soil-borne (Paulin-Mahady et al. 2002).

Aleurioconidia are typically soil-borne, though may also be present in insect frass which is ejected from trees and wind-dispersed (Souza et al. 2013). Species of *Ceratocystis* and *Thielaviopsis* produce aleurioconidia and are known soil-borne pathogens (Harrington 2007). The thick-walled aleurioconidia can survive for years in the soil (Upadhyay 1981). Subsequent germination then has the potential to infect new plant material brought into the area (Moutia & Saumtally 2001; Marín Montoya & Wingfield 2006). The status of *C. manginecans* as a soil-borne pathogen is unclear: it has been isolated from soil in Vietnamese Acacia plantations (Thu et al. 2016) and attempts to isolate from soil in Indonesian plantations have recently been successful (Marthin Tarigan pers. comm.), though DNA sequencing is yet to confirm accurate identification of the soil isolates. Ascospores produced by fruiting bodies on woody stems or branches may be transmitted through an insect vector, mechanical tools, wind and water (Kile 1993).

Disease spread can also occur via natural root grafts between infected and healthy roots, a phenomenon that occurs in *Quercus rubra* L. and that facilitates the transmission of *B. fagacearum* without first passing through the root collar (Juzwik 2008). This same pathogen was isolated from around one quarter of natural root grafts in *Quercus ellipsoidalis* E.J. Hill, the rate of transmission being influenced by host density, soil depth, soil texture and the occurrence of other species (Juzwik et al. 2011).

Dispersal by water was reported by Vigouroux and Stojadinovic (1990) who established that wounded roots of *Platanus orientalis* L. were infected by *C. platani* spores carried in irrigation water. Dispersal by wind or water occurs more often in the asexual stage because conidia have rounded surfaces, allowing easy removal from the conidiophores. Conversely in the sexual stage, ascospores are produced on perithecia and held together by a sticky, hydrophobic matrix; their concave surfaces also promote adherence to each other (Malloch & Blackwell 1993). The sticky matrix facilitates dispersal by insects or other vectors though reduces transmission by wind and rain, at least when the intensity of wind and rain are low (Harrington 2007).

Wounds created by insects, animals, or human activities through the use of pruning implements or tapping knives, are the most common infection courts for *Ceratocystis* spp. (Kile 1993; Brawner et al. 2015). For example, monkeys strip bark from *A. mangium* to feed on the sweet-tasting cambium and outer wood of young trees (Hardie et al. 2017). The wounds created act as the initial entry point for the fungal spores into the plant tissues. Direct transmission occurs via known associations with fungal feeding insects (Kile 1993; Harrington 2013), particularly nitidulid beetles and flies (Diptera) (Heath et al. 2009b), which then play an important role as disease vectors (Harrington 2007). These insects feed on mycelial mats in the infected plants which contain abundant perithecia where ascospores are produced (Harrington 2007).

The relationship between fungal feeding insects and *Ceratocystis* is facilitated by the production of fruity odours by *Ceratocystis* and a fermentation exudate (Kile 1993; Harrington 2007; Brawner et al. 2015). The fruity aromas contain fatty acids and esters that are toxic to insects which do not rely on fungi for their nutrition. Only insects with a high tolerance of mycotoxins, like nitidulid beetles, are attracted to and feed on the mycelial mats of *Ceratocystis* spp. (Kile 1993; Dowd 1995). When the insects feed on the fungi, the sticky ascospores adhere to their bodies; these spores are then transmitted to healthy trees with fresh wounds (Heath et al. 2009a). However, even though the relationship between these insects and mycelial mats is well-recognised, most species of *Ceratocystis* do not have specific insect vectors for their dispersal (Kile 1993).

Although disease transmission in Acacia plantations can be caused by all or a combination of the above mechanisms, the initial development of the disease in Indonesia was linked to singling and removal of lower branches to facilitate silvicultural operations (Anthony Francis pers. comm.). *Acacia mangium* trees were singled in plantations at age 4–8 months to reduce the incidence of multiple stems and to increase tree diameter. An investigation showed that these practices

increased *Ceratocystis* transmission and significantly increased rates of tree death (Tarigan et al. 2010).

Biosecurity

Many *Ceratocystis* species represent biosecurity threats to regions where they are not already present, including to Australian forestry and horticulture. *Ceratocystis manginecans* poses a threat to the forestry and horticulture sectors, though the threat to Acacias and Eucalypts in natural ecosystems is unclear as the susceptibility of plantation Acacias and mangoes is undoubtedly enhanced by planting at close spacing in large monocultures. The close proximity of Indonesia to Australia has encouraged a high level of tourist traffic and carved wooden products are popular souvenirs. *Ceratocystis*, though not identified to species level, has been isolated from Balinese wooden statues carved from Acacia wood (Proborini 2016).

Similarly, *Ceratocystis* species infecting Eucalypts pose a threat to Australian forestry and potentially to natural ecosystems. The threat to horticulture is less clear as the mechanism for host adaptation has not been elucidated, though recent studies indicate that a small number of genes are involved (Fourie at al. 2018). Species infecting Eucalypts include *C. curvata, C. diversiconidia* and *C. ecuadoriana*, all present in Colombia and Ecuador, *C. neglecta* in Colombia, *C. eucalypticola, C. pirilliformis* and *C. tsitsikammensis* in South Africa, *C. fimbriatomima* in Venezuela, and *C. fimbriata s.l.* in Brazil (Table 1). The host ranges of these species have not been delineated though *C. fimbriata* has an extremely broad host range in Brazil. Biosecurity is underpinned by the knowledge of which plant pathogenic species occur in which areas, thus taxonomic certainty, or at least agreement, is critical to biosecurity processes. The name of a pathogen is expected to provide information as to which host plants may be threatened should the pathogen invade a new region; thus, regulations are generally invoked against pathogens at the species level. This is a strong driver for taxonomic research and delineation of new fungal species. The picture can become a little blurred when a genetically diverse species is composed of strains adapted to different hosts, such as occur in *Austropuccinia psidii* (Graça et al. 2013), *C. fimbriata s.l.*, (Harrington et al. 2014) and even in *C. manginecans*, which causes a severe disease of mango trees in Oman but not in Indonesia, where it is prevalent on Acacias. It is unclear whether this disparity in host range is due to environmental, genetic or epigenetic factors. A similar puzzle is posed by *A. psidii* in Brazil; in this case, the different host-adapted lineages occur in the same geographic location and can be distinguished only by microsatellite analyses (Graça et al. 2013; Stewart et al. 2018).

Disease management and control

Strategies for managing forest diseases and their vectors parallel those used in agriculture: avoidance, exclusion, eradication, protection, host resistance, curative treatments and integrated management (Edmonds 2013; Harrington 2013). However, as plantation forests are usually grown in monoculture over larger areas for several years before harvest and attain a much greater biomass than agricultural crops, the range of viable strategies is often more limited. For environmental and economic reasons, control using pesticides is rarely used (Wingfield et al. 2001a; Harrington 2013). As is the case for Acacia in Southeast Asia, the species planted is often an exotic, and *Ceratocystis* has emerged as a new disease. As a result, disease management strategies must first be based on a clear understanding of the biology of the host and the behaviour of the pathogen (Wingfield et al. 2001a).

Detection, surveying and monitoring

Diseases can be detected from an on-ground examination of individual trees (Bechtold & Patterson 2005; Carnegie et al. 2018), or at the landscape level using aerial photography and aerial sketch mapping, satellite imagery and LiDAR (Stone & Mohammed 2017). Landscape-level detection can be followed up by ground-based surveying and monitoring. For *Ceratocystis* wilt disease, ground monitoring is an important first step in developing strategies for its control. Importantly, it is also necessary for detecting the presence of insect vectors (Juzwik 2008; Heath et al. 2009a) as there can be a significant correlation between vector activity and disease severity (Hayslett et al. 2008).

