$h^2 = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_c^2 + \sigma_e^2}$ = narrow sense individual heritability in the block;

$c^2 = \sigma_c^2 / (\sigma_a^2 + \sigma_c^2 + \sigma_e^2)$ = correlation due to the common environmental effect in the plot;

σ_a^2 = additive genetic variance;
σ_c^2 = variance among plots;
σ_e^2 = residual variance (environmental within plot + non additive);
A = additive genetic correlation matrix among individuals under evaluation.

REML estimators for variance components using the EM (Expectation-Maximization) algorithm were:

$$\hat{\sigma}_e^2 = [y'y - \hat{f}'b'y - \hat{a}'Z'y - \hat{c}'W'y]/[N - r(x)]$$

$$\hat{\sigma}_a^2 = [\hat{a}'A^{-1}\hat{a} + \hat{\sigma}_e^2 \; tr \; A^{-1} \; C^{22})/q$$

$\hat{\sigma}_c^2 = [\hat{c}'c + \hat{\sigma}_e^2 \; tr \; C^{33})/s$, where:

tr = trace operator;
r(x) = rank of the matrix X;
N-r(x) = error degrees of freedom;
q = number of individuals;
s = number of plots;
N = Total number of data.
C22 and C33 come from:

$$C = \begin{bmatrix} C^{11} & C^{12} & C^{13} \\ C^{21} & C^{22} & C^{23} \\ C^{31} & C^{32} & C^{33} \end{bmatrix}$$ = generalised inverse of the coefficient matrix of the mixed model equations.

The same model, excluding the plot effect, was applied to BSO data sets. For the interaction analyses involving the progeny and BSO data sets the family x trial interaction effect was added.

Variation in *Eucalyptus globulus* LABILL. and *E. nitens* DEAN and MAIDEN in Susceptibility of Adult Foliage to Disease Caused by *Mycosphaerella cryptica* (COOKE) HANSF.

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Summary

Severity of disease caused by *Mycosphaerella cryptica* (COOK) HANSF. was assessed on the adult foliage of *Eucalyptus globulus* LABILL. in two provenance trials (encompassing all four subspecies) and a progeny trial of *E. globulus* ssp. *globulus* LABILL. located in Victoria, Australia. Disease was relatively low in all trials (most trees with less than 15% crown severity), except for two provenances at one trial, Judbury (*E. globulus* ssp. *globulus*) and Mansfield (*E. globulus* ssp. *bicostata*), that had mean crown severities of approximately 25% and 40%, respectively. *Eucalyptus globulus* ssp. *bicostata* (MAIDEN *et al.*) KIRKPATR. was significantly ($P < 0.01$) more susceptible than *E. globulus* ssp. *globulus*, *E. globulus* ssp. *pseudoglobulus* (NAUDIN ex MAIDEN) KIRKPATR. and *E. globulus* ssp. *maidenii* (F. MUELL.) KIRKPATR., with subspecies *maidenii* significantly less diseased than all other subspecies. There was significant variation between provenances within subspecies *globulus* ($P < 0.01$) but not within subspecies *pseudoglobulus*, *maidenii* or *bicostata*. Subspecies *globulus* also showed significant ($P < 0.01$) variation between families. There was a moderate to high genetic correlation between disease of the adult foliage and disease of the juvenile foliage (caused by both *M. cryptica* and *M. nubilosa* (COOKE) HANSF.) assessed several years earlier, both at the provenance ($r_G = 0.67$) and family ($r_G = 0.33$) levels. Narrow sense heritability of disease of the adult foliage (*M. cryptica*) was low ($h^2 = 0.17$), compared to that of the juvenile foliage ($h^2 = 0.35$) and juvenile defoliation ($h^2 = 0.45$) assessed previously. Selection for overall disease resistance (both adult- and juvenile-phase foliage) can be carried out more quickly and accurately at the juvenile stage when trees are 2–3 years old, potentially reducing the time required for resistant trees to be selected and deployed in the field. Mycosphaerella leaf disease on adult *E. nitens* (DEAN and MAIDEN) MAIDEN was also assessed in two provenance trials; however, there was very little disease observed and no significant differences were found between provenances.

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Key words: provenances, families, *Mycosphaerella nubilosa*, adult-juvenile leaf phase correlations, genetic parameters.

Introduction

Eucalyptus globulus LABILL., which is endemic to south-eastern Australia, is planted extensively throughout the temperate regions of the world and is the focus of intensive breeding programs (eg. VOLKER and ORME, 1988; VOLKER and RAYMOND, 1989; BORRALHO *et al.,* 1992). Four subspecies of *E. globulus* have been described: *E. globulus* ssp. *globulus* LABILL., *E. globulus* ssp. *pseudoglobulus* (NAUDIN ex MAIDEN) KIRKPATR., *E. globulus* ssp. bicostata (MAIDEN *et al.*) KIRKPATR. and *E. globulus* ssp. *maidenii* (F. MUELL.) KIRKPATR. (KIRKPATRICK, 1974; JORDON *et al.,* 1993). Of these, subspecies *globulus* is the most commonly planted in south-eastern and south-western Australia because of its generally superior growth rate and pulping qualities (VOLKER and RAYMOND, 1989; BORRALHO *et al.,* 1992). As a consequence of the increased economic importance of *E. globulus* the fungi that occur on it, and the damage they cause, have been studied more intensively in recent years.

Leaf diseases caused by species of *Mycosphaerella* JOHANSON have become a serious problem on *E. globulus* in Australia (PARK and KEANE, 1982a, b; CARNEGIE *et al.,* 1994; DUNGEY *et al.,* 1997; MILGATE *et al.,* 2001; MAXWELL *et al.,* 2003; CARNEGIE and ADES, 2003), New Zealand (HOOD *et al.,* 2002) and Chile (AHUMADA *et al.,* 2003). *Mycosphaerella* species also cause significant damage to *E. nitens* (DEAN and MAIDEN) MAIDEN in Australia (DUNGEY *et al.,* 1997; CARNEGIE *et al.,* 1998), New Zealand (HOOD *et al.,* 2002) and South Africa (LUNDQUIST and PURNELL, 1987; CROUS and WINGFIELD, 1996; HUNTER *et al.,* 2004).

