ELSEVIER

Forest Ecology and Management 176 (2003) 427–437

Forest Ecology and Management

www.elsevier.com/locate/foreco

Pathogenicity of *Cryphonectria eucalypti* to *Eucalyptus* clones in South Africa

Marieka Gryzenhout[a,*], Ben E. Eisenberg[a], Teresa A. Coutinho[a], Brenda D. Wingfield[b], Michael J. Wingfield[a]

[a]*Department of Microbiology and Plant Pathology, Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, 0002 Pretoria, South Africa*

[b]*Department of Genetics, Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, 0002 Pretoria, South Africa*

Received 19 September 2001; received in revised form 8 April 2002; accepted 16 May 2002

Abstract

Eucalyptus spp. are planted in many parts of the world for the production of timber and pulp, and in South Africa, they form a major component of the forestry industry. The canker pathogen, *Cryphonectria eucalypti*, is pathogenic to *Eucalyptus* spp. in Australia and Tasmania and occurs in all of the major *Eucalyptus* growing areas of South Africa. This study was undertaken to consider the pathogenicity of *C. eucalypti* to *Eucalyptus* clones in South Africa. Fifteen isolates of *C. eucalypti* were initially screened for their virulence on a susceptible *Eucalyptus grandis* clone (ZG14) in the field. A plot consisting of 42 different clones of *Eucalyptus* was subsequently challenged with a selected virulent isolate of *C. eucalypti* to determine whether clones differ in their tolerance to the pathogen. Results showed that *C. eucalypti* is capable of causing significant lesions on *Eucalyptus* clones and that disease development is strongly dependent on environmental factors. All of the clones tested were susceptible to *C. eucalypti*, but exhibited varying levels of tolerance to the pathogen.

Keywords: *Cryphonectria eucalypti*; *Endothia gyrosa*; Pathogenicity; *Eucalyptus*; Tolerance; Interaction

1. Introduction

Cryphonectria eucalypti M. Venter and M.J. Wingf., previously known as *Endothia gyrosa* (Schw.: Fr.) Fr. (Venter et al., 2002), is a canker pathogen of *Eucalyptus* spp. in mainland Australia (Old et al., 1986; Walker et al., 1985), Tasmania (Wardlaw, 1999; Yuan and Mohammed, 1997, 1999, 2000) and South Africa (Van der Westhuizen et al., 1993). In Australia, typical symptoms of *C. eucalypti* infection on *Eucalyptus* include bark cracks, cankers with kino exudation and die-back of coppice shoots, branches and stems (Old et al., 1986; Walker et al., 1985). In severe cases the pathogen can also kill trees (Old et al., 1986; Walker et al., 1985; Wardlaw, 1999).

C. eucalypti occurs exclusively on *Eucalyptus* spp. and was previously known as *E. gyrosa*, an opportunistic canker pathogen of various hardwood species in the USA (Appel and Stipes, 1986; Shear et al., 1917; Stipes and Phipps, 1971; Roane et al., 1974). Recent studies have shown that the fungus

* Corresponding author. Tel.: +27-12-420-3938; fax: +27-12-420-3960.
E-mail address: marieka.gryzenhout@fabi.up.ac.za (M. Gryzenhout).

0378-1127/02/$ – see front matter
PII: S0378-1127(02)00309-2

Fig. 1. Disease symptoms associated with *C. eucalypti* on *Eucalyptus* spp. in South Africa: (a) longitudinal cracking of the bark associated with cankers caused by *C. eucalypti*; (b) orange stromata of *C. eucalypti* sporulating around cankers (indicated with arrow).

reported as *E. gyrosa* on *Eucalyptus* from Australia and South Africa, is not the same species as *E. gyrosa* from North America (Venter et al., 2001, 2002). This finding emerged from phylogenetic studies based on DNA sequences of the ITS1, ITS2 and 5.8S regions of the Internal Transcribed Spacer region of the ribosomal RNA operon, and the β-tubulin genes (Venter et al., 2001, 2002). This DNA-based grouping was supported by morphological and physiological characteristics, and led to the description of the new species, *C. eucalypti* (Venter et al., 2002).

Pathogenicity trials have generally led to the view that *C. eucalypti* is a mild pathogen in the absence of stress, although, it may cause girdling and death of seedlings (Old et al., 1986, 1990). In some cases, *C. eucalypti* has been associated with severe cankers. In Tasmania, annual cankers developed in the bark of *E. nitens* Maiden, while more severe cankers also extended into the cambium (Wardlaw, 1999). The annual cankers were shed from trees with the bark, thereby, causing little damage. Infections reaching the cambium caused die-back and tree death. At this site, the severe disease was due more to a combination of factors, than to highly virulent strains of the pathogen (Yuan and Mohammed, 2000). *C. eucalypti* is, therefore, considered a potential threat to *Eucalyptus* plantings (Yuan and Mohammed, 1999, 2000).

In South Africa, *C. eucalypti* occurs in all major *Eucalyptus* growing areas. The fungus causes superficial cankers that cause the bark to crack (Fig. 1a), and cankers are covered with orange fruiting bodies (Fig. 1b) (Van der Westhuizen et al., 1993). These cankers rarely cause kino exudation or damage to the cambium and have no apparent influence on timber quality or yield. *C. eucalypti* has, however, recently been found associated with serious cankers extending into the cambium of *E. smithii* R.T. Baker near Pietermaritzburg (KwaZulu/Natal province) (Dr. J. Roux, personal communication), and it was closely associated with stunted *Eucalyptus* seedlings in Tzaneen and Barberton (Mpumalanga and Limpopo province).