Remote sensing and geographic information systems (GIS)

Remote sensing of forest diseases has been applied since aerial photography first became available in the 1930s (Coppin & Bauer 1996). Combined with GIS and ground-based surveys, both spatial coverage and objective assessments of forest health can be realised (Wulder et al. 2006). Remote sensing using Colour Infra-Red (CIR) digital imagery detected oak wilt disease caused by *B. fagacearum* (Everitt et al. 1999). The imagery was first interpreted from the radiometric reflectance measurements and then verified by ground observations. To distinguish between healthy, infected and dead oak trees, the data was linked to leaf chlorophyll concentration using three spectral bands: visible green (0.52–0.60 μm), visible red (0.63–0.69 μm) and near-infrared (0.63–0.69); infected trees had veinal necrosis and tip burn (Everitt et al. 1999). In a second study (Souza et al. 2015), a camera mounted in an unmanned aerial vehicle (UAV) detected wilt disease caused by *C. fimbriata* on *Eucalyptus* spp. The images were analysed and compared using four distinct machine learning techniques: K-Nearest Neighbours (K-NN), Random Forest (RF), Artificial Neural Network (ANN) and Gaussian Processes (GP). The last, which is a Bayesian non-parametric tool that learns the input-output transformation function based on training data, was best able to reliably and accurately distinguish between healthy and infected trees, and was suitable for detecting wilt disease in other species and other diseases affecting large-scale plantations. A spectral signature of *Ceratocystis*-infected *Metrosideros polymorpha* has been detected at leaf and canopy level and provides a basis for mapping and monitoring of disease spread in Hawaii (Asner et al. 2018). A clear timeline of symptom development is the first requirement for aerial assessment of *Ceratocystis* disease in Acacias. A preliminary study indicates that a reduction in leaf area index is associated with early external visible symptoms of *Ceratocystis* disease in *A. mangium* (Nasution unpubl.) and this could be exploited for aerial detection.

Silvicultural and chemical control of *Ceratocystis*

The most frequently suggested strategy for preventing infection by *Ceratocystis* is wound avoidance (Kile 1993; Harrington 2013), and efforts to manage *Ceratocystis* wilt and canker disease in Acacia plantations have focused on better managing the causes of wounding, minimising wound size and limiting pruning and singling activities to periods when insect vectors are less active (Heath et al. 2009a). The role of wounds in disease epidemics is well-established, particularly when linked to pruning (Hayslett et al. 2008; Roux & Wingfield 2009). In Indonesia, wilt and canker symptoms and mortality in *A. mangium* plantations caused by *C. manginecans* occurred after trees were singled at 6–8 months old using poor pruning practice (Tarigan et al. 2010; Tarigan et al. 2011b). Timing of singling or pruning may also be important, with lower disease incidence if conducted during dry than wet weather (Pilotti et al. 2016). Climate is also correlated with insect-vector activity; in South Africa beetle activity is much higher during spring and early summer than winter (Heath et al. 2009a). Silvicultural interventions that lead to wounds should therefore be avoided during periods of high insect activity (Hayslett et al. 2008; Heath et al. 2009b). In tropical Southeast Asia, nitidulid and ambrosia beetles are present in Acacia plantations and considered important vectors of *C. manginecans*, though their activity has not been monitored (Tarigan et al. 2011a; Thu et al. 2012; Brawner et al. 2015). There is an expectation that insect activity will occur year round (Wolda 1988) though, and because of the dynamics of parasitism and plant investment in anti-herbivore defences, populations may be higher during the dry than wet season (Dyer et al. 2012).

Wound dressings provide a physical barrier to *Ceratocystis* infection as well as inhibiting fungal growth (Harrington 2013) and low toxicity treatments have been developed. Application of latex paint to pruning wounds in *Quercus* spp. has been shown to be non-toxic and to reduce wilt disease (French & Juzwik 1999; Camilli et al. 2007).

Removal of symptomatic trees and stumps as well as neighbouring trees that may have been infected by *Ceratocystis* may help to limit disease spread (Kile 1993). This approach aims to minimise the inoculum load by reducing the potential for fungal dispersal (Kile 1993; Harrington 2013).

Stem injecting 20 ml of 14.3% propiconazole into the root collar of young *Q. rubra* two weeks before inoculation by *B. fagacearum* delayed disease development and extended the life of infected trees for at least two years (Blaedow 2009). Two years after injection, the fungicide could still be detected in the primary root but its concentration decreased with distance from the injection point which would have reduced its ability to combat the infection (Blaedow 2009). Chemical control can be a high-cost strategy, however and as in this case, may only delay the expression of disease symptoms; the fungicides may also be toxic to other organisms. The use of fungicides in this way, as well as wound dressings and removal of symptomatic trees and stumps are also not suitable for large-scale plantation estates being managed for pulpwood. Because of these limitations, the selection of disease resistant and tolerant host materials is currently considered the most effective and economic strategy for managing *Ceratocystis* disease (Kile 1993).

Genetic control of *Ceratocystis*

The first line of defence to pest and disease invasion is avoidance which includes physical barriers such as hairs, thorns and resin ducts; these generally work as a defence mechanism against animal attack (Vale et al. 2001) and may decrease the incidence of wounding caused by animals. Resistance and tolerance mechanisms come into play after contact has been established and are more common for protecting plants from infection by fungal pathogens (Vale et al. 2001). Schafer (1971) has defined tolerance as the ability of a cultivar to minimise loss of yield or quality, in comparison with other cultivars, after pathogen infection, whereas resistance is the ability of a plant to overcome the infection process and prevent development of the pathogen, resulting in a disease-free plant. Resistance is considered more important for plant defence than avoidance and tolerance (Vale et al. 2001) and the best way to deal with plant disease problems in forest trees, including *Ceratocystis* (Wingfield et al. 2001a; Harrington 2013). Such an approach requires genetic variation within the host species that has the potential to be exploited to increase the resistance of trees to disease through breeding. A successful example is *Platanus acerifolia* (Aiton) Willd. (London plane); trees resistant to *C. platani* that remain free of canker and sap stain symptoms were obtained in this way (Harrington 2013). The adoption of mango cultivars resistant to *C. fimbriata* has been the most effective strategy for disease control in mangoes in Brazil (Araujo et al. 2014b). In Eucalypts, four out of 18 commercial clones of *E. grandis* × *Eucalyptus urophylla* S.T. Blake hybrid expressed no discolouration symptoms on the stem and showed an ability to overcome fungal infection caused by *C. fimbriata* (Zauza et al. 2004).

Genetic resistance to disease in plants can be monogenic and controlled by a single major gene which is race-specific and relatively easy to identify and manipulate in a breeding program; it can be expected to provide complete resistance (Poland et al. 2009), but this only provides protection against a specific strain or race of the pathogen; within a short time, it may easily be broken down (Maramorosch & Loebenstein 2009). Genetic resistance can also be polygenic and controlled by a number of genes (Brun et al. 2010). This provides a similar level of protection against all the races of a pathogen, although it may be difficult to identify and manipulate the genes in a breeding program to confer this type of resistance; if successful it is not easily broken down by the pathogen and should give the host long-lasting protection (Lindhout 2002).

Polygenic resistance is better suited to Acacia forestry as breeders work with populations rather than genotypes of both host and pathogen in order to protect a range of genotypes of a given host species from all known races of a pathogen (Carson & Carson 1989). As plantation growing cycles are usually several years, polygenic resistance against disease is expected to have a long-term utility (Sniezko 2006). The development of resistant clones may be expedited by mapping of quantitative trait loci, as has been done for *Ceratocystis* in Eucalypts in Brazil (Rosado et al. 2016).

To source the breeding material requires selecting a range of phenotypes in natural forests or forest plantations and conducting pathogenicity tests either *in situ* or, more commonly, by using artificial inoculation under controlled conditions (Sniezko

2006; Tarigan et al. 2010). This enables selection of putative genes that may confer disease resistance before the breeding program commences. This is currently considered the most effective method for controlling *Ceratocystis* vascular wilt and canker diseases in forest plantations (Kile 1993; Wingfield et al. 2001a). The trees with the highest rates of survival and lowest levels of disease symptoms are selected for inclusion in breeding programs (Sniezko 2006).

Inoculation tests *in situ* (natural) can provide the best information for a given location because the influence of the natural environment on host-pathogen interactions is captured (Brawner et al. 2015). However, there is inevitably a risk of disease spread from the introduced pathogen. This approach also first requires trees rather than seedlings, which slows the screening process (Tarigan et al. 2010; Sniezko & Koch 2017).