Both *E. globulus* and *E. nitens* are markedly heterophyllous (WILLIAMS and BROOKER, 1989), with distinct differences in juvenile- and adult-phase foliage. Juvenile foliage of *E. globulus* is susceptible to both *M. nubilosa* (COOKE) HANSF. and *M. cryptica* (COOKE) HANSF. (PARK and KEANE, 1982a; CROUS *et al.,* 1995), with *M. nubilosa* commonly causing the most damage (CARNEGIE *et al.,* 1994; MILGATE *et al.,* 2001; MAXWELL *et al.,* 2003). This highly susceptible juvenile foliage phase lasts only for 2-3 years and is replaced by intermediate then adult foliage, which is highly resistant to *M. nubilosa* but still susceptible to *M. cryptica* (PARK and KEANE, 1982a, b; CROUS *et al.,* 1995; CARNEGIE and ADES, 2002). *Mycosphaerella nubilosa* has been isolated from adult foliage of *E. globulus* only in recent years, and only very rarely (MAXWELL *et al.,* 2003; BARBER, 2005; KULARATNE *et al.,* 2004). Juvenile foliage of *E. nitens* is susceptible to *M. cryptica* and not *M. nubilosa* in Australia (MARKS *et al.,* 1982; PARK and KEANE, 1982b; CARNEGIE *et al.,* 1998; CARNEGIE and ADES, 2002), but in South Africa it is susceptible to *M. nubilosa* (HUNTER *et al.,* 2004). The reasons for these conflicting results have not yet been resolved. *Mycosphaerella cryptica* has not been recorded from South Africa (CROUS and WINGFIELD, 1996; HUNTER *et al.,* 2004). Adult foliage of *E. nitens* remains susceptible to *M. cryptica* but not *M. nubilosa* in Australia (CROUS *et al.,* 1995; CARNEGIE and ADES, 2002). Thus *M. cryptica* can cause problems throughout the life of the plantation and, while usually causing less dramatic symptoms, may ultimately cause the larger loss in growth.

Mycosphaerella leaf disease (caused by both pathogens, either singly or together) can cause premature defoliation of affected trees (up to 90% in severe cases) and has been known to markedly affect the growth and form of young, susceptible trees (DICK, 1982; LUNDQUIST and PURNELL, 1987; CARNEGIE *et al.,* 1994; CARNEGIE and ADES, 2003).

Wide variation in susceptibility to Mycosphaerella leaf disease has been reported between *Eucalyptus* species (CARNEGIE *et al.,* 1998; HOOD *et al.,* 2002), provenances (WILCOX, 1982a; PURNELL and LUNDQUIST, 1986; CARNEGIE *et al.,* 1994, 1998; DUNGEY *et al.,* 1997; HOOD *et al.,* 2002) and families (WILCOX, 1982b; REINOSO, 1992; STEFANATOS, 1993; DUNGEY *et al.,* 1997; BARBER and KEANE, 2001; CARNEGIE *et al.,* 2004). Most research has focused on young eucalypt plantations where trees are still in their juvenile-phase foliage, as this is when the disease is most dramatic and is relatively easy to assess. Standard disease assessment diagrams for severity and defoliation have been developed for use in 2-3-year-old stands (LUNDQUIST and PURNELL, 1987; CARNEGIE *et al.,* 1994) and these have been used widely (e.g., REINOSO, 1992; STEFANATOS, 1993; DUNGEY *et al.,* 1997). However, as both *E. globulus* and *E. nitens* remain susceptible to *M. cryptica* throughout the rotation it is important to understand variation in susceptibility at later ages. If susceptibility in the adult phase is substantially genetically correlated with that in the juvenile phase then practical breeding for resistance would be a great deal easier than it could otherwise have been as indirect selection for adult resistance could be carried out successfully at the juvenile stage.

This paper describes how the field assessment technique has been successfully extended to disease caused by *M. cryptica* on adult foliage on older trees to assess two *E. globulus* provenance trials and one *E. globulus* progeny trial. One of the provenance trials and the progeny trial were previously assessed during the juvenile growth phase for severity of Mycosphaerella leaf disease, jointly caused by *M. nubilosa* and *M. cryptica* (REINOSO, 1992; CARNEGIE *et al.,* 1994). Two *E. nitens* provenance trials were also assessed for disease caused by *M. cryptica* on the juvenile and adult foliage.

Materials and Methods

Description of trials

Silver creek: Established by Grand Ridge Plantations in June 1986 at Silver Creek, Victoria, Australia (36° 23' S, 146° 44' E). It consists of 28 provenances of *E. globulus*, encompassing all four subspecies (*Table 1*), and four provenances of *E. nitens* (*Table 2*). The experimental design for the trial was a randomised complete block with single-tree plots and 40 replicates, with trees planted at a square spacing of 3 m.

Tostaree: A eucalypt species and provenance trial established in October 1988 by the Department of Sus-

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tainability and Environment, Victoria, near Nowa Nowa in Victoria, Australia (37° 47' S, 148° 11' E). The experimental design was a randomised complete block with 25-tree square plots and 5 replicates. Trees were spaced 3 m x 3 m on a square grid. Nine provenances of *E. globulus* (encompassing all four subspecies) (*Table 3*) and six provenances of *E. nitens* (*Table 2*) were assessed. This trial was previously assessed for severity of Mycosphaerella leaf disease in the juvenile growth phase in 1991 (CARNEGIE *et al.,* 1994, 1998).

Mount Worth: This *E. globulus* ssp. *globulus* progeny trial was established in June 1989 near Mount Worth State Park, Victoria, Australia (38° 16' S, 145° 59' E) by Grand Ridge Plantations. It compares 52 open pollinated families collected from wild trees growing in the vicinity of Apollo Bay, Victoria (38° 46' S, 143° 40' E). The experimental design for the trial was a randomised complete block with single-tree plots and 20 replicates. Trees were spaced 3.6 m x 2.8 m on a rectangular grid. This trial was previously assessed for severity of Mycosphaerella leaf disease in the juvenile growth phase in 1992 (REINOSO, 1992).

