Mycosphaerella leaf disease: genetic variation in damage to *Eucalyptus nitens*, *Eucalyptus globulus*, and their F_1 hybrid

H.S. Dungey, B.M. Potts, A.J. Carnegie, and P.K. Ades

Abstract: Severity of *Mycosphaerella* leaf disease was assessed on the adult and juvenile foliage of both controlled crossed and open-pollinated families of *Eucalyptus globulus* ssp. *globulus* Labill., *Eucalyptus nitens* (Deane & Maiden) Maiden, *Eucalyptus globulus* ssp. *bicostata* (Maiden, Blakely & J. Simm.) Kirkpatr., and their F_1 hybrids in a trial in northwest Tasmania, Australia. Within ssp. *globulus*, disease was more severe on one provenance, Taranna, than another, King Island. For interprovenance hybrids, differences between parents were inherited in an additive manner, whereas interspecific hybrids were generally more susceptible than predicted intraspecific midparent values and occasionally, were more susceptible than the more susceptible parent. Within populations, the narrow-sense heritabilities for *Mycosphaerella* disease severity were low to moderate (0.004–0.506), but were consistently higher for adult than for juvenile foliage despite disease severity being higher on juvenile foliage. Parental breeding values and heritabilities estimated from open-pollinated progeny were similar to estimates obtained from controlled crosses involving the same parents. Complex genetic interactions were detected between growth, vegetative phase change, and disease severity. It is possible that selection for rapid growth in an environment without disease may result in indirect selection for susceptibility.

Résumé : La sévérité de la maladie de feuilles causée par *Mycosphaerella* a été évaluée sur le feuillage adulte et juvénile de familles de *Eucalyptus globulus* ssp. *globulus* Labill., *Eucalyptus nitens* (Deane & Maiden) Maiden, *Eucalyptus globulus* ssp. *bicostata* (Maiden, Blakely & J. Simm.) Kirkpatr. et leurs hybrides de première génération obtenus par pollinisation contrôlée et libre dans un essai mené dans le Nord-Ouest de la Tasmanie en Australie. À l'intérieur de la sous-espèce *globulus*, la maladie était plus sévère sur la provenance de Taranna que sur celle de King Island. Dans le cas des hybrides inter-provenances, les différences entre les parents étaient transmises de façon additive, tandis que les hybrides interspécifiques étaient généralement plus sensibles que le prédisaient les valeurs intraspécifiques mitoyennes entre les parents et étaient occasionnellement plus sensibles que le parent le plus sensible. À l'intérieur des populations, les héritabilités génétiques pour la sévérité de la maladie causée par *Mycosphaerella* variaient de faibles à modérées (0,004 à 0,506), mais elles étaient systématiquement plus élevées pour le feuillage adulte que pour le feuillage juvénile même si la maladie était plus sévère sur le feuillage juvénile. Les valeurs en croisement et les héritabilités des descendances de pollinisation libre étaient semblables à celles de pollinisation contrôlée avec les mêmes parents. Il y avait des interactions génétiques complexes entre la croissance, le changement de phase végétative et les niveaux de sévérité de la maladie. Il est possible que la sélection pour la capacité de croissance dans un environnement exempt de maladie sélectionne indirectement pour la sensibilité à la maladie. [Traduit par la Rédaction]

Introduction

Eucalyptus nitens (Deane & Maiden) Maiden and *Eucalyptus globulus* ssp. *globulus* Labill. (hereafter referred to as *E. globulus*), are two of the most important eucalypts for plantations in temperate Australia (Tibbits 1986; Eldridge et al. 1993). *Eucalyptus globulus* is the more widely planted of the two species (e.g., Volker and Orme 1988; Davidson 1989; Eldridge et al. 1993). It occurs naturally in coastal regions of Tasmania, the Bass Strait Islands, and southern Victoria (Kirkpatrick 1975; Jordan et al. 1993). *Eucalyptus nitens* naturally occurs from central Victoria to northern New South Wales in a number of widely disjunct populations (Pederick 1979; Neish et al. 1995). *Eucalyptus nitens* is becoming increasingly important as a plantation species, particularly in Tasmania, largely because of its high level of frost resistance compared with *E. globulus* (Tibbits and Reid 1987*a*, 1987*b*; Volker et al. 1994).