Artificial inoculations using fungal isolates are conducted by wounding tree stems or branches before placing a plug of mycelium or spore suspension of the pathogen under the bark. This is normally carried out on juvenile plant materials under controlled conditions (Green et al. 1985; Tarigan et al. 2010). Levels of resistance to *Ceratocystis* wilt and canker disease are evaluated by measuring the length of xylem discolouration (Zauza et al. 2004). This screening method is time-consuming as disease resistance is only expected to occur in a low percentage of the population. As large numbers of cultivars or clones must be used, rapid screening technologies are required (Brawner et al. 2015). One possible option for *Ceratocystis* is inoculation of detached leaves; this has been shown to give a similar result to tree wounding in screening assays using leaves of *Castanea dentata* (Marshall) Borkh., *Castanea molissima* Blume and *Castanea pumila* (L.) Mill. against *Cryphonectria parasitica* (Murrill) M.E. Barr (the chestnut blight pathogen; Newhouse et al. 2014). This approach is currently being tested for *Acacia* (Nasution et al. 2016b).

To date, no strains of *A. mangium* resistant to *Ceratocystis* have been identified, though there is evidence of some variation in disease tolerance (Brawner 2015). *Acacia auriculiformis* shows higher levels of tolerance to *C. manginecans*, and this tolerance may also be expressed in *A. mangium* × *A. auriculiformis* hybrids (Trang et al. 2018).

Biological control

Biological control is the use of fungi, bacteria, actinomycetes and viruses to suppress or decrease the population density of a plant pathogen (Bale et al. 2008). These microorganisms or BCAs may also have beneficial interactions internally or externally with their host plants (McInroy & Kloepper 1994). The attraction of biological control is that it can be applied to broad or narrow targets depending on the biocontrol organism, is less site-specific and less prone to build-up of resistance, and is cost-effective for specialised applications; it can also be integrated with other control strategies, and may enhance plant growth (Whipps & Lumsden 2001).

In practice, there are few examples of successful biological control in forest trees (Wingfield et al. 2001a; Garnas et al. 2012). This may be related to their large biomass, complex anatomy and longevity. The inoculum load of the pathogen also has an opportunity to build up during each rotation. Thus, repeated infection through a large root system by a soil borne pathogen allows the disease to persist in soils as dormant, quiescent or resistant propagules such as chlamydospores, aleurioconidia or microsclerotia after tree death and after the trees are harvested (Cazorla & Mercado-Blanco 2016). These propagules can then infect new trees or any root tissues that are not reached by the BCA, thereby reducing its effectiveness.

A notable success story has been use of the fungus *Phlebiopsis gigantea* (Fr.) Jülich for controlling root-rot disease in pine trees caused by *Heterobasidion annosum* (Fr.) Bref. This fungal pathogen can colonise and survive in the stumps of harvested trees which then act as an inoculum source. Spores of *P. gigantea* applied to the surface of freshly cut stumps rapidly germinate and colonise the wood, preventing subsequent growth of *H. annosum* and thus reducing disease incidence in the next rotation (Rishbeth 1979). A commercially produced BCA based on *P. gigantea* is used routinely to control root-rot disease in pine plantations (Pratt et al. 2000). While a potential *Phlebiopsis* sp. has been isolated that shows a microparasitic reaction against *G. philippii*, a root-rot pathogen that kills *A. mangium* (Agustini et al. 2014), further refinement of application methods is required to commercialise this fungus as a BCA.

Do BCAs have any potential application in controlling vascular wilt diseases? Injection of 100–400 ml (10^8 cells ml^{-1}) of *Pseudomonas syringae* Van Hall using a specially developed 'gouge-pistol' into the trunk base of *Ulmus* sp. suppressed the expression of *Ophiostoma ulmi* (Buisman) Melin & Nannf. (Dutch elm disease) and resulted in a high proportion of healthy trees (Scheffer 1983). However, due to a lack of correlation between *in vitro* and field tests, it was concluded that *P. syringae* had triggered plant defence against *Ceratocystis* infection through induced resistance rather than antagonism (Scheffer 1990; Scheffer et al. 2008). Another potential BCA, *Verticillium dahliae* Kleb. isolate WCS850 was injected into mature clonal trees of *Ulmus minor* Mill., (syn. *Ulmus carpinifolia* Rupp. Ex Suckow) that were either resistant or susceptible to *C. ulmi* resulting in disease suppression in susceptible as well as resistant trees (Scheffer 1990). Injecting *V. dahliae* isolate Vd-48 into 6–7-year-old *U. minor* reduced wilting symptoms associated with *Ophiostoma* infection (Solla & Gil 2003). In both studies *V. dahliae* could be re-isolated only from at or near the injecting point, an indication that translocation of this fungus had been minimal and any contact with *C. ulmi* unlikely. Thus, it was concluded that *V. dahliae* acts by triggering host defences against *Ceratocystis* infection.

Bacteria that live internally in plant tissue without causing any negative impact on their host are recognised as endophytic bacteria (Schulz & Boyle 2006). Some have mutualistic relationships with their host plants and an ability to live as obligate or facultative endophytes at different stages in their life cycle (Hardoim et al. 2008). The majority of research into endophytic bacteria has been with agricultural and horticultural crops (Kobayashi & Palumbo 2000), nevertheless, several species of endophytic bacteria have been isolated from woody species (Kobayashi & Palumbo 2000; Bacon & Hinton 2006; Izumi 2011). The large biomass and perennial nature of trees potentially provide a stable habitat for a diverse range of endophytes (Izumi 2011) and raise the possibility that they could play a role as BCAs against vascular diseases such as *Ceratocystis* (Yadeta & Thomma 2013).

Bacterial endophytes suppress plant diseases through the production of enzymes, antifungal and antibacterial compounds (allellochemicals), by competition with pathogens for nutrients or niches and stimulation of induced systemic resistance (ISR) (Compant et al. 2005; Bacon & Hinton 2006).

The main focus for the beneficial effects of endophytic bacteria in woody trees has been their role in promoting growth; it just happens that plant growth promoting rhizobacteria also produce enzymes and antibiotics that can prevent or reduce pathogen infection. In this way rhizobia can play an indirect role as BCAs (Ryan et al. 2008). However, there are few reports of endophytes acting as BCAs in forest trees (Chanway 1998). This delay in their development and application is in part because their performance in the field has not matched their apparent potential when tested in assay. Thus, a selected endophyte may be replaced by or act differently in the presence of other endophytes already present in the host. To be effective, assay-based selection techniques are required that capture the environment where the introduced endophyte is to be inoculated (Newcombe 2011; Hilszczańska 2016).

Nevertheless, a potential for using endophytes in managing vascular diseases in forest trees has been demonstrated. As both endophytes and pathogen live in the vascular system, endophytic bacteria can potentially act as antagonists (Yadeta & Thomma 2013). Application of *Pseudomonas fluorescens* Migula and *Psuedomonas putida* (Trevisan) Migula into the roots of *E. urophylla* reduced by 45% the incidence of bacterial wilt caused by *Ralstonia solanacearum* (Smith) Yabuuchi et al. emend Safni et al. (Ran et al. 2005). Endophytic bacteria have been applied to control wilt disease caused by *B. fagacearum*. A total of 889 endophytic bacterial isolates were obtained from healthy *Quercus fusiformis* Small; 183 isolates of mostly *Bacillus* spp. and *Pseudomonas* spp. showed high inhibition through chitinase activity against *B. fagacearum* in an *in vitro* assay. Two species, *Pseudomonas denitrificans* (*nomen rejiciendum*) isolate 1–15 and *P. putida* isolate 5–48 were selected and injected into potted oak trees which were then inoculated with a spore suspension of *B. fagacearum*. The experiment showed that *P. denitrificans* 1–15 was able to reduce disease incidence by 50% and the proportion of crown loss by 17% (Brooks et al. 1994).

This indicates that there may be potential for exploiting endophytes in the control of *Ceratocyctis* wilt and canker diseases in Acacia plantations. Their slow movement in plant tissues is a potential problem as contact with the pathogen may be limited in an organism as large as a tree (Hallmann et al. 1997). However, the ability of endophytic bacteria to elicit ISR in plants or to produce inhibitory secondary compounds suggests this may be a complementary approach to breeding strategies (Percival 2001; Hossain et al. 2016). Endophytic bacteria that produce secondary metabolites which restrict the growth of *C. manginecans in vitro* have been isolated from *A. mangium* and methods for inoculating seedlings or cuttings are under investigation (Nasution et al. 2016a).