Assessment of Mycosphaerella leaf disease on the adult foliage

Mycosphaerella cryptica on adult foliage was associated with irregular lesions, often 10mm wide by 20 mm long with callused margins and distorting the leaf, similar to those described by CARNEGIE and ADES (2002). Due to the size of trees (many over 15 m tall), an ordinal disease assessment scale was developed for subjectively assessing severity of *M. cryptica* in the whole crown: 0 (no infection), 1 (< 1% leaf area affected), 2 (1 to < 15%) and 3 (> 15%). This scale was used for assessing the Silver Creek trial but for subsequent assessment of the Tostaree and Mount Worth trials, a finer scale with more categories was used: 0, 1 (< 1%), 2 (1 to < 15%), 3 (15% to < 30%), 4 (30% to < 45%) and 5 (> 45%). This was developed to account for higher disease in trees at Tostaree, especially in provenances of subspecies *bicostata*. The whole crown of each tree was assessed for severity of *M. cryptica*, with the aid of binoculars, by an observer walking along the rows.

At Silver Creek, each tree in 20 replicates was assessed for *M. cryptica* on adult foliage in December 1992 and again in March 1993. At Tostaree, the nine internal trees from each plot of *E. globulus* and *E. nitens* in the five replicates were assessed in March 1993. Each tree in 10 replicates was assessed at Mount Worth in early April 1994.

Statistical analyses

Analyses of variance were carried out for the severity scores for all sites to test for variation in susceptibility of adult foliage to disease, and for both assessments of *M. cryptica* at Silver Creek to test for provenance by time interaction. Genetic and phenotypic correlations were estimated between disease severity in juvenile and adult foliage for families at Mount Worth, and phenotypic and "genetic" correlations for provenances at Tostaree. All analyses were carried out using the GLM procedure of SAS (SAS, 1996). At Silver Creek there were no significant differences in disease scores between the original 21 Jeeralang provenances. All were collected from different trees along a forest road, so they were combined as one provenance (Jeeralang) for analysis.

Genetic parameters

SQUILLACE (1974) suggested that in real situations the coefficient of relationship between putative half-sibs from open-pollinated seedlots will usually be appreciably larger than that which is commonly used (1/4) and that this will cause an overestimate of genetic variance and therefore of heritability. MORAN and BELL (1983) estimated outcrossing rates for a wide range of *Eucalyptus* species and determined an average rate of outcrossing of 70%. In this study, narrow-sense individual tree heritability (h^2), additive genetic variance and covariance were estimated assuming an average outcrossing rate of 70% resulting in a coefficient of relationship among the sibs of $1/_{2.5}$. This value has been used by various authors to estimate genetic parameters from open-pollinated families of *E. globulus* (e.g., VOLKER *et al.,* 1990; REINOSO, 1992; DUNGEY *et al.,* 1997) and *E. nitens* (DUNGEY *et al.,* 1997).

Under this assumption, additive genetic variance was estimated as 2.5 times the family variance and narrow sense heritability was calculated as:

$$h^2 = 2.5\hat{\sigma}^2_f / (\hat{\sigma}^2_e + \hat{\sigma}^2_f + \hat{\sigma}^2_r)$$

where $\hat{\sigma}^2_e$, $\hat{\sigma}^2_f$ and $\hat{\sigma}^2_r$ represent respectively the residual, family and replicate variance components.

Least square estimates of variance and covariance components were obtained by equating expected and actual mean squares and products. Additive genetic correlations between traits were calculated by using additive genetic variances and covariances as indicated by HAZEL *et al.* (1943). These genetic parameters were calculated for families at Mount Worth.

A "genetic correlation" between traits (disease in the juvenile foliage and disease in the adult foliage) were calculated for provenance means at Tostaree using the formula:

$$r_G = Cov_{1,2} / \hat{\sigma}_1 \hat{\sigma}_2$$

where $Cov_{1,2}$ is the provenance covariance component of traits 1 (disease in the juvenile foliage) and 2 (disease in the adult foliage), $\hat{\sigma}_1$ and $\hat{\sigma}_2$ are the square roots of the provenance variance components.

Results

Silver Creek

Overall, disease in this trial was low, with most trees having a score of 0 or 1 (i.e. less than 1% crown severity). Many trees in subspecies bicostata had higher scores (many trees with a score of 2 and rarely 3), but still with provenance averages of less than 15% crown severity (mean scores less than 2). There were significant ($P < 0.01$) differences in disease severity on adult foliage between the four *E. globulus* subspecies at Silver

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Table 1. – Subspecies, origin and disease severity caused by *M. cryptica* on the adult foliage (mean of 2 assessments[1]) of the *E. globulus* provenances at Silver Creek.

E. globulus subspecies	Origin				Mean[1] disease severity (score)
	Provenance	Latitude o "S	Longitude o "E	Elevation m	
bicostata	Mount Cole, Vic[2]	37 17	143 17	700	1.69
bicostata	Toombullup, Vic	36 53	146 00	840	1.20
bicostata	Strathbogie, Vic	36 51	145 44	870	1.93
globulus	Macquarie Harbour, Tas	42 24	145 14	20	0.02
globulus	Henty River, Tas	42 01	145 17	50	0.08
globulus	Leprena, Tas	43 31	146 55	20	0.16
globulus	Bruny Island, Tas	43 23	147 19	180	0.14
globulus	Channel, Tas	43 15	147 11	100	0.16
globulus	Geeveston, Tas	43 12	146 58	200	0.17
globulus	Denison, Tas	42 59	146 50	240	0.32
globulus	Uxbridge, Tas	42 47	146 50	500	0.81
globulus	Jericho, Tas	42 25	147 45	500	0.21
globulus	Pepper Hill, Tas	41 36	147 47	560	0.30
globulus	Taranna, Tas	43 02	147 53	120	0.22
globulus	Swansea, Tas	42 08	148 02	100	0.12
globulus	Rheban, Tas	42 40	147 53	80	0.09
globulus	Seymour, Tas	41 13	148 17	20	0.07
globulus	Scamander, Tas	41 27	148 13	50	0.05
globulus	St. Helens, Tas	41 12	148 15	50	0.18
globulus	North Flinders Island, Tas	39 47	147 55	50	0
globulus	South Flinders Island, Tas	40 13	148 02	50	0.01
globulus	Otways, Vic	38 40	143 44	44	0
globulus	Portugal	-	-	-	0.18
maidenii	Tantawanglo, NSW	36 46	149 38	380	0
maidenii	Mount Dromedary, NSW	36 36	150 04	305	0.03
maidenii	Nerrigundah, NSW	36 07	149 54	245	0
pseudoglobulus	Jeeralang, Vic[3]	38 34	146 15	400	0.05
pseudoglobulus	Kuark, Vic	37 35	148 46	-	0.18
Least Significant Difference (P < 0.01)					0.22

[1] Mean disease scores for December and March are presented for ease in comparison of provenances.