Eucalyptus spp. make up approximately 40% of forestry plantations in South Africa (Anonymous, 1998). Vegetative propagation of *Eucalyptus* clones to improve timber quality and yield, is widely practised (Denison and Kietzka, 1993). Clonal propagation may increase the threat of disease outbreaks. A clear understanding of disease susceptibility in *Eucalyptus* clones is needed in order to best manage disease (Chou, 1981; Wingfield et al., 1991). Our aim in this study was to test the virulence of *C. eucalypti* on several clones of *Eucalyptus* in field inoculations to establish whether different degrees of tolerance toward the pathogen exists.

2. Materials and methods

2.1. Source of isolates

Stromata associated with typical cankers were collected on *Eucalyptus* spp. from commercial plantations in various regions of South Africa. The fungus was isolated by placing the stromata on malt extract agar (Biolab, Merck, Midrand, South Africa) and isolates were maintained on 2% malt extract agar at 4 °C. All isolates are preserved in the culture collection of the Tree Pathology Co-operative Programme (TPCP), Forestry & Agricultural Biotechnology Institute (FABI), University of Pretoria, Pretoria, South Africa. Fifteen isolates were randomly chosen for our initial inoculations (Table 1).

2.2. Screening of isolates

Trunks of trees were inoculated using the technique described by Van der Westhuizen et al. (1993) and a negative control was included (a sterile MEA plug) as well. Lesions were measured by removing the bark and exposing the cambial discolouration, since no external discolouration of the bark around the lesions was visible on the first clone used. Re-isolations were conducted to ensure that lesions were caused by the inoculated fungi.

Fifteen isolates of *C. eucalypti* (Table 1) were inoculated in a 2-year-old coppice stand of an *E. grandis* W. Hill. ex Maiden clone (ZG14) at the Flatcrown farm near KwaMbonambi, Northern KwaZulu/Natal province. Twenty trees per isolate were inoculated in January 1998 and lesion lengths were measured 7 weeks later. In a second inoculation in March 1998, two blocks of trees of the same clone were included, at different locations in the same compartment. In this case, the same isolates used in the first inoculation were used, and results were recorded after 7 weeks. In the second inoculation, trees were inoculated in two different blocks to determine whether the variation in the data obtained for isolates in the first inoculation, was due to the isolates or the environment. Combined analysis of variance was performed on all data (Freeman, 1973). Differences between isolates were evaluated using Tukey's Multiple Range and the *t*-test (SAS, 1989). Possible interactions between isolates and the environment were investigated by means of an Additive Main Effects and Multiplicative Interaction Model (AMMI) analysis (Eisenberg et al., 1996; Gauch and Zobel, 1996).

Table 1
Isolates of *C. eucalypti* from South Africa used in the inoculation trials

Isolate number[a]	Host	Origin[b]	Collector	Date
CMW8532	*Eucalyptus* sp.	Amangwe	J. Roux	July 1997
CMW8533	*Eucalyptus* sp.	Amangwe	J. Roux	July 1997
CMW8534	*E. grandis*	Dukuduku	J. Roux	July 1997
CMW8535	*E. grandis*	Dukuduku	J. Roux	July 1997
CMW8536	*E. grandis*	Sabie	M. Gryzenhout	December 1997
CMW8537	*Eucalyptus* sp.	Futululu	J. Roux	July 1997
CMW8538	*E. grandis*	Graskop	M. Gryzenhout	December 1997
CMW8539	*Eucalyptus* sp.	KwaMbonambi area	J. Roux	July 1997
CMW8540	*Eucalyptus* sp.	KwaMbonambi town	J. Roux	July 1997
CMW8541	*Eucalyptus* sp.	Nseleni	J. Roux	July 1997
CMW8542	*Eucalyptus* sp.	Nyalazi	J. Roux	July 1997
CMW8543	*Eucalyptu* sp.	Nyalazi	J. Roux	July 1997
CMW8544	*E. grandis*	Piet Retief	M. Gryzenhout	December 1997
CMW8545	*E. saligna*	Tzaneen	J. Roux	October 1997
CMW8546	*E. saligna*	Tzaneen	J. Roux	October 1997

[a] Culture collection of the Forestry and Agricultural Biotechnology Institute (FABI), University of Pretoria, Pretoria 0002, South Africa.
[b] All areas or estates other than Tzaneen, Piet Retief, Graskop and Sabie, are in the KwaZulu/Natal province, while the former regions except for Tzaneen which is in the Limpopo province are in the Mpumalanga province.

2.3. Screening of Eucalyptus clones

One isolate (CMW8541) with a high level of virulence was selected from the isolate screening trial for inoculation of 42, 18-month-old clones located on the Amangwe estate near KwaMbonambi, northern KwaZulu/Natal province. The clones included the following: *E. grandis* with *E. camaldulensis* Dehnh. cross (GC); *E. grandis* with *E. urophylla* S. T. Blake cross (GU); *E. tereticornis* Sm. × *E. saligna* Sm. cross (TS); a Transvaal adapted *E. grandis* clone (TAG); a Zululand *E. grandis* clone (ZG); and *E. grandis* with *E. tereticornis* cross (GT). The 42 clones were planted in a randomised block design consisting of 10 blocks, in which each clone was planted once. Each block consisted of two identical rows. Four extra rows, identical to the design of the first block, were planted at the side of the trial. One of these extra rows was inoculated with a negative control (sterile agar plugs), and all other rows with the test fungus. Due to blanks in the plot where trees have died after planting, the maximum of 23 trees per clone could not always be inoculated for each row. The trial was surrounded by two border rows of trees.