Preliminary investigations into application of endophytic bacteria and fungi as external treatments for the control of *Ceratocystis* wilt and canker diseases in Acacia plantations have also been carried out (Tran et al. 2018). In this study, treatments were applied externally following inoculation of one-year-old seedlings with *C. manginecans*. All three biological products reduced the disease index compared to controls after 40 days, though none were as effective as the fungicides tested.

Conclusion

An integrated approach incorporating several strategies is necessary to make *A. mangium* once again a viable plantation species in Southeast Asia. The development of hybrid Acacias that retain the growth and pulping properties of *A. mangium* but incorporate the increased levels of resistance to *C. mangenicans* demonstrated by *A. auriculiformis* is one approach. Appropriate silvicultural techniques, augmented by deployment of BCAs would provide an extra level of protection that may keep pathogen inoculum levels low and thus help extend the lifespan of any resistant clones that may be developed. Some progress towards each of these aims is being made in Indonesia and Vietnam with the support of ACIAR.

Acknowledgements

Aswardi Nasution is the recipient of a John Allwright Fellowship. This work was supported by ACIAR project FST2014–068, Management strategies for Acacia plantation diseases in Indonesia and Vietnam.

Disclosure statement

No potential conflict of interest was reported by the authors.

Funding

This work was supported by the Australian Centre for International Agricultural Research [FST/2014/068].

References

Agustini L, Wahyuno D, Indrayadi H, Glen M. 2014. In vitro interaction between *Phlebiopsis* sp. and *Ganoderma philippii* isolates. Forest Pathology. 44:472–476.

Al Adawi A, Barnes I, Khan I, Al Subh A, Al Jahwari A, Deadman M, Wingfield B, Wingfield M. 2013. *Ceratocystis manginecans* associated with a serious wilt disease of two native legume trees in Oman and Pakistan. Australasian Plant Pathology. 42:179–193.

Alam MW, Rehman A, Iqbal M, Saira M, Aslam S, Muhammad S, Hameed A, Gleason ML. 2017. First report of *Ceratocystis fimbriata* causing *Eucalyptus* wilt in Pakistan. Plant Disease. 101:1050.

Anderson PK, Cunningham AA, Patel NG, Morales FJ, Epstein PR, Daszak P. 2004. Emerging infectious diseases of plants: pathogen pollution, climate change and agrotechnology drivers. Trends in Ecology & Evolution. 19:535–544.

Araujo L, Bispo WMS, Cacique IS, Moreira WR, Rodrigues FÁ. 2014a. Resistance in mango against infection by *Ceratocystis fimbriata*. Phytopathology. 104:820–833.

Araujo L, Bispo WMS, Cacique IS, Cruz MFA, Rodrigues FA. 2014b. Histopathological aspects of mango resistance to the infection process of *Ceratocystis fimbriata*. Plant Pathology. 63:1282–1295.

Asner GP, Martin RE, Keith LM, Heller WP, Hughes MA, Vaughn NR, Hughes RF, Balzotti C. 2018. A spectral mapping signature for the rapid ohia death (ROD) pathogen in Hawaiian forests. Remote Sensing. 10:404–414. doi: 10.3390/rs10030404.

Bacon CW, Hinton DM. 2006. Bacterial endophytes: the endophytic niche, its occupants, and its utility. In: Gnanamanickam SS, editor. Plant-associated bacteria. Dordrecht: Springer Publishing; p. 155–194.

Baker CJ, Harrington TC, Krauss U, Alfenas AC. 2003. Genetic variability and host specialization in the Latin American clade of *Ceratocystis fimbriata*. Phytopathology. 93:1274–1284.

Bale J, Van Lenteren J, Bigler F. 2008. Biological control and sustainable food production. Philosophical Transactions of the Royal Society B: Biological Sciences. 363:761–776.

Barnes I, Fourie A, Wingfield MJ, Harrington TC, McNew DL, Sugiyama LS, Luiz BC, Heller WP, Keith LM. 2018. New *Ceratocystis*

species associated with rapid death of *Metrosideros polymorpha* in Hawai`i. Persoonia. 40:154–181.

Barnes I, Nakabonge G, Roux J, Wingfield B, Wingfield M. 2005. Comparison of populations of the wilt pathogen *Ceratocystis albifundus* in South Africa and Uganda. Plant Pathology. 54:189–195.

Barnes I, Roux J, Wingfield B, O'Neill M, Wingfield M. 2003b. *Ceratocystis fimbriata* infecting *Eucalyptus grandis* in Uruguay. Australasian Plant Pathology. 32:361–366.

Barnes I, Roux J, Wingfield BD, Dudzinski MJ, Old KM, Wingfield MJ. 2003a. *Ceratocystis pirilliformis*, a new species from *Eucalyptus nitens* in Australia. Mycologia. 95:865–871.

Bechtold WA, Patterson PL. 2005. The enhanced forest inventory and analysis program: national sampling design and estimation procedures, vol. 80. Asheville (NC): Southern Research Station, US Department of Agriculture Forest Service.

Blaedow RA. 2009. Use of the systemic fungicide propiconazole for oak wilt management: an assessment of uncharacterized host-pathogen-fungicide interactions [dissertation]. Minneapolis-St. Paul (MN): University of Minnesota.

Brawner J, Japarudin Y, Lapammu M, Rauf R, Boden D, Wingfield MJ. 2015. Evaluating the inheritance of *Ceratocystis acaciivora* symptom expression in a diverse *Acacia mangium* breeding population. Southern Forests. 77:83–90.

Brooks DS, Gonzalez CF, Appel DN, Filer T. 1994. Evaluation of endophytic bacteria as potential biological-control agents for Oak Wilt. Biological Control. 4:373–381.

Brun H, Chèvre A-M, Fitt BD, Powers S, Besnard A-L, Ermel M, Huteau V, Marquer B, Eber F, Renard M, et al. 2010. Quantitative resistance increases the durability of qualitative resistance to *Leptosphaeria maculans* in *Brassica napus*. New Phytologist. 185:285–299.

Camilli K, Appel DN, Watson WT. 2007. Studies on pruning cuts and wound dressings for oak wilt control. Arboriculture and Urban Forestry. 33:132–139.

Carnegie AJ, Lawson S, Wardlaw T, Cameron N, Venn T. 2018. Benchmarking forest health surveillance and biosecurity activities for managing Australia's exotic forest pest and pathogen risks. Australian Forestry. 81:14–23.

Carson S, Carson M. 1989. Breeding for resistance in forest trees – a quantitative genetic approach. Annual Review of Phytopathology. 27:373–395.

Cazorla FM, Mercado-Blanco J. 2016. Biological control of tree and woody plant diseases: an impossible task? BioControl. 61:233–242.

Chanway C. 1998. Bacterial endophytes: ecological and practical implications. Sydowia-Horn. 50:149–170.

Coetzee MPA, Wingfield BD, Golani GD, Tjahjono B, Gafur A, Wingfield MJ. 2011. A single dominant *Ganoderma* species is responsible for root rot of *Acacia mangium* and *Eucalyptus* in Sumatra. Southern Forests. 73:175–180.

Compant S, Duffy B, Nowak J, Clément C, Barka EA. 2005. Use of plant growth-promoting bacteria for biocontrol of plant diseases: principles, mechanisms of action, and future prospects. Applied and Environmental Microbiology. 71:4951–4959.

Coppin PR, Bauer ME. 1996. Digital change detection in forest ecosystems with remote sensing imagery. Remote Sensing Reviews. 13:207–234.

Crous PW, Denman S, Taylor JE, Swart L, Bezuidenhout CM, Hoffman L, Palm ME, Groenewald JZ. 2013. Cultivation and diseases of Proteaceae: *Leucadendron, Leucospermum*, and *Protea*. 2nd ed. Utrecht: CBS Biodiversity Series. N/A: 360.

De Beer Z, Duong T, Barnes I, Wingfield B, Wingfield M. 2014. Redefining *Ceratocystis* and allied genera. Studies in Mycology. 79:187–219.

