



Infected nursery stock and poor silvicultural practices contribute to development of *Ceratocystis manginecans* wilt and canker disease in *Eucalyptus pellita*

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Abstract

In Indonesia and Malaysia, *Eucalyptus pellita* has replaced large areas of *Acacia mangium* plantations as the latter species is highly susceptible to *C. manginecans*. This strategy may not be effective in the long term as *E. pellita* is also susceptible to Ceratocystis wilt and canker disease though it has higher tolerance than *A. mangium*. Furthermore, the pathogen has the capacity to evolve and adapt to new hosts. To highlight the need for careful sanitation in nurseries and the potential impact of wounding during silvicultural operations, two experiments were conducted to (i) demonstrate the ability of cryptic *Ceratocystis* infections in nursery plants to develop into Ceratocystis wilt and canker disease after planting out and (ii) assess the risk of Ceratocystis infection and disease development from different wound types. In the nursery, three-month-old mini-cuttings of seven *E. pellita* clones were artificially wounded and inoculated with two isolates of *Ceratocystis manginecans* near the base of the stem. The disease incidence and lesion length were measured one month later, just prior to planting out. After four months of growth in the plantation, the trees were harvested and stems sliced longitudinally to measure the length of xylem discolouration. Six of 36 ramets of susceptible clones died and the length of xylem discolouration was significantly greater in susceptible clones than in tolerant clones. The second experiment was based on inoculation of 12-month-old plantation-grown trees of one clone of *E. pellita* with one isolate of *C. manginecans* using six different wounding methods. The inoculated wounds on the trees all produced xylem discolouration, except for those that only penetrated the outer bark. Disease incidence was greater at stem heights of 30 to 90 cm than on the basal stem or branch stub. The experiment emphasised the importance of minimising the risk of *C. manginecans* infection following wounding in the nursery and in the field as the discolouration is an indication of xylem blockage that can lead to tree mortality.

Keywords Wound · Symptom · Xylem discolouration

Introduction

As a suitable species for tropical plantation forestry, *Eucalyptus pellita* is currently widely planted in South East Asia; it is also resistant to rust and leaf blight, has some tolerance

to Ceratocystis wilt diseases (Guimarães et al. 2010) and has good pulp and solid wood properties (Japarudin et al. 2020). In Indonesia and Malaysia, *E. pellita* has replaced *Acacia mangium* as the latter species is highly susceptible to *C. manginecans* (Inail et al. 2019; Lee 2018; Nambiar et al. 2018). However, one of the consequences is that inoculum deposits associated with *A. mangium* pose a potential high risk of infection for *E. pellita* and other species (Fourie et al. 2016).

Symptoms associated with disease caused by *Ceratocystis* spp. have been recorded in *Eucalyptus* spp. in plantations on several continents (Hardie et al. 2018; Liu et al. 2015; Roux et al. 2000, 2001; Souza et al. 2015; van Wyk

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et al. 2012). The symptoms identified include lesions and mottling on the bark, a dark-brown roseate streaking pattern in the wood, wilting, leaf chlorosis and death (Roux and Wingfield 2009a; Roux et al. 2020). However a tree may suffer from disease caused by a *Ceratocystis* sp. for some time before any symptoms are apparent (United States Forest Service 1985). *Ceratocystis* diseases in plantations may stem from planting stock infected in the nursery as the symptoms of infection in the nursery may be cryptic or difficult to distinguish from other common nursery diseases (Indrayadi et al. 2023b) but disease may develop rapidly once planted out (Ferreira et al. 2011).