Eucalyptus globulus and *E. nitens* are both susceptible to *Mycosphaerella* leaf disease, which causes leaf necrosis and defoliation (Park and Keane 1982*b*; Wilcox 1982; Purnell and Lundquist 1986; Lundquist and Purnell 1987; Carnegie et al. 1994). Leaf damage can be severe and highly detrimental to growth (Park and Keane 1982*a*; Lundquist 1985; Lundquist and Purnell 1987; Carnegie et al. 1994). Two species of *Mycosphaerella* have been recorded as causing significant disease on *E. globulus*: *Mycosphaerella molleriana* (Thüm.) Lindau (Syn. *M. nubilosa* (Cooke) Hansf., Crous et al. 1991), which occurs on the juvenile foliage, and *Mycosphaerella cryptica* (Cooke) Hansf., which occurs on both juvenile and adult foliage (Park and

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Table 1. The number of individuals in each controlled-cross and open-pollinated family for *E. globulus* intra- and inter-provenance crosses, *E. nitens* × *E. globulus* F_1 hybrids and *E. nitens* half-diallel in the hybrid trial at West Ridgley in 1992.

(A) *E. globulus* factorial

	Male parents																									
Female parents	T 1	T 2	T 3	T 4	T 5	T 6	T 7	T 8	T 9	T 10	T 11	T 12	T 13	T 14	T 15	T 16	K 17	K 18	K 19	K 20	K 21	K 22	K 23	K 24	K 25	K 26	GSOP
K-a	20	19	19	18	20	18	20	16	20	19	20	20	20		18	20	20	20	19	19	20	20	20	20	20	20	17
K-b	20	20	20	19		18	10	16	14	19	20	17	19	20	17	32	14	20		20	19	17	20	13	20	20	19
K-c	20	13				16	12	13	18	12	18	11	18	20	17	14	19	20	20	19		20	19	18	20	20	14
SF-d	20	19		20	16	17	20	19	20	20	15	11	20	14	9	19	20	20	20	20	20	20	20	20	20	20	20
T-e	18	19	10			15	20	13	17		18	18		15	18	20		18		16							13
T-f	15	11	15	15	9	10	15	15	14	7	12	13	14	17	4	14	17							15		13	16
T-g	18	20	20	15	19	18	20	20	20	20	19	19	20	18	18	20	20	20	20	20	20		20	20	20		19
T-h	17	16	12			15		13		16	12	16	12	15		13		19	17		19				16		11
GOP	15	18		19	20	16	10	23		19	9	16	15	14	17	16	19	18	20	20	18	20	20	20	20	19	

(B) F_1 hybrid factorial

	Male parents																									
Female parents	T 1	T 2	T 3	T 4	T 5	T 6	T 7	T 8	T 9	T 10	T 11	T 12	T 13	T 14	T 15	T 16	K 17	K 18	K 19	K 20	K 21	K 22	K 23	K 24	K 25	K 26	GSOP
TO-i	14				11																						
TO-j				19			20			12	10	9			20					7					15	
TO-k				20		9					5	12	14		17											9	
TO-l	12			20		17				14	11														17	8	
TO-m				17		16					10									10						9	
TO-n						15				19		7								12						4	

(C) *E. nitens* half-diallel

Female parents	Male parents								
	TO-k	TO-l	TO-m	TO-n	TO-o	TO-p	TO-q	TO-r	NSOP
TO-i	20	13	11	20	20	17	17	20	20
TO-j	20	18	19	20	20	18	20		20
TO-k		20		20	19		17	19	20
TO-l			20		20	18			19
TO-m				20	20		14	17	20
TO-n		20			20	20	20	17	20
TO-o						14	20		20
TO-p								20	20

Note: Parents from the Taranna (T), King Island (K), and South Flinders Island (SF) provenances of *E. globulus* and from the Toorongo provenance of *E. nitens* (TO) were included. GSOP is *E. globulus* open-pollinated progeny from a seedling seed orchard, GOP is *E. globulus* open-pollinated progeny from parents in natural stands, and NSOP is open-pollinated progeny from an *E. nitens* seedling seed orchard (see Volker et al. 1994).

Keane 1982*b*; Carnegie et al. 1994). Disease of both the juvenile and adult foliage of *E. nitens* in Australia is believed to be caused by *M. cryptica* (Carnegie et al. 1994).