De Beer ZW, Marincowitz S, Duong TA, Wingfield MJ. 2017. *Bretziella*, a new genus to accommodate the oak wilt fungus, *Ceratocystis fagacearum* (Microascales, Ascomycota). MycoKeys. 27:1–19.

Dowd PF. 1995. Sap beetles and mycotoxins in maize. Food Additives & Contaminants. 12:497–508.

Dyer LA, Carson WP, Leigh EG Jr. 2012. Insect outbreaks in tropical forests: patterns, mechanisms, and consequences. In: Barbosa P, Letourneau DK, Agrawal AA, editors. Insect outbreaks revisited. Hoboken (NJ): Wiley-Blackwell; p. 219–245.

Edmonds RL. 2013. General strategies of forest disease management. infectious forest diseases. Wallingford: CABI; p. 29–49.

Engelbrecht CJ, Harrington TC, Alfenas AC, Suarez C. 2007b. Genetic variation in populations of the cacao wilt pathogen, *Ceratocystis cacaofunesta*. Plant Pathology. 56:923–933.

Engelbrecht CJB, Harrington TC. 2005. Intersterility, morphology and taxonomy of *Ceratocystis fimbriata* on sweet potato, cacao and sycamore. Mycologia. 97:57–69.

Engelbrecht CJB, Harrington TC, Alfenas A. 2007a. Ceratocystis wilt of cacao – a disease of increasing importance. Phytopathology. 97:1648–1649.

Everitt J, Escobar D, Appel D, Riggs W, Davis M. 1999. Using airborne digital imagery for detecting oak wilt disease. Plant Disease. 83:502–505.

Ferreira MA, Harrington TC, Piveta G, Alfenas AC. 2017. Genetic variability suggests that three populations of *Ceratocystis fimbriata* are responsible for the Ceratocystis wilt epidemic on kiwifruit in Brazil. Tropical Plant Pathology. 42:86–95.

Firmino AC, Tozze HJ Jr, Furtado EL. 2012. First report of *Ceratocystis fimbriata* causing wilt in *Tectona grandis* in Brazil. New Disease Reports. 25:24.

Fourie A, Wingfield MJ, Wingfield BD, Barnes I. 2015. Molecular markers delimit cryptic species in *Ceratocystis* sensu stricto. Mycological Progress. 14:1–18.

Fourie A, Wingfield MJ, Wingfield BD, van der Nest MA, Loots MT, Barnes I. 2018. Inheritance of phenotypic traits in the progeny of a *Ceratocystis* interspecific cross. Fungal Biology. 122:717–729.

Francis A, Beadle C, Puspitasari D, Irianto R, Agustini L, Rimbawanto A, Gafur A, Hardiyanto E, Hidyati N, Tjahjono B. 2014. Disease progression in plantations of *Acacia mangium* affected by red root rot (*Ganoderma philippii*). Forest Pathology. 44:447–459.

French D, Juzwik J. 1999. Oak wilt in Minnesota. University of Minnesota extension service, MI-3174-Z.

Friday JB, Keith L, Hughes F, Cannon P. 2016. Ceratocystis wilt of oh'ia (rapid Oh'ia death): a new disease in Hawaii. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Garnas JR, Hurley BP, Slippers B, Wingfield MJ. 2012. Biological control of forest plantation pests in an interconnected world requires greater international focus. International Journal of Pest Management. 58:211–223.

Graça RN, Ross-Davies AL, Klopfenstein N, Kim M-S, Peever TL, Cannon PG, Aun CP, Mizubuti ESG, Alfenas AC. 2013. Rust disease of eucalypts, caused by *Puccinia psidii*, did not originate via host jump from guava in Brazil. Molecular Ecology. 22:6033–6047.

Green CE, Guries R, Smalley E. 1985. Early screening of elms for resistance to *Ceratocystis ulmi*. Plant Disease. 69:60–63.

Griffin A, Midgley S, Bush D, Cunningham P, Rinaudo A. 2011. Global uses of Australian acacias – recent trends and future prospects. Diversity and Distributions. 17:837–847.

Hallmann J, Quadt-Hallmann A, Mahaffee W, Kloepper J. 1997. Bacterial endophytes in agricultural crops. Canadian Journal of Microbiology. 43:895–914.

Halsted BD, Fairchild D. 1891. Sweet-potato black rot (*Ceratocystis fimbriata* Ell& Hals.). The Journal of Mycology. 7:1–11.

Hardie M, Akhmad N, Mohammed C, Mendham D, Corkrey R, Gafur A, Siregar S. 2017. Role of site in the mortality and production of *Acacia mangium* plantations in Indonesia. Southern Forests. 80:37–50.

Hardoim PR, van Overbeek LS, van Elsas JD. 2008. Properties of bacterial endophytes and their proposed role in plant growth. Trends in Microbiology. 16:463–471.

Harrington TC. 2007. The genus *Ceratocystis*. where does the oak wilt fungus fit? In: Billings RF, Appel DN, editors. Proceedings of the 2nd National Oak Wilt Symposium; 2007 June 4–7; Austin, Texas.

Harrington TC. 2013. *Ceratocystis* diseases. In: Gonthier P, editor. Infectious forest diseases. Wallingford: CABI; p. 230–255.

Harrington TC, Huang Q, Ferreira MA, Alfenas AC. 2015. Genetic analyses trace the Yunnan, China population of *Ceratocystis fimbriata* on pomegranate and taro to populations on *Eucalyptus* in Brazil. Plant Disease. 99:106–111.

Harrington TC, Kazmi MR, Al-Sadi AM, Ismail SI. 2014. Intraspecific and intragenomic variability of ITS rDNA sequences reveals taxonomic problems in *Ceratocystis fimbriata* sensu stricto. Mycologia. 106:224–242.

Harwood C, Nambiar K. 2014. Productivity of acacia and eucalypt plantations in Southeast Asia. 2. Trends and variations. International Forestry Review. 16:249–260.

Hayslett M, Juzwik J, Moltzan B, Appel D, Camilli K. 2008. Insect vectors of the oak wilt fungus in Missouri and Texas. In: Proceedings of the 2nd National Oak Wilt Symposium; Austin (Texas). p. 109–120.

Heath RN, Wingfield MJ, Van Wyk M, Roux J. 2009a. Insect associates of *Ceratocystis albifundus* and patterns of association in a native savanna ecosystem in South Africa. Environmental Entomology. 38:356–364.

Heath RN, Wingfield MJ, Wingfield BD, Meke G, Mbaga A, Roux J. 2009b. *Ceratocystis* species on *Acacia mearnsii* and *Eucalyptus* spp in eastern and southern Africa including six new species. Fungal Diversity. 34:41–67.

Hilszczańska D. 2016. Endophytes – characteristics and possibilities of application in forest management. Forest Research Papers. 77:276–282.

Hossain MT, Chung EJ, Chung YR. 2016. Biological control of rice bakanae by an endophytic *Bacillus oryzicola* YC7007. The Plant Pathology Journal. 32:228–242.

Huang Q, Zhu Y, Chen H, Wang Y, Liu Y, Lu W, Ruan X. 2003. First report of pomegranate wilt caused by *Ceratocystis fimbriata* in Yunnan, China. Plant Disease. 87:1150.

Irsan C, Suwandi AKD, Umayah A, Hamidson H. 2016. *Ceratocystis* wilt of *Lansium* tree: new disease and threat to duku fruit production in Indonesia. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Izumi H. 2011. Diversity of endophytic bacteria in forest trees. In: Pirttilä AM, Frank C, editors. Endophytes of forest trees. Dordrecht: Springer; p. 95–105.

Johnson JA, Harrington TC, Engelbrecht C. 2005. Phylogeny and taxonomy of the North American clade of the *Ceratocystis fimbriata* complex. Mycologia. 97:1067–1092.

Juzwik J. 2008. Epidemiology and occurrence of oak wilt in Midwestern, Middle, and South Atlantic states. In: Billings RF, Appel DN, editors. Proceedings of the 2nd National Oak Wilt Symposium; 2007 Jun 4–7; Austin, Texas. p. 55–67.

Juzwik J, Appel DN, MacDonald WL, Burks S. 2011. Challenges and successes in managing oak wilt in the United States. Plant Disease. 95:888–900.