The trees were inoculated in October 1998 and the lesions were measured in December 1998. Both external bark lesions as well as lesions at the cambium surface developed in all the clones inoculated in this trial (unlike the ZG14 clones where no external bark lesions were visible). Because reading of internal cambial lesions resulted in excessive damage to trees, lengths and widths of external lesions were measured on all trees. The data were analysed using one and two way analyses of variance. Differences between the clones were evaluated using Tukey's Multiple Range and the *t*-test (SAS, 1989).

3. Results

3.1. Screening of isolates

Two weeks after inoculation, localised cracking and kino exudation were visible around the inoculation points (Fig. 2a). No symptoms were visible in the control trees (Fig. 2b). After 7 weeks, *C. eucalypti* was found sporulating on the surface of most lesions that developed after inoculation. No external discoloration of the epidermis was produced and lesions consisted only of kino veins in the cambium (Fig. 2c) that resulted in a swelling on the bark surface. Re-isolations consistently resulted in the recovery of the inoculated fungus.

Table 2
Combined ANOVA for lesion length measurements of the three inoculation sets of the ZG14 trial with *C. eucalypti*

Source	SS	d.f.	MS	*F*	*P*
Isolate	604184.8	15	40279.0	30.6	0.0001
Inoculation	353943.9	2	176972.0	130.4	0.0001
Isolate × inoculation	117328.6	30	3911.0	2.97	0.0001

S.E.M. for inoculations = 9.2. S.E.M. for isolates = 21.0. S.E.M. for isolates × inoculation interaction = 36.3.

Significant differences were observed ($P > 0.0001$) between lesion lengths for the two inoculation times and also for trees in the two separate blocks that comprised the second inoculation (Table 2). The first inoculation gave rise to the greatest mean lesion length for all trees (136.9 mm), while mean lesion length in the second inoculation was higher in the one block (103.8 mm) than the other (82.6 mm).

Significant differences existed between isolates in all inoculations ($P > 0.0001$) (Table 2, Fig. 3). All lesions differed significantly in size from the control ($P > 0.0001$), which produced no or only small responses to wounding. Means per isolate for all inoculations combined were of the same size order and represented a continuum of values between 132 and 100 mm when ranked from the highest to the lowest overall mean for the three data sets combined (Fig. 3). In this ranking, lesion lengths for isolates did not differ significantly from those directly above or below them in the ranking. Isolates that ranked low, however, did differ significantly from those with a high ranking (Fig. 3). Due to the continuous change between values, it was not possible to establish distinct groupings based on the virulence of the isolates. Isolates CMW8537, CMW8544, CMW8541 and CMW8545, however, were the most virulent. The close size order of lesion lengths was also observed for each of the two inoculation studies separately, including the two separate blocks inoculated on the same date (data not shown).

Ranking positions of isolates based on mean lesion length differed for each of the three inoculation events.

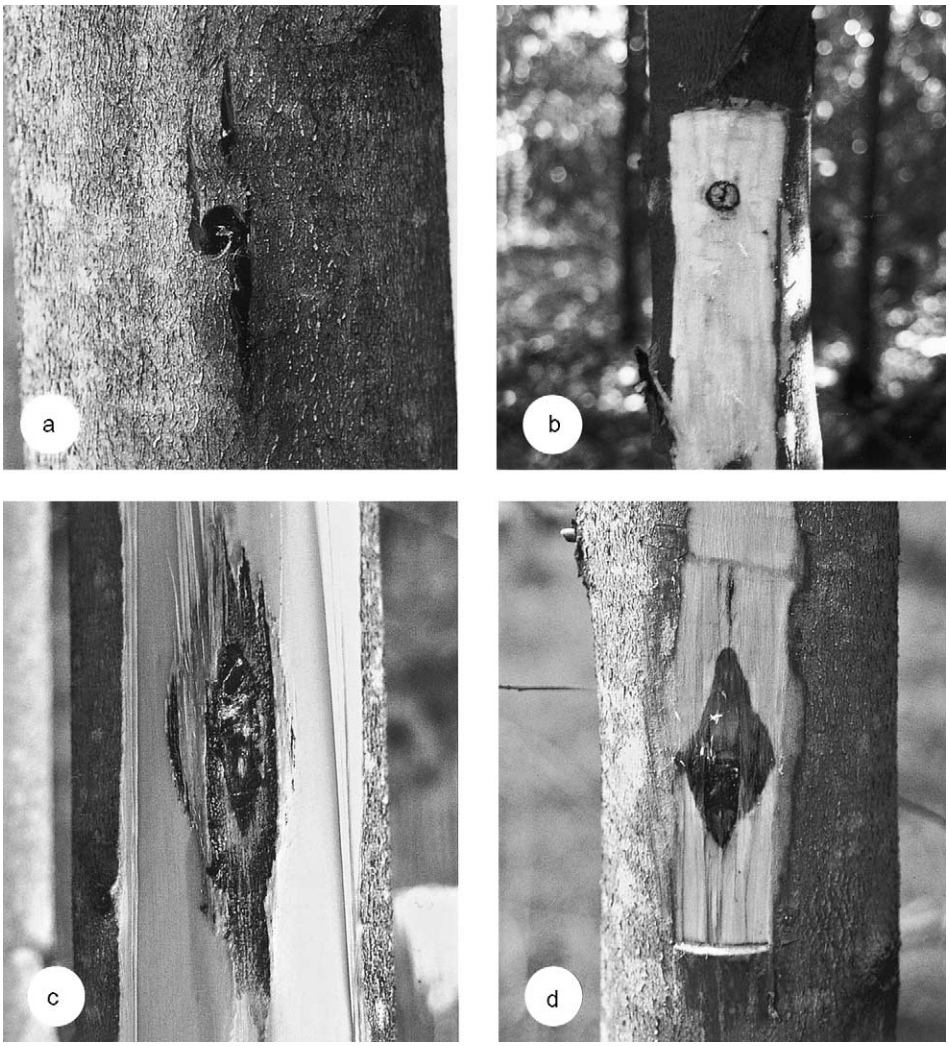

Fig. 2. Lesion types associated with inoculation by *C. eucalypti* on *E. grandis* clone ZG14: (a) cracking and kino exudation around inoculation point; (b) healthy control inoculation; (c) cambial lesions produced in the cambium; (d) discolouration of the bark.