Ceratocystis diseases of woody plants depend on wounding created by animals, human activities, and wind-driven breakages to initiate infection (Hardie et al. 2017; Harrington 2013; Roux et al. 2007; Roy et al. 2020; van Wyk et al. 2012). *Ceratocystis* spores can also be spread by insect vectors and water splashing (Ferreira et al. 2013; Harrington 2013). Thus, human activities, cultivation tools and mother plants associated with the mass propagation of *Eucalyptus* species from mini-cuttings pose a risk of contamination by pathogens such as *Ceratocystis* and subsequent disease spread post-planting (Ferreira et al. 2011, 2013; Harrington 2013). Several plantation companies have adopted artificial inoculation of young potted plants as a screening procedure for resistance or tolerance to *C. manginecans* (Nasution et al. 2022), though the efficacy of this practice is not without controversy (Wingfield et al. 2023). In addition, it is unclear whether the isolates that infect young plants in the nursery will become aggressive pathogens if infected nursery plants are transferred to plantations, as isolates of *C. manginecans* and *C. fimbriata* vary in host specificity and relative aggressiveness against different host genotypes (Harrington et al. 2011; Indrayadi et al. 2023c; Valdetaro et al. 2019) and host responses vary with phenology (Baker et al. 2003).

In this study, two experiments were established to examine whether and how disease spread in *E. pellita* plantations is affected by contamination with *C. manginecans* in the nursery compared to subsequent infection in the plantation. In the first experiment, the development of disease symptoms was followed after artificial inoculation of mini-cuttings in the nursery and transplanting into the field; in the second, disease development was assessed four months after the creation and inoculation of six contrasting wound types in 12-month-old trees. The objectives were to show that: (1) *C. manginecans* infection in the nursery was linked to symptom development after planting; and (2) disease development can be associated with a range of wound types in the field.

Materials and methods

Inoculum preparation

Two *C. manginecans* isolates, EP0313C and EP0106C which were obtained from *E. pellita* clone SMAA7700EP growing in a nursery and plantation, respectively, were cultured on Potato Sucrose Agar (PSA) in Petri dishes for use in this experiment. The PSA was prepared by boiling 200 g potato to produce 1 L of broth, adding 20 g agar and 20 g sucrose, followed by autoclaving for 20 min at 121 °C. The isolates were cultured for 14 days before inoculum plugs containing mycelia, perithecia and spores, were removed using a 3-mm cork borer. Mycelial plugs are preferred to spore suspensions as the inoculum can be more accurately applied with lower risk of environmental contamination.

Plant materials

For experiment 1, six ramets from each of seven *E. pellita* clones were grown to age 3 months from mini-cuttings. The clones were produced by taking cuttings from six seedlings of a control-pollinated cross and their female parent, clone SMAA7700EP (EP77). In a previous study (Indrayadi et al. 2023b) three of the clones (EP3, EP123, and EP76) had been ranked as susceptible, and four (SMAA7700EP, EP114, EP117, and EP176) as tolerant to *C. manginecans*. For experiment 2, twelve-month-old plantation-grown trees of *E. pellita* clone SMAA7700EP were used.

Inoculation, planting and disease assessment

Experiment 1: For each clone, at age 3 months an approximately 3-mm-long wound was created with a scalpel centred at 30-mm height above the stem base of the cutting. An inoculum needle was used to place the inoculum plug onto the wound which was then sealed with parafilm® (Bemis NA). Twelve individual plants of each clone including clone SMAA7700EP were used, six inoculated with isolate EP0313C and six with isolate EP0106C; to test for contamination, a control treatment of six plants of clone SMAA7700EP were wounded without pathogen inoculation. External lesion length was measured one month after inoculation as this was shown to be a suitable proxy for xylem internal discolouration in an earlier trial (Supplementary Fig. 1).

Inoculated plants and the controls were then planted in an isolated area which was at least 5 km from the nearest *Eucalyptus* plantation. A randomised complete block design with six blocks was adopted; each treatment (clone × isolate) was represented once in each block. Each planting hole was prepared by digging a 0.3- × 0.3-wide × 0.4-m depth hole

and then returning the cultivated soil back into the hole. The plant was placed in the centre of the hole using a dibbler. Fertilizer, 100 g 15:15:15 N: P:K and 200 g TSP (P_2O_5) was applied 0.1–0.2 m from the plant, and divided between two holes created by a dibbler. Additional fertilizer, 100 g TSP (P_2O_5) and 50 g KCl, were applied three months after planting using the same application procedure. Four months after planting, plant survival and mortality were assessed, and length of xylem discolouration (XD) measured by opening the bark and slicing the stem longitudinally from above to below the inoculation point. Plant height was measured on all the live plants.