Kajitani Y, Masuya H. 2011. *Ceratocystis ficicola* sp. nov., a causal fungus of fig canker in Japan. Mycoscience. 52:349–353.

Keith LM, Hughes RF, Sugiyama LS, Heller WP, Bushe BC, Friday JB. 2015. First report of Ceratocystis wilt on Ohia (*Metrosideros polymorpha*). Plant Disease. 99:1277.

Kha LD. 2000. Studies on natural hybrids of *Acacia mangium* and *A. auriculiformis* in Vietnam. Journal of Tropical Forest Science. 12:794–803.

Kile G. 1993. Plant diseases caused by species of *Ceratocystis* sensu stricto and *Chalara*. In: Wingfield MJ, Seifert KA, Webber JF, editors. *Ceratocystis* and *Ophiostoma*: taxonomy, ecology, and pathogenicity. St Paul (MN): APS Press; p. 173–183.

Kobayashi D, Palumbo J. 2000. Bacterial endophytes and their effects on plants and uses in agriculture. In: Bacon CW, White J, editors. Microbial endophytes. Boca Raton (FL): CRC Press; p. 199–233.

Krisnawati H, Kallio M, Kanninen M. 2011. *Acacia mangium* Willd.: ecology, silviculture and productivity. Bogor (Indonesia): CIFOR.

Laia M, Alfenas A, Harrington T. 2000. Isolation, detection in soil, and inoculation of *Ceratocystis fimbriata*, causal agent of wilting, die-back and canker in *Eucalyptus*. In: Proceedings of the 12th Biennial Conference of the Australasian Plant Pathology Society; 1991 September 27–30; Canberra, Australia.

Lee DH, Roux J, Wingfield BD, Barnes I, Wingfeld MJ. 2016. New host range and distribution *Ceratocystis pirilliformis* in South Africa. European Journal of Plant Pathology. 146:483–496.

Lee S. 2004. Diseases and potential threats to *Acacia mangium* plantations in Malaysia. Unasylva. 217:31–35.

Lehtijärvi A, Oskay F, Doğmus-Lehtijärvi H, Aday Kaya AG, Pecori F, Santini A, Woodward S. 2018. *Ceratocystis platani* is killing plane trees in Istanbul (Turkey). Forest Pathology. 48:e12375.

Li Q, Harrington TC, McNew D, Li J. 2017. *Ceratocystis uchidae*, a new species on Araceae in Hawaii and Fiji. Mycoscience. 58:398–412.

Lindhout P. 2002. The perspectives of polygenic resistance in breeding for durable disease resistance. Euphytica. 124:217–226.

Linnakoski R, de Beer ZW, Rousi M, Niemela P, Pappinen A, Wingfield MJ. 2008. Fungi, including *Ophiostoma karelicum* sp. nov., associated with *Scolytus ratzeburgi* infesting birch in Finland and Russia. Mycological Research. 112:1475–1488.

Liu FF, Barnes I, Roux J, Wingfield MJ, Chen SF. 2018. Molecular phylogenetics and microsatellite analysis reveal a new pathogenic *Ceratocystis* species in the Asian-Australian clade. Plant Pathology. 67:1097–1113.

Liu FF, Mbenoun M, Barnes I, Roux J, Wingfield MJ, Li GQ, Li JQ, Chen SF. 2015. New *Ceratocystis* species from *Eucalyptus* and *Cunninghamia* in South China. Mycoscience. 107:1451–1473.

Mace ME, Bell AA, Beckman CH. 1981. Fungal wilt diseases of plants. New York (London): Academic Press.

Malloch D, Blackwell M. 1993. Dispersal biology of the ophiostomatoid fungi. In: Wingfield MJ, Seifert KA, Webber JF, editors. *Ceratocystis* and *Ophiostoma*: taxonomy, ecology, and pathogenicity. St Paul (MN): APS Press; p. 195–206.

Maramorosch K, Loebenstein G. 2009. Plant disease resistance: natural, non-host innate or inducible. In: Schaechter M, editor. Encyclopaedia of Microbiology. Amsterdam (Netherlands): Elsevier Science; p. 589–596.

Marín Montoya M, Wingfield MJ. 2006. A review of *Ceratocystis* sensu stricto with special reference to the species complexes *C. coerulescens* and *C. fimbriata*. Revista Facultad Nacional de Agronomía Medellín. 59:3045–3375.

Martín-García J, Espiga E, Pando V, Diez JJ. 2011. Factors influencing endophytic communities in poplar plantations. Silva Fennica. 45:169–180.

Mbenoun M, de Beer ZW, Wingfield MJ, Wingfield BD, Roux J. 2014. Reconsidering species boundaries in the *Ceratocystis paradoxa* complex, including a new species from oil palm and cacao in Cameroon. Mycologia. 106:757–784.

McInroy J, Kloepper J. 1994. Novel bacterial taxa inhabiting internal tissues of sweet corn and cotton. In: Ryder MH, Stephens PM, Bowen GD, editors. Improving plant productivity with rhizosphere bacteria. Melbourne (Australia): CSIRO; p. 190.

Midgley S, Turnbull J. 2003. Domestication and use of Australian acacias: case studies of five important species. Australian Systematic Botany. 16:89–102.

Misse AC, Barnes I, Roets F, Mbenoun M, Wingfield MJ, Roux J. 2017. Ecology and population structure of a tree wound-infecting fungus in a native South African forest environment. Fungal Biology. 121:69–81.

Mohammed C, Rimbawanto A, Page D. 2014. Management of basidiomycete root- and stem-rot diseases in oil palm, rubber and tropical hardwood plantation crops. Forest Pathology. 44:428–446.

Morris M, Wingfield M, de Beer C. 1993. Gummosis and wilt of *Acacia mearnsii* in South Africa caused by *Ceratocystis fimbriata*. Plant Pathology. 42:814–817.

Moutia Y, Saumtally S. 2001. Detection from soil and distribution of *Ceratocystis paradoxa* Moreau, causal agent of the pineapple disease of sugarcane. Reduit (Mauritius): AMAS Sugar Industry Research Institute, Food And Agricultural Research Council; p. 75.

Muramoto N, Tanaka T, Shimamura T, Mitsukawa N, Hori E, Koda K, Otani M, Hirai M, Nakamura K, Imaeda T. 2012. Transgenic sweet potato expressing thionin from barley gives resistance to black rot disease caused by *Ceratocystis fimbriata* in leaves and storage roots. Plant Cell Reports. 31:987–997.

Naidoo K, Steenkamp ET, Coetzee MP, Wingfield MJ, Wingfield BD. 2013. Concerted evolution in the ribosomal RNA cistron. PloS One. 8: e59355.

Nasution A, Glen M, Gafur A, Evans K, Mohammed CL 2016a. Endophytic bacteria from *Acacia mangium* and their potential antagonism against *Ceratocystis* sp. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Nasution A, Glen M, Gafur A, Evans K, Mohammed CL 2016b. Developing a rapid screening protocol for resistance of *Acacia mangium* against *Ceratocystis* wilt and canker disease. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Newcombe G. 2011. Endophytes in forest management: four challenges. In: Pirttalä AM, Frank C, editors. Endophytes of forest trees. Dordrecht: Springer Publishing; p. 251–262.

Newhouse AE, Spitzer JE, Maynard CA, Powell WA. 2014. Chestnut leaf inoculation assay as a rapid predictor of blight susceptibility. Plant Disease. 98:4–9.

Nkuekam GK, Barnes I, Wingfield MJ, Roux J. 2009. Distribution and population diversity of *Ceratocystis pirilliformis* in South Africa. Mycologia. 101:17–25.

Nkuekam GK, Jacobs K, de Beer ZW, Wingfield MJ, Roux J. 2008. *Ceratocystis* and *Ophiostoma* species, including three new taxa, associated with wounds on native South African trees. Fungal Diversity. 29:37–59.

Nkuekam GK, Wingfield MJ, Mohammed C, Carnegie AJ, Pegg GS, Roux J. 2012. *Ceratocystis* species, including two new species associated with nitidulid beetles, on eucalypts in Australia. Antonie van Leeuwenhoek. Suppl. 101:217–241.