For example, isolate CMW8538 ranked highest in the first inoculation (longest lesion), but was 15th in one block of the second inoculation, sixth in the other block of the second inoculation, and eighth in the ranking based on overall mean for all three inoculations (Table 3). Some isolates, such as CMW8544 and CMW8546, held constant positions for all inoculations. This shift in ranking made selection of the most virulent isolates difficult, and ultimately this choice needed to be relatively arbitrary.

Combined analysis of variance for the results of the three inoculation events showed a significant interaction of experiment with isolates ($P > 0.0001$) (Table 2). Analysis of data using the AMMI model confirmed that levels of interaction existed between isolates and external factors (Fig. 4). Some isolates exhibited greater interaction with external factors, while others were more stable. All isolates, having the same size order of lesions, could be divided into three groups: a group with little interaction (score values 0 ± 1), a group with moderate interaction (± 1 to ± 2), and a group showing high levels of interaction (± 2 to ± 4). Isolates CMW8537, CMW8538 and CMW8544 showed a high degree of

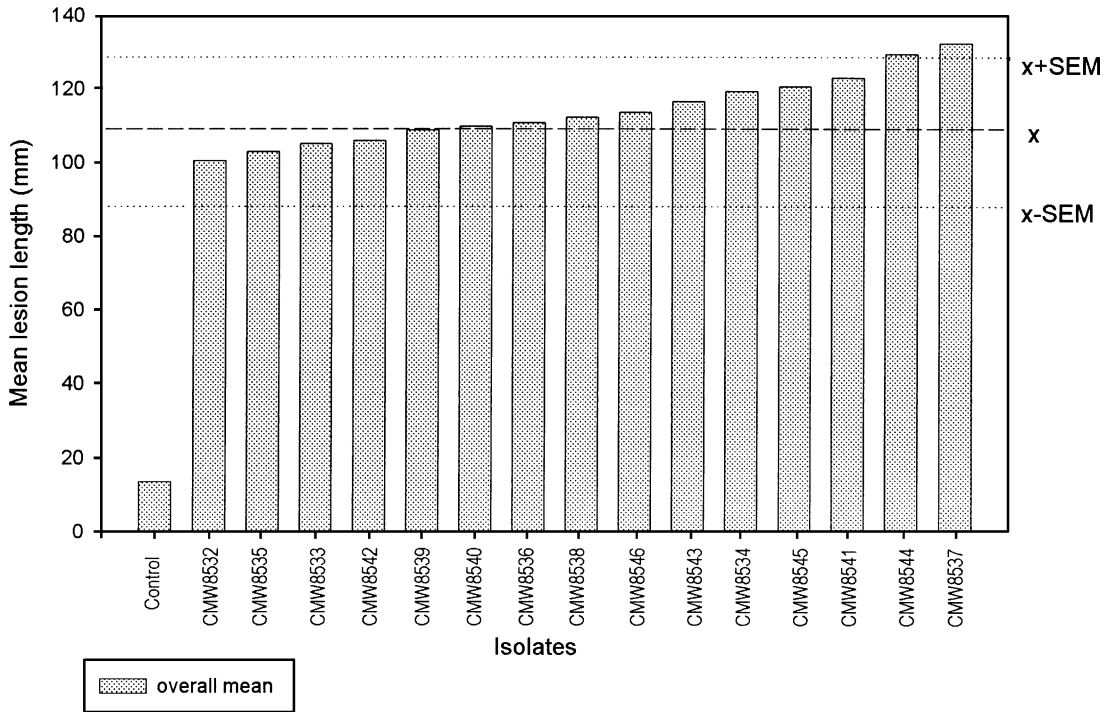

Fig. 3. Mean lesion length values, sorted from the lowest to the highest, for 15 isolates of *C. eucalypti* and a negative control over all three inoculation sets on the *E. grandis* clone ZG14, in KwaZulu/Natal. Lesions were measured 7 weeks after inoculation.

interaction, while isolates CMW8543, CMW8534 and CMW8546 showed little interaction. Each of the three inoculation trials also had high interaction with external factors. The one block of the second inoculation trial had a negative value (−5.8), while the first inoculation and the other block of the second inoculation had positive values (1.9 and 3.9, respectively; Fig. 4).