Experiment 2: For the field inoculation study, six distinct 10- × 10-mm wound-type treatments were created with a scalpel (Table 1). An inoculum plug of isolate EP0106C was placed onto each wound with an inoculum needle; parafilm® (Bemis NA) was used to seal the wounds on the upper part of stem and lanolin the wounds on the basal stem and branches. Control trees were wounded, and the wounds sealed as above but without pathogen inoculation. There were three replicate trees for each wound type and their control, giving a total of nine inoculated wounds and nine control wounds per treatment. Xylem discolouration was measured four months after inoculation. Percentage rate of infection (%) in each treatment was calculated by the ratio of the number of inoculated wounds from which xylem discolouration developed to the total number of wounds (9).

Table 1 Wound inoculation treatments in experiment 2

Wound type	Size and depth of wound	Location of wound	Number of trees	Number of wounds/tree
Type 1	10 × 10 mm, into bark, not penetrating to the living wood	Main stem at heights of 30, 60 and 90 cm above ground	3	3
Type 2	10 × 10 mm, through the bark to the surface of the living wood	Main stem at heights of 30, 60 and 90 cm above ground	3	3
Type 3	10 × 10 mm, through the bark and 3 mm into the living wood	Main stem at heights of 30, 60 and 90 cm above ground	3	3
Type 4	10 × 10 mm, through the bark to the surface of the living wood	Main stem at 1–2 cm above ground level	3	3
Type 5	Branch excision	2 cm diameter branch removed at 2 cm from the main stem	3	3
Type 6	Branch excision	2 cm diameter branch removed flush with the main stem	3	3

The PROC GLM function of the SAS statistical software (SAS Institute, North Carolina) was used for the analysis of variance of the length of xylem discolouration, and Fisher's LSD to determine whether the overall F-test was significant at the $P < 0.05$ level. In Experiment 1, the mean XD for each treatment was calculated by combining the values for both isolates. For comparing the aggressiveness of the two isolates, the mean XD for each isolate was calculated by combining the values across all clones.

Results

Experiment 1. Linking infection in the nursery to symptom development after planting

In the nursery, infection was confirmed in all inoculated plants, one month after inoculation, by a reddish patch on the bark surrounding a sunken inoculation point (Fig. 1a, red circle); on the controls, no symptoms were observed. Four months after planting in the field, the outer boundaries of the lesion were no longer distinguishable. Tree death was only observed in the susceptible clones (Fig. 1b), and one-third of “dead” trees sprouted new shoots from the stem base (Fig. 1c, red arrow). A cross-section through an advanced-stage infected stem was characterised by a black-brown striped discolouration (Fig. 1d), but this was absent at an early stage of infection (Fig. 1e). A longitudinal section through the stem of a dead tree showed the vertical spread, both upward and downward from the point of inoculation, of this black-brown striped discolouration (Fig. 1f, g). However, in most trees this spread was contained (Fig. 1g); and in most control trees, xylem discolouration was absent (Fig. 1h). Four months after planting, XD in the living trees ranged from 2.7 to 113.5 cm.