Nkuekam GK, Wingfield MJ, Roux J. 2013. *Ceratocystis* species, including two new taxa, from *Eucalyptus* trees in South Africa. Australasian Plant Pathology. 42:283–311.

Oliveira LS, Damacena MB, Guimarães LM, Siqueira DL, Alfenas AC. 2016. *Ceratocystis fimbriata* isolates on *Mangifera indica* have different levels of aggressiveness. European Journal of Plant Pathology. 145:847–856.

Oliveira LSS, Harrington TC, Ferreira MA, Damacena MB, Al-Sadi AM, Al-Mahmooli HIS, Alfenas AC. 2015. Species or genotypes? Reassessment of four recently described species of the *Ceratocystis* wilt pathogen, *Ceratocystis fimbriata*, on *Mangifera indica*. Phytopathology. 105:1229–1244.

Paulin-Mahady AE, Harrington TC, McNew D. 2002. Phylogenetic and taxonomic evaluation of *Chalara, Chalaropsis*, and *Thielaviopsis* anamorphs associated with *Ceratocystis*. Mycologia. 94:62–72.

Percival GC. 2001. Induction of systemic acquired disease resistance in plants: potential implications for disease management in urban forestry. Journal of Arboriculture. 27:181–192.

Pilotti M, Di Lernia G, Modesti V, Lumia V, Brunetti A. 2016. Outcome of *Ceratocystis platani* inoculations in *Platanus* × *acerifolia* in relation to season and inoculum dose. iForest. 9:608–617.

Poland JA, Balint-Kurti PJ, Wisser RJ, Pratt RC, Nelson RJ. 2009. Shades of gray: the world of quantitative disease resistance. Trends in Plant Science. 14:21–29.

Pornsuriya C, Sunpapao A. 2015. A new sudden decline disease of bullet wood in Thailand is associated with *Ceratocystis manginecans*. Australasian Plant Disease Notes. 10:26.

Pratt J, Niemi M, Sierota Z. 2000. Comparison of three products based on *Phlebiopsis gigantea* for the control of *Heterobasidion annosum* in Europe. Biocontrol Science and Technology. 10:467–477.

Proborini MW. 2016. Ceratocystis as fungal parasitic on wooden statues. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Ran L, Liu C, Wu G, Van Loon L, Bakker P. 2005. Suppression of bacterial wilt in *Eucalyptus urophylla* by fluorescent *Pseudomonas* sppn China. Biological Control. 32:111–120.

Réblová M, Gams W, Seifert KA. 2011. *Monilochaetes* and allied genera of the *glomerellales*, and a reconsideration of families in the *Microascales*. Studies in Mycology. 68:163–191.

Rishbeth J. 1979. Modern aspects of biological control of *Fomes* and *Armillaria*. European Journal of Forest Pathology. 9:331–340.

Rodas CA, Roux J, van Wyk M, Wingfield BD, Wingfield MJ. 2008. *Ceratocystis neglecta* sp. nov., infecting *Eucalyptus* trees in Colombia. Fungal Diversity. 28:73–84.

Rosado CCG, Guimarães LM, Da S, Faria DA, de Resende MDV, Cruz CD, Grattapaglia D, Alfenas AC. 2016. QTL mapping for resistance to *Ceratocystis* wilt in *Eucalyptus*. Tree Genetics and Genomes. 12:1–10.

Roux J, Coutinho T, Mujuni Byabashaija D, Wingfield M. 2001a. Diseases of plantation *Eucalyptus* in Uganda. South African Journal of Science. 97:16–18.

Roux J, Coutinho TA, Wingfield MJ, Bouillet JP. 2000a. Diseases of plantation *Eucalyptus* in the Republic of Congo. South African Journal of Science. 96:454–456.

Roux J, Heath R, Meke G, Nguvulu C, Miambo F, Geldenhuis C, Wingfield M. 2004a. Fungi associated with bark wounds on indigenous African trees. Phytopathology. 94:S89.

Roux J, Heath RN, Labuschagne L, Nkuekam GK, Wingfield MJ. 2007. Occurrence of the wattle wilt pathogen, *Ceratocystis albifundus* on native South African trees. Forest Pathology. 37:292–302.

Roux J, Meke G, Kanyi B, Mwangi L, Mbaga A, Hunter G, Nakabonge G, Heath R, Wingfield M. 2005. Diseases of plantation forestry trees in eastern and southern Africa: review article. South African Journal of Science. 101:409–413.

Roux J, Van Wyk M, Hatting H, Wingfield M. 2004b. *Ceratocystis* species infecting stem wounds on *Eucalyptus grandis* in South Africa. Plant Pathology. 53:414–421.

Roux J, Wingfield M, Bouillet JP, Wingfield B, Alfenas A. 2000b. A serious new wilt disease of *Eucalyptus* caused by *Ceratocystis fimbriata* in Central Africa. Forest Pathology. 30:175–184.

Roux J, Wingfield M, Byabashaija DM. 2001b. First report of *Ceratocystis* wilt of *Acacia mearnsii* in Uganda. Plant Disease. 85:1029.

Roux J, Wingfield MJ. 2009. *Ceratocystis* species: emerging pathogens of non-native plantation *Eucalyptus* and *Acacia* species. Southern Forests. 71:115–120.

Roux J, Wingfield MJ. 2013. *Ceratocystis* species on the African continent, with particular reference to *Ceratocystis albofundus*, an African species in the *Ceratocystis fimbriata sensu lato* species complex. In: Seifert KA, de Beer ZW, Wingfield MJ, editors. Ophiostomatoid fungi: expanding frontiers. CBS Biodiversity Series. Vol. 337; p. 131–138.

Ryan RP, Germaine K, Franks A, Ryan DJ, Dowling DN. 2008. Bacterial endophytes: recent developments and applications. FEMS Microbiology Letters. 278:1–9.

Samuels G. 1993. The case for distinguishing *Ceratocystis* and *Ophiostoma*. In: Wingfield MJ, Seifert KA, Webber JE, editors. *Ceratocystis* and *Ophiostoma*: taxonomy, ecology, and pathogenicity. St Paul (MN): APS Press; p. 15–20.

Schafer JF. 1971. Tolerance to plant disease. Annual Review of Phytopathology. 9:235–252.

Scheffer R. 1983. Biological control of Dutch elm disease by *Pseudomonas* species. Annals of Applied Biology. 103:21–30.

Scheffer R. 1990. Mechanisms involved in biological control of Dutch elm disease. Journal of Phytopathology. 130:265–276.

Scheffer R, Voeten J, Guries R. 2008. Biological control of Dutch elm disease. Plant Disease. 92:192–200.

Schulz B, Boyle C. 2006. What are endophytes?. In: Schulz BJE, Boyle CJC, Sieber TN, editors. Microbial root endophytes. Berlin: Springer Publishing; p. 21–33.

Sniezko RA. 2006. Resistance breeding against nonnative pathogens in forest trees—current successes in North America. Canadian Journal of Plant Pathology. 28:S270–S279.

Sniezko RA, Koch J. 2017. Breeding trees resistant to insects and diseases: putting theory into application. Biological Invasions. 19:3377–3400.

Solla A, Gil L. 2003. Evaluating *Verticillium dahliae* for biological control of *Ophiostoma novo-ulmi* in *Ulmus minor*. Plant Pathology. 52:579–585.

Souza AGC, Maffia LA, Murta HM, Alves YH, Pereira RM, Picanco MC. 2013. First report on the association between *Ceratocystis fimbriata*, an agent of mango wilt, *Xyleborus affinis*, and the sawdust produced during beetle colonization in Brazil. Plant Disease. 97:1116.

Souza JR, Mendes CC, Guizilini V, Vivaldini KC, Colturato A, Ramos F, Wolf DF. 2015. Automatic detection of *Ceratocystis* wilt in *Eucalyptus* crops from aerial images. IEEE International Conference on Robotics and Automation (ICRA); May 26–30; Seattle. p. 3443–3448. doi: 10.1109/ICRA.2015.7139675

Spatafora JW, Blackwell M. 1994. The polyphyletic origins of ophiostomatoid fungi. Mycological Research. 98:1–9.