Table 3
Ranking of isolates of *C. eucalypti,* from longest to shortest lesion, based on mean lesion length in the three inoculation events, respectively, and the three inoculation events combined

Rank	Inoculation 1	Inoculation 2, block 1	Inoculation 2, block 2	All inoculations combined
1	CMW8538	CMW8544	CMW8537	CMW8537
2	CMW8541	CMW8541	CMW8543	CMW8544
3	CMW8537	CMW8545	CMW8544	CMW8541
4	CMW8534	CMW8540	CMW8534	CMW8545
5	CMW8544	CMW8546	CMW8542	CMW8534
6	CMW8536	CMW8537	CMW8538	CMW8543
7	CMW8545	CMW8533	CMW8545	CMW8546
8	CMW8539	CMW8534	CMW8546	CMW8538
9	CMW8543	CMW8532	CMW8541	CMW8536
10	CMW8546	CMW8542	CMW8539	CMW8540
11	CMW8540	CMW8543	CMW8536	CMW8539
12	CMW8535	CMW8536	CMW8535	CMW8542
13	CMW8533	CMW8539	CMW8540	CMW8533
14	CMW8542	CMW8535	CMW8532	CMW8535
15	CMW8532	CMW8538	CMW8533	CMW8532

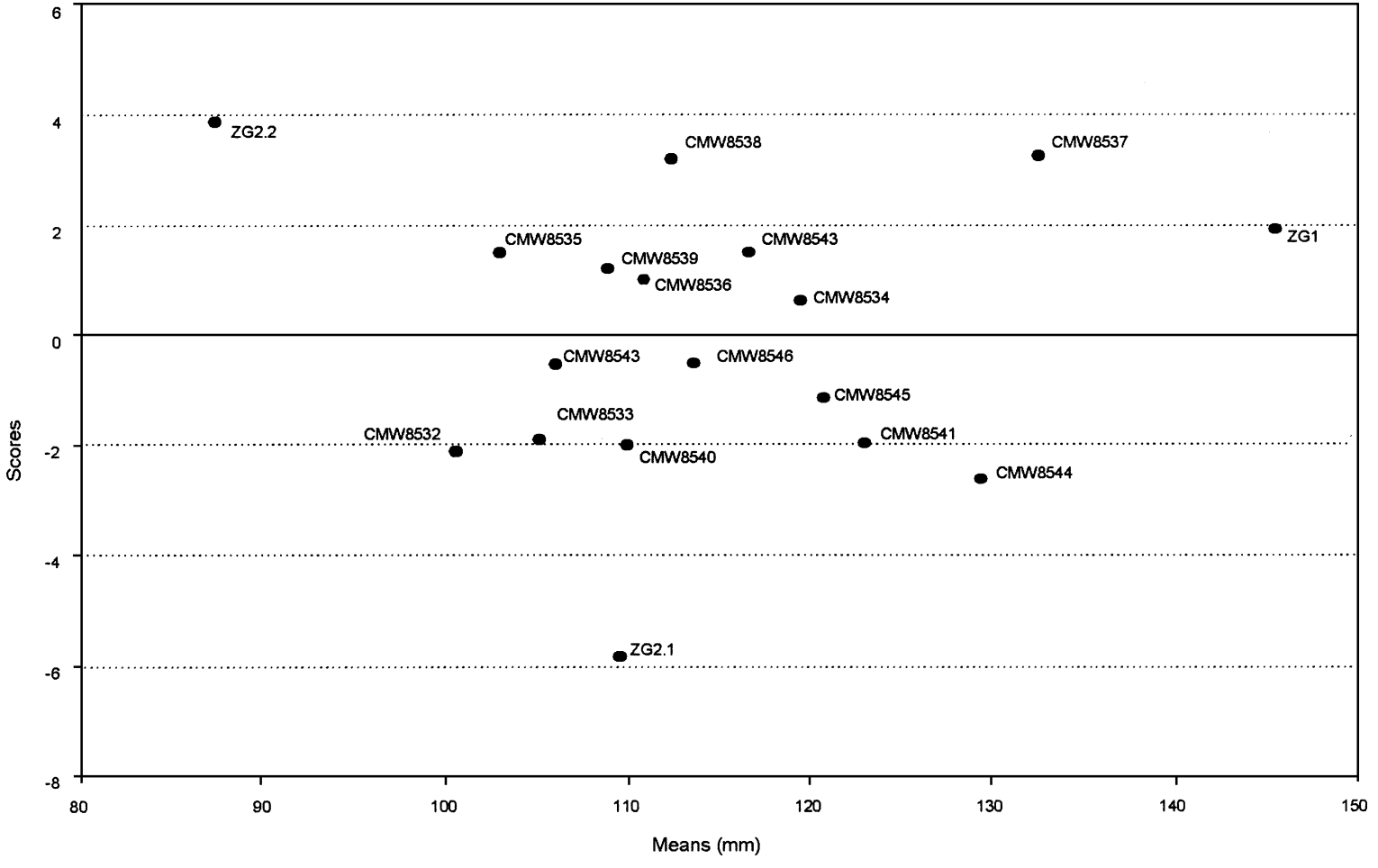

Fig. 4. Score values obtained from an AMMI analysis plotted against the overall lesion length for each isolate of *C. eucalypti* in the ZG14 inoculation trial. Score values for the different inoculation events of the ZG14 trial are also included: ZG1 represents the first inoculation, ZG2.1 represents the one block of the second inoculation and ZG2.2 represents the other block of the second inoculation trial.

Table 4
Two way ANOVA for lesion length, width and area ($l \times b$) associated with *C. eucalypti* inoculations of 42 *Eucalyptus* clones

Source	SS	d.f.	MS	*F*	*P*
Block (length)	4198.7	10	419.9	2.0	0.0321
Clone (length)	40121.4	41	978.6	4.7	0.0001
Block (width)	3849.1	10	384.9	2.3	0.0137
Clone (width)	40607.3	41	990.4	5.8	0.0001
Block (area)	44396136.0	10	4439613.6	2.6	0.0055
Clone (area)	319068267.8	41	7782152.9	4.5	0.0001

Length: S.E.M. (clones) = 4.4; width: S.E.M. (clones) = 3.9; area: S.E.M. (clones) = 398.2.