One month after inoculation, the mean external lesion lengths of the susceptible clones EP123 and EP3 were significantly greater ($p < 0.05$) than for susceptible clone EP76 and the tolerant clones (Fig. 2a). There was no significant difference between the tolerant clone, EP176 and susceptible clone EP76, both were significantly shorter than the two remaining susceptible clones and significantly longer than the two other tolerant clones. Two of the tolerant clones, EP114 and EP117 had similar lesion lengths to their maternal clone, SMAA7700EP ($p < 0.05$) and these three all had significantly smaller visible lesions than the three susceptible clones and the remaining tolerant clone, EP176. It was not possible to directly compare the length of xylem discolouration before and after planting out, but comparison of external lesion length with xylem discolouration one month after inoculation showed a strong correlation ($r = 0.81$) in a duplicate set (Supplementary Fig. 1).

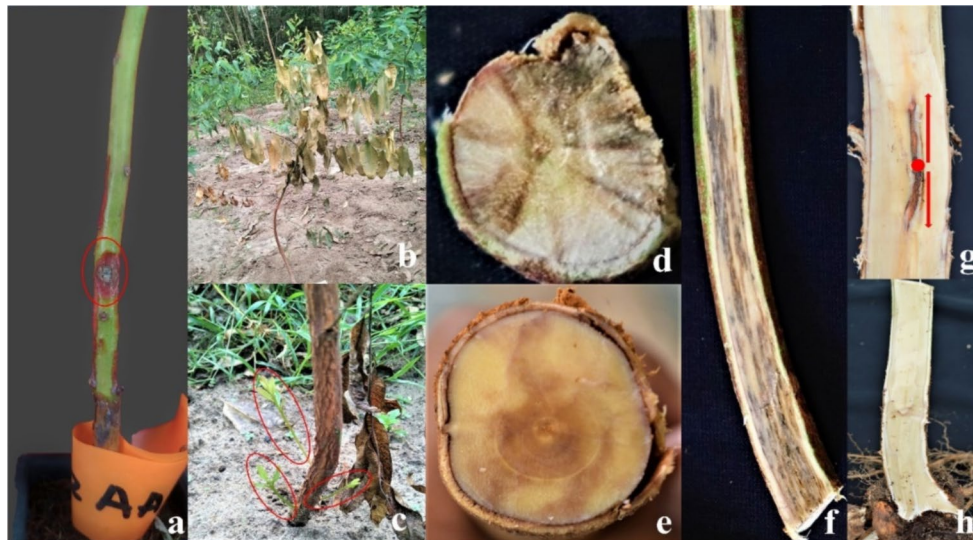


Fig. 1 Symptoms following inoculation of cuttings in the nursery and transplanting into the field: (a) a reddish area on the bark surrounding a sunken lesion before planting out; (b) wilting leading to tree death; (c) new shoot growing on basal stem of “dead” tree; (d, e) cross-section of stems four months after planting out showing advanced xylem discoloration near the inoculation point (d) and an early stage of

xylem discoloration further away from the inoculation point; (f, g, h) longitudinal sections showing well-developed (f) and developing (g) xylem discoloration; note its upward and downward progression (red arrows) from the infection point (red circle) in the stem, (h) control without pathogen inoculation (h)

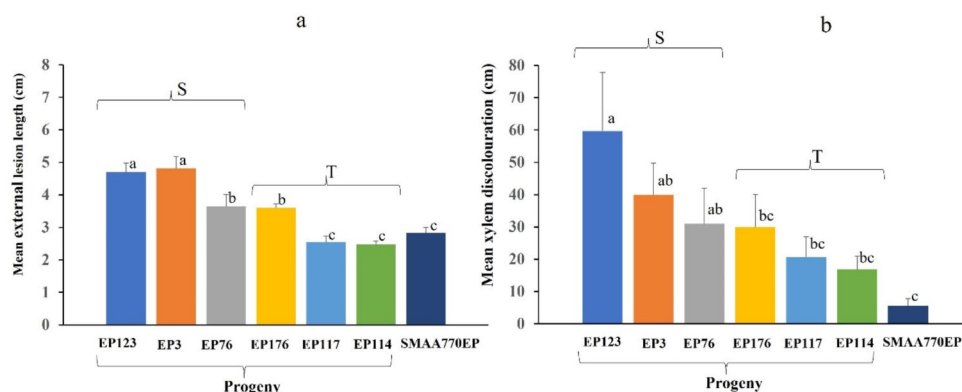


Fig. 2 Comparison of (a) external lesion length of mini-cuttings in the nursery, one month after inoculation and (b) length of xylem discoloration of the same mini-cuttings four months after planting out. Clones are grouped as susceptible (S), or tolerant (T) based on pre-