[2] Vic = Victoria; Tas = Tasmania; NSW = New South Wales.

[3] There were no significant differences in disease scores between the original 21 Jeeralang provenances. All were collected from different trees along a forest road, so they were combined as one provenance ("Jeeralang") for analysis in this study.

Creek (*Tables 1* and *4*) with subspecies bicostata being significantly more susceptible than subspecies *globulus*, *pseudoglobulus* and *maidenii*, and subspecies *maidenii* significantly less susceptible than the other three. Subspecies *bicostata* had the highest mean severity in December (1.72) and March (1.50), and these values were significantly higher than all other subspecies. Subspecies *globulus* was the next most severely affected with a mean of 0.14 in December and 0.20 in March while subspecies *pseudoglobulus* had a mean severity of 0.06 in December and 0.19 in March. Subspecies *maidenii* had very low severity scores, with only one tree having any trace of the disease (score of 1) in the trial in either year.

Disease severity differed significantly between the 20 provenances within subspecies *globulus* ($P < 0.01$), but variation in disease severity within subspecies *pseudoglobulus*, *bicostata* or *maidenii* was not significant (*Tables 1* and *5*). Provenances of subspecies *bicostata* were the most severely affected with mean scores ranging from 1.20 (Toombullup) to 1.93 (Strathbogie). Uxbridge had the highest mean disease score within subspecies *globulus* with 0.81, which was higher than for any provenance other than the three in subspecies *bicostata.* Denison was the next most severely affected, and the fifth overall, with a score of 0.32, while Pepper Hill had a severity score of 0.30. No disease was recorded in either year on a few provenances of subspecies *globulus* (viz. North Flinders Island and Otways). Severity scores for the remaining subspecies *globulus* provenances ranged from 0.01 to 0.22.

In general, disease severity increased for each provenance from December 1992 to March 1993 at Silver Creek (data not shown) and there was a high phenotypic correlation ($r = 0.47$, $P < 0.01$) between provenance means for the two assessment dates. Mean severity for subspecies *globulus* and subspecies *pseudoglobulus* increased from 0.14 to 0.20 and 0.06 to 0.19, respectively, in the three months from December 1992 to March 1993. In both subspecies, severity for some provenances increased markedly in the three months between assessments. Portugal (subspecies *globulus*) increased from 0.06 to 0.31, while for some of the Jeeralang families (subspecies *pseudoglobulus*) the average score increased

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three-fold. Disease was observed for subspecies *maidenii* only at the March 1993 assessment, there being no disease seen in these trees in December 1992. For subspecies *bicostata* there was a decrease in mean disease severity from 1992 (1.72) to 1993 (1.50). Severity on Mount Cole and Strathbogie decreased from 1.84 and 2.14 to 1.53 and 1.71, respectively, between December 1992 and March 1993, and hence brought down the average for this subspecies. Premature shedding of leaves severely affected in December 1992 may account for the reduction in observed disease severity in March 1993. 'Shot-hole' damage (holes in leaves due to biotic agents such as insects or fungi) to the leaves of these and other trees was observed during the 1993 assessment but it could not be determined for certain whether this was damage caused by *M. cryptica* or by herbivorous insects. This may also account for the reduction.

Severity of *M. cryptica* on adult *E. nitens* foliage at Silver Creek was very low, with few trees having any disease and these only in one provenance (*Table 2*). There were no significant differences between provenances so correlations with disease of juvenile foliage were not calculated.

Table 2. – *E. nitens* provenances assessed at Silver Creek (SC) and Tostaree (T): origin and severity of *M. cryptica* (score) on the adult foliage in March 1993.

Provenance	Origin		Adult Severity
	Latitude o "S	Longitude o "E	
Snobs Creek, Vic (SC)	37 16	145 53	0
Snobs Creek, Vic (SC)	37 16	145 53	0
Mount St. Gwinear, Vic (SC)	-	-	0
Loch Valley, Vic (SC)	37 48	146 01	0.08
Mt. Erica, Vic (T)	37 53	146 21	0.06
Mt. Kaye, Vic (T)	37 24	149 15	0.17
Powelltown, Vic (T)	37 52	145 45	0.17
Snobs Creek, Vic (T)	37 16	145 53	0.06
Tallaganda, NSW (T)	35 39	149 39	0.11
Tooronga Plateau, Vic (T)	37 50	146 06	0.06

Tostaree

Disease severity was higher at Tostaree than either Silver Creek or Mount Worth. Most trees scored 0, 1 or 2; with most provenance means below 2 (i.e. crown severity of less than 15%). However, two provenances had higher means: Judbury (subspecies *globulus*) had a mean score of 2.89, which equates to approximately 25% crown severity, and Mansfield (subspecies *bicostata*) had a mean score of 4.49, which equates to a crown severity of approximately 40%.

There were highly significant ($P < 0.01$) differences in disease severity for adult foliage between the *E. globulus* subspecies and provenances assessed at Tostaree in March 1993 (*Table 3*). The Mansfield provenance, subspecies *bicostata*, had the highest adult disease score with 4.49 and Judbury, subspecies *globulus*, was the next most severely affected provenance with a mean of 2.89. Eden, subspecies *maidenii*, and Wye River, subspecies *globulus*, had the lowest severities for adult foliage with 0.56 and 0.50, respectively. There was wide variation within subspecies *globulus* which ranged from 0.50 (Wye River) through to 1.38 (Otway National Park) and up to 2.89 (Judbury). Note that Wye River and Otway National Park are located in close geographical proximity to one another in the Otway region in western Victoria but have almost a three-fold difference in disease severity.