In eucalypts, studies of genetic variation in resistance to fungal pathogens have concentrated on differences between provenances (Marks and Idczak 1977; Dianese 1984). Provenance variation in resistance to *Mycosphaerella* leaf disease has been reported in *E. globulus* (Carnegie et al. 1994), *E. nitens* (Purnell and Lundquist 1986; Lundquist and Purnell 1987), *Eucalyptus regnans* (Wilcox 1982; Dick and Gadgil 1983), and *Eucalyptus delegatensis* (Dick and Gadgil 1983). Significant differences between families within a provenance have also been reported in *E. globulus* (Reinoso 1992) and *E. regnans* (Wilcox 1982), but there have been few detailed studies of the genetic control of resistance to *Mycosphaerella* leaf disease. Reinoso (1992) reported individual narrow-sense heritabilities (h^2) of 0.31 and 0.32 for *Mycosphaerella* leaf disease on open-pollinated progeny from a single provenance of *E. globulus* grown at two sites. However, because of unpredictable, and possibly differential, inbreeding in open-pollinated progeny, the accuracy of such estimates has been questioned (Hardner and Potts 1995; Potts et al. 1995; Hodge et al.1996). Accurate estimates of genetic parameters often require fully pedigreed controlled crosses (Potts et al. 1995; Hodge et al.

Table 2. The number of families and individuals of each cross type in the trial at West Ridgley.

Cross type	No. of families	No. of individuals
E. globulus pooled	168	3486
E. globulus (King Island)	28	579
E. globulus (Taranna)	55	1096
E. globulus OP	25	516
E. globulus seed orchard OP	8	155
E. nitens (Toorongo)	36	714
E. nitens seed orchard OP	9	194
E. nitens × *E. globulus* F_1 hybrid	43	665

Note: OP, open pollinated. Provenances are in parentheses following species names. *Eucalyptus globulus* pooled contains all the *E. globulus* inter- and intra-provenance crosses in the trial.

1996). They also provide an indication of the importance of nonadditive genetic variation in the genetic control of a trait.

The aim of this study was to examine the inheritance of severity of *Mycosphaerella* leaf disease occurring in intra- and inter-specific crosses of *E. globulus* and *E. nitens*. In addition, we compare the accuracy of genetic parameters and breeding values derived from open-pollinated and fully pedigreed progenies.

Materials and methods

Field trial

Disease was assessed in a field trial containing progeny from an incomplete 8 × 26 *E. globulus* factorial, an incomplete 10 × 10 *E. nitens* half-diallel, an incomplete 6 × 14 *E. nitens* × *E. globulus* F_1 hybrid factorial, and open-pollinated progeny (see Table 1 and Volker et al. 1994). The *E. globulus* parents were from three provenances: Taranna (T), King Island (K), and South Flinders Island (SF) (provenances are detailed in Volker and Orme 1988; Jordan et al. 1993; Potts and Jordan 1994*b*). The factorial included both intra- (T and K) and inter-provenance (T × K, K × T, SF × T, or SF × K) crosses (Table 1). All *E. globulus* males were growing in native stands at Taranna or King Island. All *E. globulus* females were from open-pollinated progeny growing in a seedling seed orchard in northwest Tasmania and originated from native stands at either Taranna, south Flinders Island, or King Island (Volker et al. 1990, 1994). Because of the lack of replication for the provenance, progeny of the single female from the SF provenance were excluded from all analyses (see Table 1). All the *E. nitens* parents were from the Toorongo provenance (Pederick 1979) and were growing in seedling seed orchards or in plantations in northwest Tasmania. Open-pollinated (OP) progeny of all the males (native stand OP) and females (seed orchard OP) were also included. The *E. nitens* × *E. globulus* F_1 hybrids included *E. globulus* males from both King Island and Taranna provenances. All males used in the *E. nitens* × *E. globulus* hybrid factorial were also used in the *E. globulus* factorial, and all females were used in the *E. nitens* half-diallel. In addition to the control-cross and open-pollinated material, some unpedigreed *E. globulus* ssp. *bicostata* (Maiden, Blakely & J. Simm.) Kirkpatr. (hereafter called *E. bicostata*), and *E. bicostata* × *E. globulus* F_1 hybrids were included in the trial (four and six families, respectively), but there were insufficient families to estimate genetic parameters for these cross types.

The number of families and individuals in each cross type are given in Table 2, and trial details are given in Table 3 and Volker et al. (1994). The trial was established by CSIRO Division of Forestry and North Forest Products, at West Ridgley in northwest Tasmania. The trial contained approximately 6000 trees and was based on an alpha lattice design (Patterson and Williams 1976). Each of the four replicates of 1500 trees comprised 15 incomplete blocks, with 20 line plots of 5 trees per incomplete block (see also Hodge et al. 1996).