3.2. Screening of Eucalyptus clones

Cambial lesions, as well as external bark discoloration (Fig. 2d), were produced on all of the clones. No lesions developed in any of the control inoculations. Significant differences in lesion size were observed between blocks within the trial ($P > 0.0321$), as well as between the different clones ($P > 0.0001$; Table 4, Fig. 5). In the clone comparison experiment, greater emphasis was placed on the widths of lesions, than in the prior trial where isolates were screened. This is because lesion widths were significantly more variable for the external lesions than for the cambial lesions. Based on length, width and area (length × width; Fig. 5), means for each clone formed a continuum of values in the mean rankings. Thus, no clone differed significantly from the clone directly below or above it in ranking. Levels of tolerance to *C. eucalypti* were, however, visible because clones with a low ranking differed significantly from those with a high ranking. Rankings of the clones based on lesion width and lesion area, were similar, especially in the highly susceptible and highly tolerant groups. These, however, differed from the rankings of the measurements based on length. In a correlation analysis, rankings based on width and area of lesions were more similar to each other ($r = 0.91$) than the rankings between length and area ($r = 0.71$), while those between length and width were poorly correlated ($r = 0.39$). Lesion area appeared to be the best measure for tolerance in this case, and was thus chosen for assessing the tolerance of the clones. Based on the rankings of lesion area, clones GC575, GU115 and GC962 were the most tolerant, while clones GC747 and GC796, were the least tolerant to infection by *C. eucalypti* (Fig. 5).

4. Discussion

This study demonstrates that *C. eucalypti* is capable of causing distinct lesions on *Eucalyptus* spp. in South Africa. In the past, symptoms associated with *C. eucalypti* (reported as *E. gyrosa*) have been of superficial cracks on the surface of tree bark (Van der Westhuizen et al., 1993) without cambial cankers. The extent of the lesions produced in the pathogenicity trials conducted here, suggests that *C. eucalypti* is capable of causing more serious damage when conditions are conducive to disease.

C. eucalypti has a wide host range within *Eucalyptus* and in the past, has been found on numerous commercial clones in South Africa (Van der Westhuizen et al., 1993). In the current inoculation trials, lesions were produced on all 42 clones tested. This wide host range is similar to the situation in Australia, where *C. eucalypti* has been recorded on 20 *Eucalyptus* spp. (Davison, 1982; Davison and Coates, 1991; Old et al., 1986; Walker et al., 1985; Wardlaw, 1999; White and Kile, 1993). This pathogen thus poses a threat to many *Eucalyptus* spp. and clones.

The variation in lesion size for each isolate and the low reproducibility of results for pathogenicity tests, have been encountered before (Old et al., 1986). This may be an indication that strong genotype × environment interactions exist (Matheson and Cotterill, 1990; Basford and Cooper, 1998). This is supported in our study by the combined analysis of variance for the three inoculation events (isolate trial) together, and the differences in ranking of the isolates across the three inoculation events. Our AMMI analysis, furthermore, showed that isolates of *C. eucalypti* exhibit different levels of interaction with external factors. Such factors could include climate, nutritional