vious screening; SMAA7700EP was the female parent of the clones. Means with the same lower-case letter are not significantly different using Fisher's LSD ($\alpha=0.05$). Note: the controls without inoculation were excluded from the analysis

The mean length of xylem discoloration in the maternal clone SMAA7700EP four months after planting in the field was approximately twice the external lesion length at planting out (one month after inoculation in the nursery) while those of the tolerant clones were 6–8 times greater and the susceptible clones increased by 8–12 (Fig. 2a, b). In the field, three plants of clone EP123, two of clone EP3, and one of clone EP76 died. The most susceptible clone, EP123, had a significantly greater XD ($p < 0.05$) than the tolerant clones; and the maternal clone, SMAA7700EP, had a significantly lower XD than the susceptible clones. The remaining clones, while varying in mean XD, were not significantly different from each other (Fig. 2b). Of the six uninoculated

control trees, one plant became infected, with XD of 7.1 cm (data not shown).

The two isolates were not significantly different in aggressiveness as measured by external lesion length ($P > 0.05$) one month after inoculation in the nursery (3.1–3.4 cm). Between three and four months after planting, isolate EP0313C killed two plants of clone EP123 and two of clone EP3, and isolate EP0106C killed one of clone EP123 and one of EP76. Isolate EP0313C had a lower mean XD (25.6 cm) than isolate EP0106C (34.4 cm) and was slightly less infectious, causing XD in 83.3% vs. 92.7% of the inoculated plants, but neither of these differences were significant ($P > 0.05$).

Fig. 3 Disease symptom development four months after inoculation of 12-month-old trees using six wound types: (a) Bark recovery from superficial bark wounding; (b) a control wound type 3 without inoculation; (c) canker resulting from inoculation of deeper wound types, 2 and 3; (d) a vertical section showing the upward and downward progress of the xylem discolouration (XD; see red arrows) from the point of inoculation of wound types 2 and 3 (red dot); (e) XD in the vertical section of wound type 4; (f) a stem cross-section showing radial spread of discolouration; (g, h) vertical development of XD associated with inoculation of branch stubs; and (i) new shoot growth at the base of the stem in response to death of the upper crown. The yellow arrows indicate necrosis caused by the pathogen