Disease assessment

Disease severity on leaves still retained on the trees was assessed separately for both the juvenile and adult portions of the crown, using percent damage diagrams adapted from Carnegie et al. (1994) and Lundquist and Purnell (1987) to account for the lower disease severity in the trial. The severity classes used were 0, 1, 2.5, 5, 7.5, 10, 15, 20, 25, 30, 40, 50, and 60% leaf area killed, averaged over the canopy. Microscopic examination of lesions indicated that both *M. cryptica* and *M. molleriana* were equally important on juvenile leaves of *E. globulus*, damage to *E. nitens* was caused exclusively by *M. cryptica*, and the F_1 hybrids were intermediate, but with *M. cryptica* predominating (A.J. Carnegie, unpublished data). However, field assessment of the two pathogens separately is not feasible and, following previous studies (e.g., Carnegie et al. 1994), only total disease severity was assessed. Trees were assessed in mid-March 1993, when they were approximately 3 years old. There was no significant disease damage in the previous year and that present at the date of assessment had become apparent in the 1992–1993 summer season. *Mycosphaerella* spp. cause both leaf necrosis and later, defoliation. It appears that these responses are independent and are measures of different components of susceptibility (C. Reinoso and P. Ades, submitted). At the time of assessment, minimal defoliation due to *Mycosphaerella* disease had occurred and our results only refer to the first component of susceptibility, termed disease severity by Carnegie et al. (1994).

The relationship between percent disease severity and tree size immediately prior to the onset of infection (height, DBH, and volume 1992) and subsequent growth (increments from 1992–1993) were examined. The correlation between percent disease severity and size of the juvenile canopy was also investigated. Measurements were taken in August 1992 (2 years) and September 1993 (3 years), for diameter at breast height (DBH) and height to phase change (the transition between juvenile and adult foliage, Potts and Jordan 1994*a*). Tree height was measured only in the second and third years (August 1992 and September 1993). Individual conic volume was calculated following Potts and Jordan (1994*a*).

Data analysis

All runts and plants with highly abnormal phenotypes were excluded from the analyses (8.2% of the total number of individuals). All data were then log transformed to improve the distribution of the residuals. The *E. globulus* factorial, *E. nitens* half-diallel, and *E. nitens* × *E. globulus* F_1 factorial were analysed separately. Genetic parameters for disease severity and genetic correlations with growth characters were calculated following Volker et al. (1994) using REML VCE (Groeneveld 1995). The individual tree model used was

$$[1] \quad y = X_1c + X_2r + Z_1a + Z_2s + Z_3b + Z_4p + e$$

where y is an $n \times 1$ vector of individual *Mycosphaerella* spp. damage observations, c is a vector of fixed cross-type effects, r is a vector of fixed replicate effects, a is a vector of additive genetic effects (i.e., breeding values of individuals and parents), s is a $q \times 1$ vector of random genetic effects common to each full-sib family (i.e., specific combining ability), b is a $b \times 1$ vector of random effects common to each incomplete block (within each replicate), p is a $p \times 1$ vector of random effects common to each plot (i.e., plot effect), and e is an $n \times 1$ vector of residuals, expected to include mainly the remaining three-quarters of the dominance variance and environmental effects. X_1, X_2, Z_1, Z_2, Z_3, and Z_4 are known incidence matrices relating observations in y to effects in c, r, a, s, b, and p, respectively. Where specific cross types were examined (e.g., *E. nitens*, or within *E. globulus* Taranna or King Island provenances only), the cross effect was omitted from the

Table 3. Establishment, climatic, and measurement details for the *E. nitens* × *E. globulus* hybrid trial near West Ridgley, northwest Tasmania.

Details	West Ridgley field trial
Latitude (S)	41°09′
Longitude (E)	145°46′
Altitude	185 m
Establishment date	July 1990
Avg. annual rainfall	1200 mm
Avg. max. temp.	15.3°C
Avg. min. temp.	7.3°C
Warmest month	Feb. (avg. daily max. 22°C, min. 13°C)
Coldest month	July (avg. daily max. 10.5°C, min. 4°C)
Geology	Tertiary basalt
Soil	Kraznozem derived from tertiary basalt. Deep, well structured, fertile and well drained.
No. of replicates	4
No. of incomplete blocks/replicate	15
No. of plots/incomplete block	20
No. of trees/plot	5
Spacing	3 × 4 m