Stewart JE, Ross-Davies AL, Graça RN, Alfenas AC, Peever TL, Hanna JW, Uchida JY, Hauff RD, Kadooka CY, Kim M-S, et al. 2018. Genetic diversity of the myrtle rust pathogen (*Austropuccinia psidii*) in the Americas and Hawaii: global implications for invasive threat assessments. Forest Pathology. 48:e12378.

Stone C, Mohammed CL. 2017. Application of remote sensing technologies for assessing planted forests damaged by insect pests and fungal pathogens: a review. Current Forestry Reports. 3:75–92.

Talboys PW. 1972. Resistance to vascular wilt fungi. Proceedings Of The Royal Society Series B-Biological Sciences. 181:319–332.

Tarigan M, Roux J, Van Wyk M, Tjahjono B, Wingfield MJ. 2011a. A new wilt and die-back disease of *Acacia mangium* associated with *Ceratocystis manginecans* and *C. acaciivora* sp nov. in Indonesia. South African Journal of Botany. 77:292–304.

Tarigan M, Van Wyk M, Roux J, Tjahjono B, Wingfield M. 2010. Three new *Ceratocystis* spp. in the *Ceratocystis moniliformis* complex from wounds on *Acacia mangium* and *A. crassicarpa*. Mycoscience. 51:53–67.

Tarigan M, Wingfield MJ, Van Wyk M, Tjahjono B, Roux J. 2011b. Pruning quality affects infection of *Acacia mangium* and *A. crassicarpa* by *Ceratocystis acaciivora* and *Lasiodiplodia theobromae*. Southern Forests. 73:187–191.

Thu P, Qynh D, Dell B. 2012. *Ceratocystis* sp. causes crown wilt of *Acacia* spp. planted in some ecological zones of Vietnam. Journal of Plant Protection. 5:24–30.

Thu P, Qynh D, Fourie A, Barnes I, Wingfield M. 2016. Ceratocystis wilt – a serious threat to Acacia plantations in Vietnam. Workshop Proceedings: *Ceratocystis* in tropical hardwood plantations; Feb 15–16; Yogyakarta. Available from: http://www.forestryhealth.org/

Tran TTT, Pham PQ, Barber PA, Nguyen CM. 2018. Control of *Ceratocystis* causing wilt disease on *Acacia mangium* seedlings. Australasian Plant Pathology. 47:579–586.

Trang TT, Eyles A, Davies NW, Glen M, Ratkowsky D, Mohammed CL. 2018. Screening for host responses in *Acacia* to a canker and wilt pathogen, *Ceratocystis manginecans*. Forest Pathology. 48:e12390.

Tsopelas P, Santini A, Wingfield MJ, de Beer ZW. 2017. Canker stain: a lethal disease destroying iconic plant trees. Plant Disease. 101:645–658.

Upadhyay HP. 1981. A monograph of *Ceratocystis* and *Ceratocystiopsis*. Athens (GA): University of Georgia Press.

Upadhyay HP. 1993. Classification of the ophiostomatoid fungi. In: Wingfield MJ, Seifert KA, Webber JF, editors. *Ceratocystis* and *Ophiostoma*: taxonomy, ecology, and pathogenicity. St Paul (MN): APS Press; p. 7–13.

Valdetaro DC, Oliveira LS, Guimarães LM, Harrington TC, Ferreira MA, Freitas RG, Alfenas AC. 2015. Genetic variation, morphology and pathogenicity of *Ceratocystis fimbriata* on *Hevea brasiliensis* in Brazil. Tropical Plant Pathology. 40:184–192.

Vale FXR, Parlevliet J, Zambolim L. 2001. Concepts in plant disease resistance. Fitopatologia Brasileira. 26:577–589.

Van Wyk M, Adawi AO A, Khan IA, Deadman ML, Al Jahwari AA, Wingfield BD, Ploetz R, Wingfield MJ. 2007a. *Ceratocystis manginecans* sp. nov., causal agent of a destructive mango wilt disease in Oman and Pakistan. Fungal Diversity. 27:213–230.

Van Wyk M, Heath RN, Tarigan M, Vermeulen M, Wingfield MJ. 2010a. Comparison of procedures to evaluate the pathogenicity of *Ceratocystis fimbriata* sensu lato isolates from *Eucalyptus* in South Africa. Southern Forests. 72:57–62.

Van Wyk M, Pegg G, Lawson S, Wingfield MJ. 2007b. *Ceratocystis atrox* sp. nov. associated with *Phoracantha acanthocera* infestations on *Eucalyptus grandis* in Australia. Australasian Plant Pathology. 36:407–414.

Van Wyk M, Wingfield B, Mohali S, Wingfield M. 2009a. *Ceratocystis fimbriatomima*, a new species in the *C. fimbriata* sensu lato complex isolated from *Eucalyptus* trees in Venezuela. Fungal Diversity. 34:175.

Van Wyk M, Wingfield BD, Al-Adawi AO, Rossetto CJ, Ito MF. 2011a. Two new *Ceratocystis* species associated with mango disease in Brazil. Mycotaxon. 117:381–404.

Van Wyk M, Wingfield BD, Clegg PA, Wingfield MJ. 2009b. *Ceratocystis larium* sp. nov., a new species from *Styrax benzoin* wounds associated with incense harvesting in Indonesia. Persoonia. 22:75–82.

Van Wyk M, Wingfield BD, Marín M, Wingfield MJ. 2010b. New *Ceratocystis* species infecting coffee, cacao, citrus and native trees in Colombia. Fungal Diversity. 40:103–117.

Van Wyk M, Wingfield BD, Wingfield MJ. 2011b. Four new *Ceratocystis* spp. associated with wounds on *Eucalyptus, Schizolobium* and *Terminalia* trees in Ecuador. Fungal Diversity. 46:111–131.

Van Wyk M, Wingfield BD, Wingfield MJ. 2013. *Ceratocystis* species in the *Ceratocystis fimbriata* complex. In: Seifert KA, de Beer ZW, Wingfield MJ, editors. Ophiostomatoid fungi: expanding frontiers. Utrecht: CBS Biodiversity Series; p. 337.

Vigouroux A, Stojadinovic B. 1990. Infection of plane tree by *Ceratocystis fimbriata f. platani* after contamination of water as growing substrate of wounded roots. European Journal of Forest Pathology. 20:118–121.

Whipps JM, Lumsden RD. 2001. Commercial use of fungi as plant disease biological control agents: status and prospects. In: Butt TM, Jackdon C, Magan N, editors. Fungal biocontrol agents: progress, problems and potential. Wallingford: CABI; p. 9–22.

Wingfield MJ, de Beer C, Visser C, Wingfield BD. 1996. A new *Ceratocystis* species defined using morphological and ribosomal DNA sequence comparisons. Systematic and Applied Microbiology. 19:191–202.

Wingfield MJ, Roux J, Coutinho T, Govender P, Wingfield B. 2001a. Plantation disease and pest management in the next century. The Southern African Forestry Journal. 190:67–71.

Wingfield MJ, Slippers B, Roux J, Wingfield BD. 2001b. Worldwide movement of exotic forest fungi, especially in the tropics and the southern hemisphere: this article examines the impact of fungal pathogens introduced in plantation forestry. AIBS Bulletin. 51:134–140.

Witzell J, Martín JA, Blumenstein K. 2014. Ecological aspects of endophyte-based biocontrol of forest diseases. advances in endophytic research. New Delhi: Springer Publishing; p. 321–333.

Wolda H. 1988. Insect seasonality: why? Annual Review of Ecology and Systematics. 19:1–18.

Wulder MA, Dymond CC, White JC, Leckie DG, Carroll AL. 2006. Surveying mountain pine beetle damage of forests: a review of remote sensing opportunities. Forest Ecology and Management. 221:27–41.

Yadeta K, Thomma B. 2013. The xylem as battleground for plant hosts and vascular wilt pathogens. Frontiers in Plant Science. 4:1–12.

Zauza EAV, Alfenas AC, Harrington TC, Mizubuti ES, Silva JF. 2004. Resistance of *Eucalyptus* clones to *Ceratocystis fimbriata*. Plant Disease. 88:758–760.

Zhang ZP, Li Q, Luo LX, Li JQ, Hao JJ. 2017. First report of mango wilt caused by *Ceratocystis fimbriata* in *Mangifera indica* in China. Plant Disease. 101:142.