There was a moderately high phenotypic correlation of the provenance means ($r = 0.53$, $P < 0.01$) between severity of *M. cryptica* on the adult foliage and disease severity on the juvenile foliage at Tostaree (juvenile foliage data from CARNEGIE *et al.,* 1994). The "genetic correlation" between disease on the juvenile foliage phase with that in the adult foliage phase was moderately high ($r_G = 0.69$). In both leaf phases Judbury (subspecies *globulus*) and Mansfield (subspecies *bicostata*) had the highest disease severity scores while Eden (subspecies *maidenii*) and Kuark (subspecies *pseudoglobulus*) had amongst the lowest. The ranking of the provenances didn't change greatly between assessments of juvenile and adult foliage, except for Wye River (subspecies *globulus*), which had moderate severity in 1991 (juvenile leaf phase) but had the lowest severity when assessed in the adult phase (1993).

Table 3. – Subspecies, origin and disease severity of *M. cryptica* on adult foliage and severity of Mycosphaerella leaf disease on juvenile foliage[1] of *E. globulus* provenances at Tostaree (untransformed scores).

E. globulus subspecies	Provenance	Origin			Disease severity	
		Latitude o "S	Longitude o "E	Elevation m	Adult	Juvenile[1]
bicostata	Mansfield, Vic	37 03	146 05	160	4.49	23.1
globulus	Judbury, Tas	42 59	146 55	100	2.89	26.9
globulus	King Is., Tas	38 46	143 32	200	0.96	19.9
globulus	Otway Nat. Pk, Vic	36 55	149 30	-	1.38	21.6
globulus	St. Helens, Tas	38 34	146 15	400	1.33	18.7
globulus	Wye River, Vic	37 35	148 46	-	0.50	14.8
maidenii	Eden, NSW	36 52	146 11	780	0.58	4.2
pseudoglobulus	Jeeralang, Vic	38 23	146 27	-	1.47	17.5
pseudoglobulus	Kuark, Vic	41 12	148 15	50	0.78	9.8
Least Significant Difference ($P < 0.01$)					0.37	-

[1] From CARNEGIE *et al.* (1994)

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Table 4. – Analysis of variance of mean severity score (rank transformed[1]) on adult-phase foliage for subspecies of *E. globulus* at Silver Creek.

Source of variation	df	Mean square	F	Prob
Replications	19	0.262	7.26	0.0001
Year	1	0.001	0.004	0.950
Subspecies	3	23.800	101.624	<0.01
Year*Subspecies	3	0.234	5.74	0.0017
Error	57	0.041		

[1] The rank-transformation procedure was carried out on the Silver Creek data and gave the same conclusion as the parametric ANOVA employed, so according to ZAR (1996, p 267–271), this conclusion is dependable.

Table 5. – Analyses of variance for provenance means (rank transformed[1]) within *E. globulus* subspecies at Silver Creek.

Subspecies	*bicostata*				*globulus*				*maidenii*				*pseudoglobulus*			
Source of variation	df	Mean square	F	Prob	df	Mean square	F	Prob	df	Mean square	F	Prob	df	Mean square	F	Prob
Replications	19	238.51	1.36	0.225	19	7573.61	2.36	0.001	19	9.63	0.70	0.783	19	138.84	2.26	0.05
Provenance	2	58.32	0.33	0.719	19	10015.14	3.11	0.001	2	13.49	0.98	0.388	1	343.06	5.60	0.31
Error	27	174.78			286	3215.47			26	13.73			16	61.31		

[1] The rank-transformation procedure was carried out on the Silver Creek data and gave the same conclusion as the parametric ANOVA employed, so according to ZAR (1996, p 267–271), this conclusion is dependable.

Table 6. – Analysis of variance for family means of disease caused by *M. cryptica* on the adult foliage at Mount Worth.

Source of variation	df	Mean square	F	Prob
Replications	19	1.677	2.29	0.001
Family	36	1.634	2.24	0.001
Error	476	0.731		

Again very low levels of disease were observed on the adult foliage of the *E. nitens* at Tostaree, with only one to three trees in each provenance with any observable disease, and this only a score of 1 (*Table 2*). Consequently, there were no significant differences between provenances, and no correlations with juvenile foliage were calculated.

Mount Worth

Disease was low in this trial, with most trees scoring 0, 1, or 2, and mean severity of *M. cryptica* (scores) ranging from 0.30 to 2.02 (i.e. mean crown severity of less than 15%) (data not shown). Wide variation in severity of *M. cryptica* in the adult foliage was observed between the *E. globulus* ssp. *globulus* families at Mount Worth (*Table 6*) and differences between families were highly significant ($P < 0.01$). Data of REINOSO (1992) of severity of Mycosphaerella leaf disease and defoliation of the juvenile foliage at Mount Worth was re-analysed, with some coding errors corrected and runts removed, and revealed significant ($P < 0.01$) variation between families. There was no significant phenotypic correlation in disease severity between the two phases (*Table 7*). However, there was a significant negative phenotypic correlation ($r = –0.12$, $P < 0.01$) between severity of *M. cryptica* on the adult foliage and defoliation caused by Mycosphaerella leaf disease in the juvenile growth phase and a low but significant positive phenotypic correlation ($r = 0.17$, $P < 0.01$) between disease severity on the juvenile foliage and defoliation of the juvenile crown (*Table 7*).

Estimated heritabilities and genetic correlations (r_G) of all traits assessed at Mount Worth are given in *Table 7*. Narrow sense heritability for adult disease was low (0.17) compared to the higher heritabilities of juvenile severity (0.35) and defoliation (0.45). There was a moderate positive genetic correlation between *M. cryptica* on the adult foliage and juvenile severity (0.33). In con-

Table 7. – Estimates of genetic correlations (r_G) (above the diagonal), phenotypic correlations (below the diagonal), and individual heritability (h^2) (in bold) for damage traits on juvenile foliage[1] and adult foliage at Mount Worth.

Trait	*M. cryptica* on adult foliage	Mycosphaerella leaf disease on juvenile foliage	Defoliation of juvenile crown
M. cryptica on adult foliage	**0.17**	0.33	-0.46
Mycosphaerella leaf disease on juvenile foliage	ns	**0.35**	0.20
Defoliation of juvenile crown	-0.12	0.17	**0.45**

[1] From REINOSO (1992).

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trast, there was a negative genetic correlation between *M. cryptica* on the adult foliage and defoliation of the juvenile crown (–0.46).