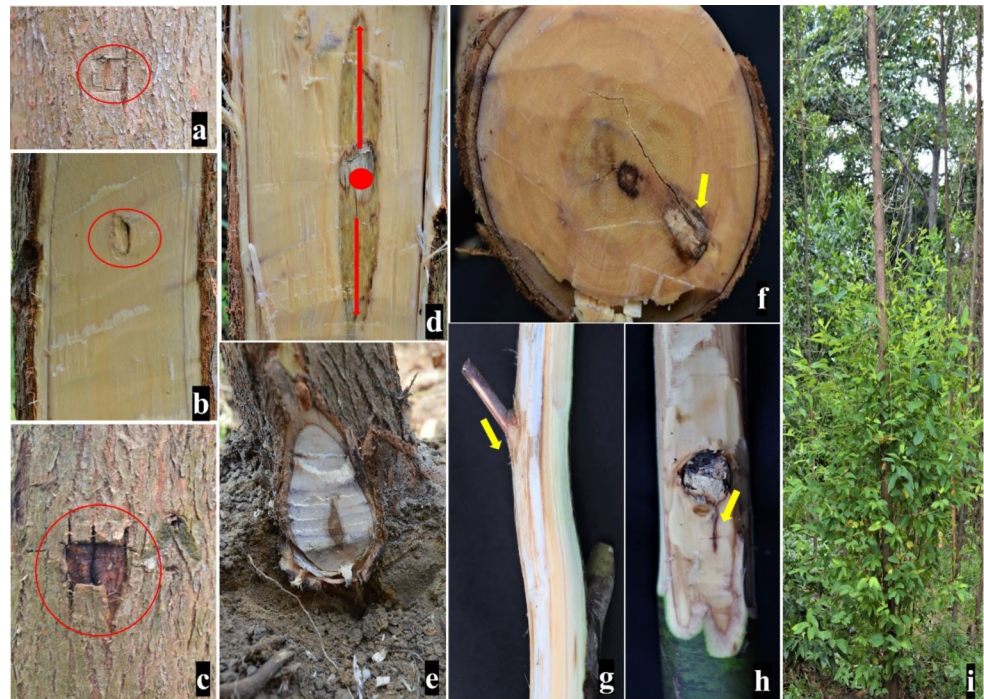
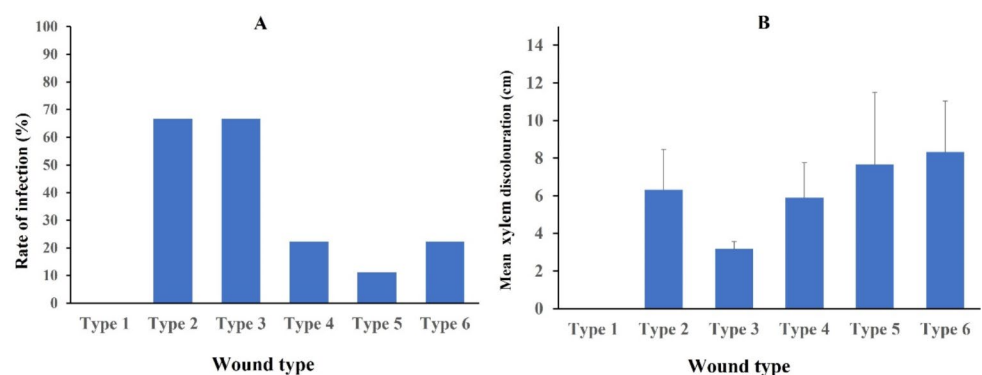


Fig. 4 The rate of infection, as demonstrated by lesion development, from each type of inoculated wound (A) and the mean length of xylem discolouration in different wound types four months after inoculation in the field (B)



Experiment 2. Disease development associated with wound types in the field

Symptom development following inoculation varied with wound type (Fig. 3). Inoculation following damage to the bark only (type 1) was followed by a full recovery (Fig. 3a), there was no external lesion development or xylem discolouration, and the wood had the same appearance as in the absence of inoculation (Fig. 3b). Inoculation following wounding to greater depths (wound types 2 and 3) led to canker-like symptoms (Fig. 3c). Xylem discolouration of wound types 2, 3 and 4 developed vertically upwards, downwards (Fig. 3d, e), and radially towards the pith (Fig. 3f). Inoculation of branch stubs, wound types 5 and 6, allowed the pathogen to spread downwards into the main stem, from where it grew both upwards and downwards (Fig. 3g-h). In response to the death of foliage in the upper crown, new

shoots sprouted in the lower part of stems of some trees inoculated at the stem base, wound type 4 (Fig. 3i).

The rate of infection following inoculation of wounds was lower for wounds located at the stem base or branch stubs, types 4–6 ($\leq 22\%$), than stem wounds at heights of 30 to 90 cm, type 2 and 3 (67%) (Fig. 4A). Plant height ranged from 635 to 1360 cm at four-months after inoculation in the field and xylem discolouration from 2.1 to 23 cm (Fig. 4B) and there was no significant difference among wound types.