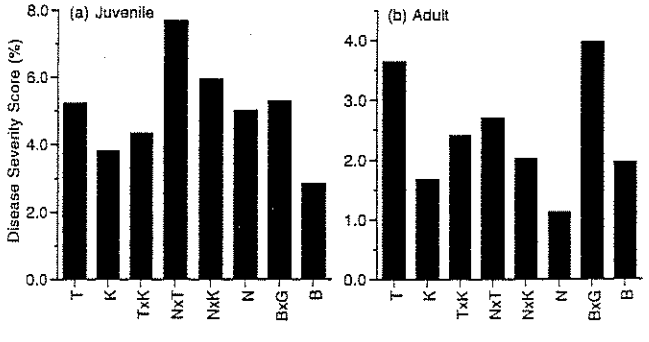


Fig. 1. Least squares means estimates of percent *Mycosphaerella* spp. damage on juvenile (*a*) and adult (*b*) foliage. All cross type means were estimated using the MIXED procedure in SAS Institute Inc. (1992). Cross types included the Taranna (T) and King Island (K) provenances of *E. globulus*, interprovenance crosses within *E. globulus* T × K (incorporating both T × K and K × T crosses), *E nitens* (N), interspecific crosses between *E. nitens* and *E. globulus* (N × T and N × K), *E. bicostata* (B), and interspecific crosses between *E. bicostata* and *E. globulus* (B × G). Specific contrasts and their significance are given in Table 4.

model. Open-pollinated progeny were analysed using model [1], excluding the full-sib family term ($Z_2 s$).

Estimates of additive (σ_a^2), SCA (σ_s^2), incomplete block (σ_b^2), plot (σ_p^2), and error (σ_e^2) variance components were used to calculate individual narrow-sense heritabilities (h^2) and the proportion of dominance variance (d^2) as follows:

$$[2] \quad h^2 = \frac{\sigma_a^2}{\sigma_a^2 + \sigma_s^2 + \sigma_p^2 + \sigma_e^2}$$

$$[3] \quad d^2 = \frac{\sigma_d^2}{\sigma_a^2 + \sigma_s^2 + \sigma_p^2 + \sigma_e^2}$$

where $\sigma_d^2 = 4\sigma_s^2$ (Falconer 1986). Estimates of heritability for the open-pollinated populations were calculated as in [2] but excluding σ_s^2. Such estimates assume that the open-pollinated families are half-sibs ($r = 0.25$). However, this estimate was adjusted (h_{op}^2) to account for selfing within open-pollinated families of *Eucalyptus* by multiplying the h^2 estimates by 0.625. This adjustment follows Griffin and Cotterill (1988) and Volker et al. (1990) and assumes an average outcrossing rate of 70% and a genetic correlation (r) among open-pollinated sibs of 0.4. Standard errors were estimated following Becker (1985).

Cross type least squares means were determined using the MIXED procedure in SAS Institute Inc. (1992) using model [1]. Contrasts were undertaken to test differences between cross types and whether hybrids were significantly different from the expected midparent value using PEST (Groeneveld et al. 1992), which performs an F test based on estimates of the error variance (Kennedy 1989).

Best linear unbiased predictions (BLUPs) of parental breeding values (BVs) were calculated with PEST, using variance component estimates from REML VCE. BVs were calculated separately for the *E. globulus*, *E. nitens*, and F_1 hybrid controlled cross populations and the *E. globulus* and *E. nitens* open-pollinated populations. This allowed direct comparison of parental BVs estimated in pure species and hybrid combination as well as under open pollination. The model used in these analyses was the same as [1], but the cross-type effect was excluded for the *E. globulus* parents to enable parental breeding values to be estimated across provenances. Parental breeding values were similarly estimated for the *E. globulus* males using the open-pollinated and hybrid populations, and these estimates were compared with those determined from controlled intraspecific crossing.

Results

Cross-type effects

There was significant provenance variation within *E. globulus*, with Taranna (T) having a significantly higher average disease severity than the King Island (K) provenance on both juvenile ($P < 0.01$) and adult foliage ($P < 0.001$) (see Table 4 and Fig. 1). The interprovenance crosses between Taranna and King Island parents (T × K and K × T) exhibited intermediate severity and were not significantly different from the mid-provenance value in either juvenile or adult foliage (Table 4). Disease severity on juvenile foliage of the Toorongo provenance of *E. nitens* (N) was not significantly different from that

Table 4. Contrasts between cross types for damage to *Mycosphaerella* spp. on juvenile and adult foliage of all the cross types within the hybrid trials.