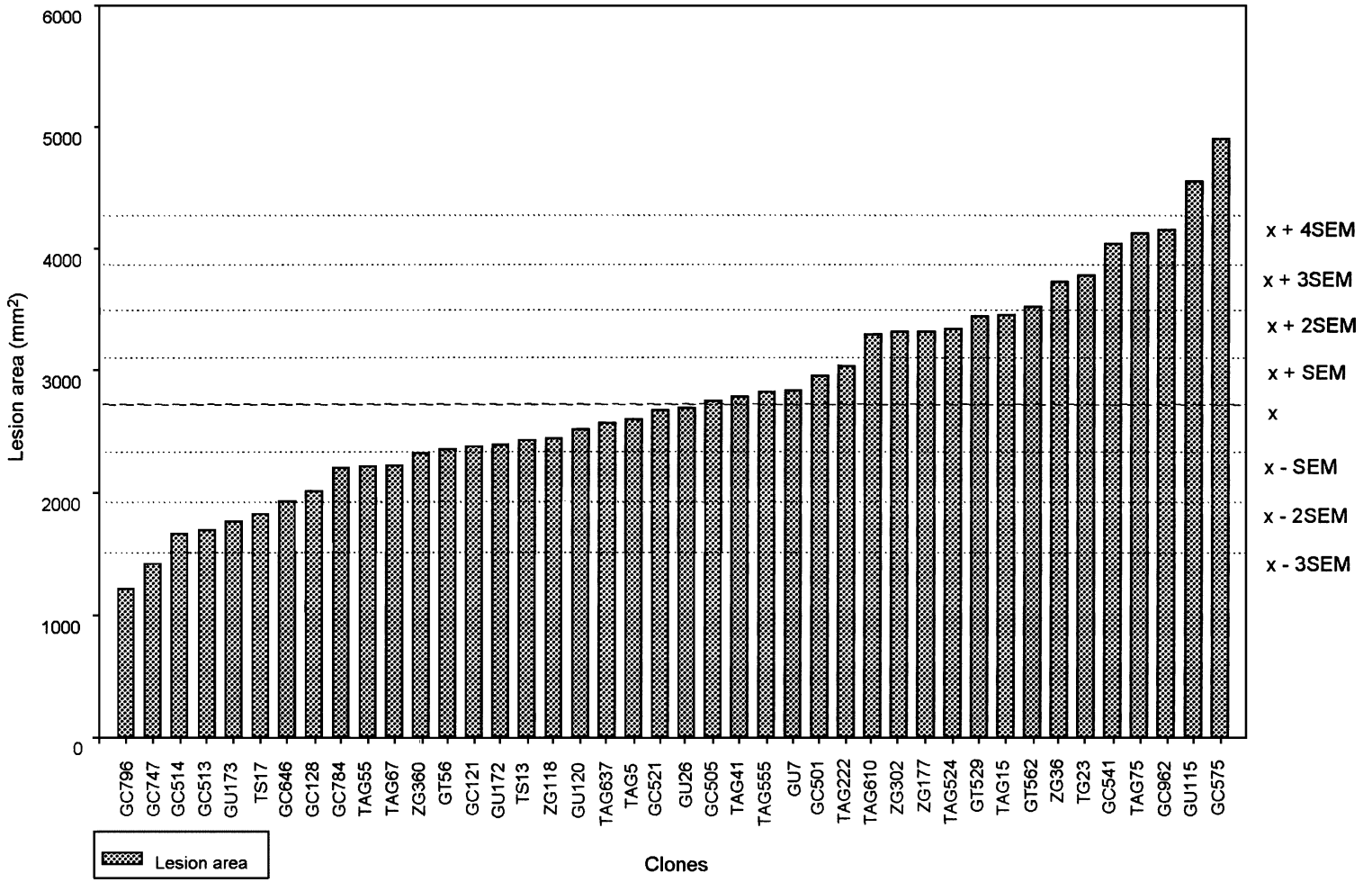

Fig. 5. Mean lesion area (length × width) values of 42 clones of *E. grandis* after inoculation with a highly pathogenic isolate of *C. eucalypti* (CMW8541). Lesions were measured 6 weeks after inoculation.

levels and temperature (Basford and Cooper, 1998; Wu and O'Malley, 1998), but the factors influencing interaction in this study, were not investigated.

The interaction exhibited by the isolates supports previous hypotheses that *C. eucalypti* is a stress-related pathogen (Old et al., 1990; Yuan and Mohammed, 1997). In Australia, trees weakened by defoliation were found to be more susceptible to *C. eucalypti* (Old et al., 1990). In the same study, water stress did not predispose the trees to *C. eucalypti*, although, it was thought that the *Eucalyptus* trees used in that study might have had a high degree of tolerance to water stress. In South Africa, *C. eucalypti* was found on virtually every tree in a dry compartment in the Tzaneen region of South Africa, and was also common in one of the coldest compartments in that same area.

Isolates showing a high degree of interaction with environmental factors, may not be highly virulent in a particular environment, but may become serious elsewhere. This is important in determining the potential damage that a pathogen might cause. The virulence of an isolate with a positive (or negative) interaction score value is augmented in environments with positive (or negative) score values, e.g. CMW8537 in the first inoculation and second block of the second inoculation (Fig. 4; Eisenberg et al., 1996). This may have been the situation in the *E. nitens* stand in Tasmania where severe cankers caused by *C. eucalypti*, led to tree death (Wardlaw, 1999). Environments with negative score values also tend to diminish the virulence of isolates with positive score values (Eisenberg et al., 1996).

Determining the level of interaction of an isolate with external factors is crucial when selecting an appropriate isolate for disease screening. Selection of an isolate producing large lesions may not be the best choice if that isolate shows a high level of interaction, since it will give more variable results. Selecting an isolate with low interaction would, in all likelihood, yield more stable results (Eisenberg et al., 1996; Gauch and Zobel, 1996). This, however, needs to be determined experimentally for every pathogen.

Different levels of susceptibility to *C. eucalypti* have previously been observed for five species of *Eucalyptus* (Old et al., 1986; Yuan and Mohammed, 1999). The varying levels of tolerance observed for different clones in the present study, indicates that a selection and breeding program to develop trees tolerant to *C. eucalypti* could be established. Such programmes have already been established for resistance to other *Eucalyptus* pathogens (Alfenas et al., 1983; Denison and Kietzka, 1993; Dianese et al., 1984) and are considered valuable tools for forest managers. This proactive approach could minimise damage by this pathogen in Australia and South Africa, in conditions that may trigger this pathogen to be highly virulent.

5. Conclusions

Pathogenicity studies using isolates of *C. eucalypti* on different clones of *Eucalyptus* in South Africa demonstrated pathogenicity on several clones. This is in contrast to the field situation where *C. eucalypti* usually does not cause serious disease. Isolates of the fungus exhibited different degrees of interaction with external factors, indicating that the pathogen can be important under certain conditions. There are also degrees of tolerance to the pathogen in *Eucalyptus* clones, which can potentially be used in breeding programmes aimed at reducing the impact of *C. eucalypti*.

Acknowledgements

We thank Dr. Jolanda Roux for reviewing this manuscript. The National Research Foundation (NRF), members of the Tree Pathology Co-operative Programme (TPCP) and the THRIP support programme of the Department of Trade and Industry, South Africa, are acknowledged for financial support.

References

Alfenas, A.C., Jeng, R., Hubbes, M., 1983. Virulence of *Cryphonectria cubensis* on *Eucalyptus* species differing in resistance. Eur. J. For. Pathol. 13, 197–205.

Anonymous, 1998. Abstract of South African forestry facts for the year 1996/97 Forest Owners Association 1–11.

Appel, D.N., Stipes, R.J., 1986. A description of declining and blighted pin oaks in eastern Virginia. J. Arboric. 12, 155–158.

Basford, K.E., Cooper, M., 1998. Genotype × environment interactions and some considerations of their implications for wheat breeding in Australia. Aust. J. Agric. Res. 49, 153–174.