Discussion

Disease severity in the three trials was relatively low, with most trees having a score of less than 2, which equates to a crown severity of less than 15%. Disease severity overall was higher at Tostaree, with two provenances in this trial having significantly higher damage: Judbury (subspecies *globulus*) with a mean score of 2.89 (equates to approximately 25% crown severity) and Mansfield (subspecies *bicostata*) with a mean score of 4.49 (equates to approximately 40% crown severity). This indicates that disease caused by *M. cryptica* in adult foliage, at least in most provenances, is not a significant health threat to older trees.

The disease assessment scale devised here for field-based assessments proved effective in establishing the relative susceptibility of the various *E. globulus* subspecies, provenances and families to damage on adult foliage caused by *M. cryptica.* Other rating scales developed for assessing leaf diseases of *Eucalyptus* have also proved useful (e.g., for Mycosphaerella leaf disease on juvenile foliage (Lundquist and PURNELL, 1987; CARNEGIE et al., 1994); for target spot caused by *Aulographina eucalypti* (COOKE and MASSEE) ARX and MÜLLER (CARNEGIE and KEANE, 2003); and for *Phaeophleospora epicoccoides* (COOKE and MASSEE) CROUS *et al.* (= *Phaeseptoria eucalypti*) (NICHOL *et al.,* 1992). Whereas the above authors assessed a single branch chosen at random, the method of assessment in this study was of the whole (adult) crown, a method which has proven to be more effective in assessing Mycosphaerella leaf disease on juvenile foliage of *E. globulus* (REINOSO, 1992; STEFANATOS, 1993; DUNGEY *et al.,* 1997; CARNEGIE and ADES, 2003) and *E. pilularis* SMITH (CARNEGIE *et al.,* 2004) and adult foliage of *E. globulus* (CARNEGIE and ADES, 2001). A method that also assesses the whole crown for damage, the Crown Damage Index (CDI), has recently been developed as a standard for assessment of insect and fungal damage to young eucalypt trees (STONE *et al.,* 2003a, b).

There are, however, possible weaknesses in using our scoring and analysis methods. Since the scale is not linear, differences between classes of damage (e.g., between class 0 and class 1, and between class 2 and class 3) are not of the same magnitude. This problem could have been avoided by measuring every tree according to its percentage of infection or by adopting a mid-point value for analysis. Assessing the percentage of infection (such as the CDI, STONE *et al.,* 2003b) was not possible as we were assessing crowns of trees that were mostly over 15 m tall. Our primary aim of this study was to rank the provenances for their susceptibility of *M. cryptica*, which our analysis did sufficiently. However, using a mid-point for analysis would have shown the magnitude of differences more clearly.

Wide variation in susceptibility of the adult foliage to *M. cryptica* was observed between the *E. globulus* subspecies at both provenance trials, with provenances of subspecies *bicostata* significantly more susceptible than provenances within subspecies *globulus*, *pseudoglobulus* and *maidenii*. Provenances from subspecies *bicostata* have previously been observed as having the most severely diseased juvenile foliage in other trials (CARNEGIE *et al.,* 1994; HOOD *et al.,* 2002). However, an unknown provenance of subspecies *bicostata* was the least damaged in a trial in north-west Tasmania (DUNGEY *et al.,* 1997) with both the adult and juvenile foliage being moderately resistant to *Mycosphaerella* spp. This suggests that there is more variation in subspecies *bicostata* than has previously been reported, and this subspecies should not be discounted for commercial plantations based on disease alone, as has previously been suggested (CARNEGIE *et al.,* 1994).

There was very little damage by *M. cryptica* on the three provenances from subspecies *maidenii* at Silver Creek, with provenances from this subspecies consistently less diseased than the majority of provenances from subspecies *globulus*, *pseudoglobulus* and *bicostata,* an observation that has been made previously (CARNEGIE *et al.,* 1994; HOOD *et al.,* 2002). This subspecies is not grown commercially in plantations in Australia. However, this work suggests that it may well need to be re-considered, assuming growth and pulping characteristics are suitable, if *E. globulus* is to be grown successfully in areas where *Mycosphaerella* species cause severe damage and growth loss. In a trial in northern New South Wales, Australia, subspecies *maidenii* is outperforming the widely planted *E. dunnii* MAIDEN, *E. grandis* HILL ex MAIDEN, *Corymbia citriodora* ssp. *variegata* (F. MUELL.) MCDONALD and BEAN and *E. grandis* hybrids in terms of growth and pest and disease resistance at five years of age (CARNEGIE and JOHNSON, unpublished data).

There were no significant differences in disease severity of adult foliage amongst provenances within subspecies *bicostata* and *maidenii* at Silver Creek. However, there were only three provenances of each subspecies, and a more extensive trial, with more provenances, may reveal more variation within these subspecies. The need to include sufficient numbers of genotypes when making comparisons has been noted previously (DUNGEY *et al.,* 1997). There was little difference in disease severity between the two provenances from subspecies *pseudoglobulus* planted at Silver Creek (Kuark and Jeeralang), but a significant difference between the same provenances (although they were different seedlots) planted at Tostaree, with Jeeralang being moderately diseased and Kuark significantly less so. This suggests that there is wide variation within *pseudoglobulus* from Jeeralang, even though this was not observed in the provenances studied at Silver Creek. At both sites, the Kuark seedlots had among the lowest disease scores of all *E. globulus* provenances. Disease at Tostaree was more severe overall than that observed at Silver Creek, and this may have affected the relative susceptibility of the two provenances at the two sites, as has been observed for other forest diseases (WHITE and HODGE, 1989).

Provenances within subspecies *globulus* consistently showed wide variation in susceptibility of adult foliage

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to *M. cryptica* in this study. At Silver Creek, Uxbridge was the fourth most diseased provenance, after the three subspecies *bicostata* provenances. Denison and Pepper Hill also had high levels of disease at Silver Creek while, in contrast, Macquarie Harbour, Scamander, North and South Flinders Island and Otways had no or very little disease. Subspecies *globulus* provenance Judbury was the second most severely diseased at Tostaree, and Wye River recorded the least disease in that trial. Both these provenances were significantly more and less diseased, respectively, than all other subspecies *globulus* planted at Tostaree. The remaining provenances of subspecies *globulus* at both sites had low to moderate severity, with no significant variation between them.