Discussion

This study confirmed that undetected infections in the nursery are a potential cause of *Ceratocystis* wilt and canker in *E. pellita* plantations. Four months after planting out, the nursery-inoculated mini-cuttings of *E. pellita* showed partial to severe development of a black-brown striped xylem

discolouration and of the 84 inoculated cuttings, six died. Only susceptible clones died and, in two of the six plants that died, this was associated with resprouting prior to death. These symptoms typify the development of wilt diseases caused by *Ceratocystis* spp. in *Eucalyptus* spp., the discolouration often not evident until after opening and removing the bark of the infected stem (Ferreira et al. 2013; Roux et al. 2020). Consequently, unlike *A. mangium* (Syazwan et al. 2021), there may be no obvious signs that a lesion is developing from a point of infection prior to plant death. Rapid death occurs because xylem function is disrupted by the horizontal and vertical development of the necrotic tissue (Al-Sadi et al. 2010; Firmino et al. 2018; Tsopelas et al. 2021). Resprouting is a common response of *Eucalyptus* spp. to a variety of stressors, and not just to disease (Bellingham and Sparrow 2000; Clarke et al. 2013); however with *Ceratocystis* diseases it is usually a portent of impending tree death (Vigouroux and Olivier 2004). Therefore, because of the high risk of infection by *C. manginecans* in nurseries, particularly in S-E Asia, when raising seedlings and cuttings of *E. pellita* and other eucalypt species, plant material with stem wounds should not be selected for planting. Furthermore, efficient phytosanitary measures in nurseries must be implemented to mitigate the spread of disease in the plantation and enhance disease management plans for *E. pellita*.

Four months after planting out, the variation in length of XD among the six clonal progenies followed a similar pattern to that observed in the nursery one month after inoculation. Clones EP123 and EP3 were consistently the most susceptible, and EP117 and EP114 the most tolerant; differences between these clones were similar to those found in a previous nursery study (Indrayadi et al. 2023a). The larger relative differences in XD between clones four months after planting than at planting may be related to a greater defence response in the tolerant than susceptible clones as the trees age (Pimenta et al. 2017; Silva et al. 2020). Differences in susceptibility among clones were mostly not significant due to the high variability within each clone. Nonetheless, the consistency of results that were significant, e.g. the low susceptibility of SMAA7700EP and high susceptibility of EP123, confirms the utility of preliminary nursery screening to reduce the number of clones for field trials (Brawner et al. 2020; Nasution et al. 2022; van Wyk et al. 2010).

The mean XD of the uninoculated control treatment in experiment 1 four months after planting was very small (< 1.5 cm), but the fact that one of the wounded but uninoculated trees became infected in the field illustrates the potential for wounding in the nursery to facilitate infection in the field. Pathogen contamination following wounding is a key factor in the spread of *Ceratocystis* diseases (Harrington 2013). Tools, propagation techniques, and other nursery practices are commonly associated with wounding

(Ferreira et al. 2011), and care in their application can minimise the potential for disease infection and spread in the field.

Although isolate EP0313C led to the death of more mini cuttings in the field than isolate EP0106 (four vs. two deaths), this difference was not statistically significant, as was the case for differences in lesion lengths and XD at one and five months after inoculation, respectively. In a previous study of 111 *E. pellita* clonal progenies and their two parental clones that were similarly inoculated at age three months in the nursery (Indrayadi et al. 2023c), the mean disease index associated with isolate EP0313C was significantly greater than for isolate EP0106C two months after inoculation. The aggressiveness of *Ceratocystis* isolates is known to vary with their origin, and this affects disease severity (Oliveira et al. 2015, 2016). In our study, EP0313C that was isolated from young plants in the nursery led to greater mortality of *E. pellita* mini-cuttings in the nursery and in the initial period after planting than that caused by EP106C that was isolated from the same clonal parent but in the field (Indrayadi et al. 2023b). However if EP0106C is better adapted to field conditions, this isolate may eventually become the more aggressive of the two isolates after planting as the host-pathogen interaction is strongly influenced by environment (Pilotti et al. 2016). In experiment 1, a period of observation longer than four months may have been required to substantiate this hypothesis.