Chou, C.K.S., 1981. Monoculture, species diversification, and disease hazards in forestry. N. Z. J. For. 26, 20–36.

Davison, E.M., 1982. *Endothia havanensis* on Jarrah. Aust. Plant Pathol. 11, 10–11.

Davison, E.M., Coates, D.J., 1991. Identification of *Cryphonectria cubensis* and *Endothia gyrosa* from eucalypts in Western Australia using isozyme analysis. Aust. Plant Pathol. 20, 157–160.

Denison, N.P., Kietzka, J.E., 1993. The development and utilisation of vegetative propagation in Mondi for commercial afforestation programmes. S. Afr. Bosboutydskr. 165, 47–54.

Dianese, J.C., Moraesde, T.S.A., Silva, A.R., 1984. Response of *Eucalyptus* species to field infection by *Puccinia psidii*. Plant Dis. 68, 314–316.

Eisenberg, B.E., Gauch, H.G., Zobel, R.W., Kilian, W., 1996. Spatial analysis of field experiments with wheat (*Triticum aestivum*) and tea (*Camellia sinensis*). In: Kang, M.J., Gauch Jr., H.G. (Eds.), Genotype-by-environment interaction. CRC Press, Boca Raton, FL, USA, pp. 373–404.

Freeman, G.H., 1973. Statistical method for the analysis of genotype–environment interactions. Heredity 31, 339–354.

Gauch, H.G., Zobel, R.W., 1996. AMMI analysis of yield trials. In: Kang, M.J., Gauch, H.G. Jr. (Eds.) Genotype-by-environment interaction. CRC Press, Boca Raton, Florida, USA, pp. 85–122.

Matheson, A.C., Cotterill, P.P., 1990. Utility of genotype × environment interactions. For. Ecol. Manag. 30, 159–174.

Old, K.M., Murray, D.I.L., Kile, G.A., Simpson, J., Malafant, K.W.J., 1986. The pathology of fungi isolated from eucalypt cankers in south-eastern Australia. Aust. For. Res. 16, 21–36.

Old, K.M., Gibbs, R., Craig, I., Myers, B.J., Yuan, Z.Q., 1990. Effect of drought and defoliation on the susceptibility of eucalypts to cankers caused by *Endothia gyrosa* and *Botryosphaeria ribis*. Aust. J. Bot. 38, 571–581.

Roane, M.K., Stipes, R.J., Phipps, P.M., Miller Jr., O.K., 1974. *Endothia gyrosa*, causal pathogen of pin oak blight. Mycologia 66, 1042–1047.

SAS Statistical Software, 1989. SAS/STAT User's Guide, Version 6, Fourth Edition, Vol. 1 & 2. SAS Institute Inc., Cary, NC, USA.

Shear, C.L., Stevens, N.E., Tiller, R.J., 1917. *Endothia parasitica* and related species. United States Department of Agriculture Bulletin 380, 1–82.

Stipes, R.J., Phipps, P.M., 1971. A species of *Endothia* associated with a canker disease of pin oak (*Quercus palustris*) in Virginia. Plant Dis. Rep. 55, 467–469.

Van der Westhuizen, I.P., Wingfield, M.J., Kemp, G.H.J., Swart, W.J., 1993. First report of the canker pathogen *Endothia gyrosa* on *Eucalyptus* in South Africa. Plant Pathol. 42, 661–663.

Venter, M., Wingfield, M.J., Coutinho, T.A., Wingfield, B.D., 2001. Molecular characterization of *Endothia gyrosa* isolates from *Eucalyptus* in South Africa and Australia. Plant Pathol. 50, 211–217.

Venter, M., Myburg, H., Wingfield, B.D., Coutinho, T.A., Wingfield, M.J., 2002. A new species of *Cryphonectria* from South Africa and Australia, pathogenic to *Eucalyptus*. Sydowia 54, 98–117.

Walker, J., Old, K.M., Murray, D.I.L., 1985. *Endothia gyrosa* on *Eucalyptus* in Australia with notes on some other species of *Endothia* and *Cryphonectria*. Mycotaxon 23, 353–370.

Wardlaw, T.J., 1999. *Endothia gyrosa* associated with severe stem cankers on plantation grown *Eucalyptus nitens* in Tasmania. Aust. Eur. J. For. Pathol. 29, 199–208.

White, D.A., Kile, G.A., 1993. Discolouration and decay from artificial wounds in 20-year-old *Eucalyptus regnans*. Eur. J. For. Pathol. 23, 431–440.

Wingfield. M.J., Swart, W.J., Kemp, G.H.J., 1991. Pathology considerations in clonal propagation of *Eucalyptus* with special reference to the South African situation. In: Schönau, A.P.G. (Ed.), Proceedings of the 1991 IUFRO Symposium 2 on Intensive Forestry Durban, South Africa: The Role of *Eucalyptus*, pp. 811–820.

Wu, R.L., O'Malley, D.M., 1998. Nonlinear genotypic response to macro- and microenvironments. Theor. Appl. Genet. 96, 669–675.

Yuan, Z.Q., Mohammed, C., 1997. Investigation of fungi associated with stem cankers of eucalypts in Tasmania. Aust. Australas. Plant Pathol. 26, 78–84.

Yuan, Z.Q., Mohammed, C., 1999. Pathogenicity of fungi associated with stem cankers of *Eucalyptus* in Tasmania. Aust. Plant Dis. 83, 1063–1069.

Yuan, Z.Q., Mohammed, C., 2000. The pathogenicity of isolates of *Endothia gyrosa* to *Eucalyptus nitens* and *E. globulus*. Australas. Plant Pathol. 29, 29–35.