Climatic conditions in the place of origin of the seed source have previously been identified to explain variation in *Eucalyptus* in disease susceptibility by species of *Mycosphaerella* (WILCOX, 1982a; LUNDQUIST and PURNELL, 1987; CARNEGIE *et al.,* 1994, 1998; DUNGEY *et al.,* 1997; CARNEGIE *et al.,* 2004). Warm, wet weather (i.e. summer rain) is reported to increase the chances of successful infection by species of *Mycosphaerella* (CHEAH, 1977; PARK, 1988a). So species of *Eucalyptus* that originated from areas where these conditions predominate may have evolved under conditions with high populations of these leaf-infecting fungi, and hence, may have had more intense natural selection for resistance to *Mycosphaerella* spp. In contrast, trees from environments that were cooler or with dry summers would likely have had little previous exposure to *Mycosphaerella,* and thus are likely to be severely damaged when exposed to these pathogens.

Similar observations have been reported for diseases in other tree genera (eg. ZHANG *et al.,* 1996; YANG *et al.,* 1997) and were also made in the current study. The majority of the provenances with severe disease at Tostaree and Silver Creek are from areas where summer rainfall and/or mean temperatures are low (e.g., Mount Cole, Toombulup and Strathbogie from subspecies *bicostata*, and Uxbridge, Judbury, Denison and Pepper Hill from subspecies *globulus*). In contrast, provenances that had no or very little disease were from areas where summer rainfall and/or mean maximum temperatures were relatively high (eg. Macquarie Harbour and Otways from subspecies *globulus*, Jeeralang from subspecies *pseudoglobulus*, and the three subspecies *maidenii* provenances, Tantawanglo, Mount Dromedary and Nerrigundah). Preliminary analysis indicates that disease severity of provenances planted at Silver Creek is negatively correlated with mean maximum yearly temperature ($r = -0.35$) and mean summer rainfall ($r = -0.28$) in their original location (CARNEGIE, unpublished data). In contrast, HOOD *et al.* (2002) reported no effect of climatic conditions in the place of origin to explain variation in *Eucalyptus* in disease susceptibility by species of *Mycosphaerella.* More studies are needed to fully understand whether climatic conditions have led to evolution of this variation in susceptibility to *Mycosphaerella* leaf disease amongst these provenances.

Detailed studies of morphological variation in *E. globulus* have been conducted to investigate geographic patterns of genetic variation (JORDAN *et al.,* 1994; DUTKOWSKI and POTTS, 1999). This work resulted in the identification of races within *E. globulus* spp. *globulus* based on variation in growth and morphological traits and geographic distribution. These "races" may also help to explain the variation in *Mycosphaerella* damage in the *E. globulus* provenances and subspecies *globulus* families studied here.

There is also considerable variation within subspecies *globulus* in the persistence of the juvenile foliage, with trees from localities in the Strzelecki Ranges in Victoria, and the north-eastern and south-eastern coastal areas of Tasmania producing juvenile foliage for longer than trees from other localities (DUTKOWSKI and POTTS, 1999). Since both *M. nubilosa* and *M. cryptica* infect juvenile foliage of *E. globulus*, it is likely that trees with more juvenile foliage will experience relatively more damage from these fungi than trees with less juvenile foliage. Assessments of a 3-year-old *E. globulus* provenance trial in Gippsland, Victoria, support this hypothesis. Damage to juvenile foliage was more severe on Judbury (south-east coast, Tasmania) and moderate on St Helens (north-east coast, Tasmania) and Jeeralang (Strezlecki Ranges) when compared to provenances from other regions in Victoria and Tasmania that had lower disease (CARNEGIE *et al.,* 1994). This trait could therefore be used to reduce the impact of *Mycosphaerella* leaf disease by reducing the length of time trees are exposed to the more damaging *M. nubilosa*. However, both susceptibility of the juvenile phase and persistence of the juvenile foliage can vary in *E. globulus*. Juvenile foliage of the Jeeralang provenance is less susceptible to *Mycosphaerella* leaf disease than that of other provenances but there is a longer juvenile foliage phase (REINOSO, 1992).

The narrow sense heritability for severity of *M. cryptica* on the adult foliage at Mount Worth was low (0.17) compared to that for juvenile severity (0.35) at the same trial. This could be due to the low severity of adult disease and the coarseness of the field scoring technique. A finer and more linear scale (eg. increments of 5% like that for juvenile foliage score) would be superior for heritability and genetic gain estimation but was impractical in the field where the observer must visually assess crowns of trees over 15 m tall. Assessment using a finer scale may have differentiated susceptibility of the families more precisely, and thus have resulted in higher estimates of heritability and genetic correlations. Expression of genetic variation in disease resistance may also be dependent on the level of infection (WHITE and HODGE, 1989) and, since the level of infection in adult foliage at the Mount Worth trial was relatively low, these differences may not have been as detectable using the broad assessment scale.

The heritability for adult disease at Mount Worth was lower than those reported by DUNGEY *et al.* (1997), which ranged from 0.19 to 0.34. DUNGEY *et al.* (1997) assessed younger trees that were in the transition from juvenile to adult foliage, thus assessments of adult foliage would have been easier and possibly more accurate as the trees were smaller. Disease was higher in

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the study by DUNGEY *et al.* (1997) compared to Mount Worth, and the lower infection at Mount Worth may have adversely affected calculations of heritability (see WHITE and HODGE, 1989). Also, there was intermediate foliage present on trees assessed by DUNGEY *et al.* (1997), which were included as "adult", whereas there was no intermediate foliage on the trees assessed in the current study. The intermediate foliage included in the assessment by DUNGEY *et al.* (1997) may be more susceptible than the truly adult foliage on older trees at Mount Worth, and the reduction in susceptibility of leaf phases may be progressive – from juvenile to intermediate to adult – thus affecting the result.

The moderate positive genetic correlation between severity of disease on the adult foliage (*M. cryptica*) and severity of disease on the juvenile foliage (*M. cryptica* and *M. nubilosa*) (0.33) suggest that indirect selection may be useful in breeding programs. Again, the scale used makes these estimates less reliable, although the main point is that they are positive. Selection for resistance to *Mycosphaerella* leaf disease of the juvenile foliage can be easily carried out on small trees at age 2–3 years. Therefore, selection can be made at this age and an increase in adult resistance should be achieved indirectly. This bodes well for breeding for resistance, as selection for overall disease resistance (both adult- and juvenile-phase foliage) can be carried out more quickly and accurately at the juvenile stage, potentially reducing the time required for resistant trees to be selected and deployed in the field.