In experiment 2, following inoculation in the field, five of the six wound types tested had a non-zero rate of infection following inoculation with *C. manginecans*. The exception was type 1, when wounding was restricted to the outer bark of the main stem and no subsequent infection was observed. This is non-living tissue (Leite and Pereira 2017) and because species of *Ceratocystis* are hemibiotrophic, living plant tissue is required for its initial development (biotrophic phase) before it can transition to infecting non-living tissue (necrotrophic phase) (Sun et al. 2020). In wound types 2 and 3 where the stem wound penetrated to the vascular tissue, a high percentage of wounds became infected and the direction of disease spread followed the upward and downward direction of the water-conducting vessel tissues (Hughes et al. 2020; Tsopelas et al. 2021). Stem damage is common during post-planting silvicultural operations and is probably the main reason for the high levels of infection by *Ceratocystis* spp. leading to high rates of mortality in plantations of acacia and susceptible clones of eucalypts (Barnes et al. 2003; Chi et al. 2019). Branch excision as in wound types 5 and 6 followed by inoculation led to downward development of xylem discolouration through conducting tissues that are oriented down towards the stem (Coder 2014). As with the basal stem (wound type), the incidence of disease following branch removal and inoculation was low, but in

those trees where infection was successful, xylem discolouration occurred and rapidly extended towards and into the stem, as with *C. fimbriata* in *E. grandis* (Barnes et al. 2003). A variety of wound types that impinge on the plant's vascular system are therefore potential portals for *C. manginecans* infection and disease development. If this experiment were to be conducted in an area already infested with *C. manginecans* it may be possible to wound the trees and wait for natural infection, but this would require the wounding of many more trees to account for the haphazard distribution of natural inoculum and many of the wounds may heal before the arrival of inoculum coincides with climatic conditions conducive to infection.

Eucalyptus pellita clone SMAA0077EP was more susceptible to *C. manginecans* infection when inoculated in the nursery at 3 months old than as twelve-month-old trees inoculated in the field. In mini-cuttings infected in the nursery at 3 months old, then planted out a month later and grown on for another 4 months, the mean length of xylem discolouration was 16 cm. In contrast, in field-grown trees four months after inoculation at age 12 months, the maximum length of xylem discolouration was 9 cm. In previous studies, disease caused by *C. manginecans* was more severe on younger than older *A. auriculiformis* (Chi et al. 2019) and by *C. fimbriata* on younger than older *Eucalyptus* spp. (Roux and Wingfield 2009b). Host phenology, particularly in relation to tissue age, influences pathogen development (Farber and Mundt 2017) and disease resistance genes associated with the synthesis of flavonoids in older plants are expressed to a greater extent than in younger plants (Wang et al. 2020). Nonetheless, the ranking of clone susceptibility was consistent between younger and older plants, contrary to the claim that screening of small plants for disease tolerance is meaningless (Wingfield et al. 2023).

Dealing with an existing inoculum load (Ferreira et al. 2011; Roux et al. 2020) can best be managed through the selection and use of *Ceratocystis*-resistant or tolerant clones that have been validated through field-testing across a range of geographic and/or climatic zones. However, in the absence of resistance, an integrated approach to disease management is required that also includes nursery sanitation and silvicultural practices. This study showed that even in the absence of observable symptoms at planting, mini-cuttings infected by *C. manginecans* in the nursery are prone to rapid disease development after planting out, potentially leading to mortality in the field. Similarly, wounding and infection of vascular tissues in the field will also lead to disease development, albeit more slowly than in those trees that are infected at a younger age. Minimizing wound damage and the risk of pathogen infection requires best practice in the nursery and in the field.

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Data availability Not applicable (all data is presented in figures and tables).

Declarations

Ethical approval Not applicable.

Competing interests The authors declare no competing interests.

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