Provenances from the Otway region of Victoria (subspecies *globulus*) are widely used in breeding programs in Australia. Otway National Park and Wye River were moderately affected by *M. cryptica* while Otways was highly resistant, despite the close geographical proximity of these provenances. CARNEGIE *et al.* (1994) reported significant variation in susceptibility of the juvenile foliage between provenances from this region. Similarly, provenances from Jeeralang (subspecies *pseudoglobulus*) are widely used in breeding programs in Australia and wide variation was observed within this provenance. Careful selection of provenances and families from these areas is necessary to achieve the greatest possible gains from plantations established in areas where there is a high risk of *Mycosphaerella* leaf disease.

The severity of *Mycosphaerella* leaf disease increased over time on the adult foliage of *E. globulus* at Silver Creek, confirming previous findings. LUNDQUIST and PURNELL (1987) reported that *Mycosphaerella* leaf disease symptoms on *E. nitens* in South Africa were well advanced by late summer. PARK (1988b) reported that severity of *M. cryptica* on *E. globulus* reached a peak during summer and that this followed high rainfall on consecutive days during warm weather. CARNEGIE *et al.* (1994) also observed that severity of *Mycosphaerella* leaf disease on juvenile foliage of *E. globulus* was highest in summer, following warm, wet conditions. Therefore, assessments of *Mycosphaerella* leaf disease should be carried out in summer when the disease is at it maximum "expression".

At Mount Worth, we observed a negative genetic correlation between disease in adult foliage and defoliation of the juvenile crown, although disease in juvenile and adult foliage was positively correlated. REINOSO (1992) reported very low genetic and phenotypic correlations between juvenile severity and defoliation at Mount Worth, in contrast to the high (r = 0.99, LUNDQUIST and PURNELL, 1987) and moderate (r = 0.53, CARNEGIE *et al.*, 1994) correlations between the same traits reported in other studies. This suggests that *Mycosphaerella* leaf disease was not causing defoliation at Mount Worth. This was discounted by REINOSO (1992), as defoliation was occurring very rapidly at the time of assessment, and leaves that were falling were heavily infected by *M. cryptica* and *M. nubilosa* with no other disease or possible cause evident. The trial was at a high quality site, also removing nutrition as a cause of premature leaf fall. The high genetic correlation between Mount Worth and its paired site (the lower site quality Tom's Cap) indicates the same factor was causing defoliation at both sites. By the same logic differential response to shading can be rejected. It could have occurred at Mount Worth where growth had been very rapid but at Tom's Cap stand growth and crown development were slow and none of the trees was significantly shaded.

Another possibility for the low correlation between juvenile disease and juvenile defoliation at Mount Worth is the consequence of severe infection beginning in the spring and increasing through the season so by the end of the summer most of the juvenile foliage had been lost (REINOSO, 1992; ADES, pers. obs.). Very few new juvenile leaves were produced on these branches subsequently. Defoliation at the time of assessment may have been more highly correlated with juvenile severity earlier in the season. Therefore, defoliation (or juvenile severity for that matter) may not have been assessed at the time for optimum "expression" of this trait. This may explain the negative correlation between defoliation and adult disease that we observed. Defoliation assessed by REINOSO (1992) was probably not a true reflection of the full extent of disease severity for these trees and, if it had been assessed later in the season when defoliation had "run its course", then we would have observed a more accurate reflection of this trait.

CARNEGIE *et al.* (2004) observed a negative correlation between target spot disease of juvenile *E. pilularis* leaves, caused by *A. eucalypti*, and defoliation of the whole crown, assessed at the same time. While contrary to what may be expected, they suggested that it was likely to be a result of *A. eucalypti* being slow growing, and mainly observed on older leaves in the lower and inner crown (WALL and KEANE, 1984). If these same leaves are also infected with *Mycosphaerella* spp. (which they were) they are more likely to be shed more quickly. Once this occurred then little *A. eucalypti* damage would be observed. CARNEGIE *et al.* (2004) assessed three traits – severity of target spot, *Mycosphaerella* leaf disease and defoliation – and suggested that there are optimum times in a season to assess these three different "disease" traits. The very high correlation between sites reported by REINOSO (1992) suggests that juvenile severity and defoliation are measures of different components

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of susceptibility. The low correlation between the two (REINOSO, 1992) suggest that they also should be assessed at different times in the disease cycle.

Disease on adult foliage of *E. nitens* was very low in this trial, consistent with *Mycosphaerella* leaf disease not being a serious problem on adult foliage for this species. However, disease can be severe on juvenile foliage, causing significant defoliation (LUNDQUIST and PURNELL, 1987; DUNGEY *et al.,* 1997; CARNEGIE *et al.,* 1998; HOOD *et al.,* 2002; HUNTER *et al.,* 2004). Species of *Mycosphaerella* were associated with moderate to severe defoliation in mature *E. denticulata* COOK and LADIGES (recently split from *E. nitens*) in Victoria in 1974, although *A. eucalypti* was identified as the main pathogen (NEUMANN and MARKS, 1976).

We recommend a multi-faceted approach to reducing the impact of *Mycosphaerella* leaf disease in *E. globulus* plantations that includes silviculture and genetics. Studies have shown that nutrient addition improves health of trees infected with *Mycosphaerella* spp. (CARNEGIE and ADES, 2001; WARDLAW, pers. comm.), which is likely to be related to tolerance (infected leaves are retained on healthy trees longer) and foliage replacement. Disease severity on juvenile foliage is moderately heritable, and correlated with severity on adult foliage, as is height to phase change. Breeding programs should concentrate on selecting trees with low juvenile disease severity, thus selecting indirectly for adult resistance, and include reduced time in juvenile foliage (assuming good growth and pulping qualities). This can be achieved when trees are 2–3 years old. Once planted, adequate fertiliser regimes should enable trees to tolerate *Mycosphaerella* infection during the juvenile phase and grow into the less susceptible adult foliage sooner, thus reducing the impact of Mycosphaerella leaf disease.

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