	Juvenile		Adult	
Contrast (cross 1 vs. cross 2)	*P* value	Difference (means)	*P* value	Difference (means)
Pooled glob vs. N	0.2524	–0.557	0.0000	1.448
Pooled glob vs. B	0.0004	1.614	0.1671	0.604
Pooled glob vs. B×G	0.0904	–0.828	0.0000	–1.406
Pooled glob vs. F_1 (N×T and TxN)	0.0443	–2.367	0.3503	0.215
T vs. K	0.0062	1.415	0.0000	1.963
T vs. (T×K and K×T)	0.0048	0.889	0.0000	1.237
K vs. (T×K and K×T)	0.0881	–0.526	0.0001	–0.726
Mid T & K vs. pooled T×K & K×T	0.4016	0.889	0.1258	0.256
N vs. T	0.5562	–0.211	0.0000	–2.515
N vs. K	0.0341	1.204	0.0190	–0.552
N×T vs. N	0.0000	2.684	0.0000	1.566
N×K vs. N	0.0856	0.936	0.0000	0.900
N×T vs. T	0.0007	2.473	0.0018	–0.949
N×K vs. K	0.0004	2.139	0.2302	0.348
N vs. (N×T and N×K)	0.0003	–1.810	0.0000	–1.233
Mid N & K vs. N×K	0.0014	–1.537	0.0016	–0.624
Mid N & T vs. N×T	0.0000	–2.578	0.0060	–0.308
B vs. B×G	0.0000	–2.442	0.0000	–2.010
Mid B & pooled glob vs. B×G	0.0005	–1.635	0.0000	–1.708

on *E. globulus* from Taranna, but was significantly ($P < 0.05$) higher than on the King Island provenance. On adult foliage, disease severity on *E. nitens* was significantly lower than for any other cross type ($P < 0.05$). Disease severity was significantly lower on the juvenile foliage of *E. bicostata* when compared with *E. globulus* ($P < 0.001$). However, on adult foliage severity was not significantly different between *E. bicostata* and *E. globulus* ($P = 0.167$).

In general, the disease severity on juvenile foliage of the *E. nitens* × *E. globulus* F_1 hybrids was significantly ($P < 0.05$) greater than for either *E. nitens* or *E. globulus* (see Fig. 1, Table 4), regardless of whether the provenances of *E. globulus* were pooled (except N × K vs. N (not significant)). The disease severity on the adult foliage of the F_1 hybrids was significantly greater ($P < 0.001$) than on *E. nitens*. However, the N × K hybrids had similar severity to the King Island provenance, while the N × T hybrids were intermediate, with significantly less disease than the Taranna provenance. The *E. bicostata* × *E. globulus* hybrids (B × G) had the higher disease severity on adult foliage than any other cross type in the trial, including both parent species ($P < 0.001$). On juvenile foliage, the *E. globulus* × *E. bicostata* hybrids had significantly higher severities than *E. bicostata*, but were not significantly different from the pooled *E. globulus* provenances (King Island and Taranna). In contrast with the *E. globulus* interprovenance crosses, the interspecific crosses of *E. globulus* with *E. nitens* or *E. bicostata* exhibited disease severities significantly ($P < 0.01$) greater than the predicted midspecies values for both adult and juvenile foliage types (Table 4). In all cross types, disease severity was greater on juvenile than adult foliage (Fig. 1).

Genetic parameters

Controlled-cross estimates of individual narrow-sense heritability (h^2) of the percentage of leaf area damaged ranged from 0.115 to 0.343 for *E. globulus* and 0.004 to 0.208 for *E. nitens* (Table 5). Heritability estimates for adult foliage were always greater than those for juvenile foliage, despite the disease severity being greater on juvenile foliage (see Table 5, Fig. 1). There was a tendency for estimates of h^2 in both adult and juvenile foliage to be lower for *E. nitens* than for *E. globulus*. Furthermore, when the provenances of *E. globulus* were separated, the h^2 for the King Island population (juvenile 0.115, adult 0.343) were comparable with those obtained for the Taranna population (juvenile 0.120, adult 0.250). The proportion of dominance variation (d^2) was low (Table 5), particularly for the *E. nitens* population, and in most cases was less than half of the h^2 estimate. The h^2 estimate for damage on juvenile foliage in the King Island population was the lowest of the *E. globulus* estimates, and this was the only case where the d^2 estimate exceeded h^2. These results suggest that in most cases, there is little nonadditive genetic variation for susceptibility in these eucalypt populations and most of the genetic variation is due to additive effects. Estimates of d^2 for the F_1 hybrid population were comparable with those obtained for the pure species. In contrast, the h^2 estimate for juvenile foliage was nearly double that found in any of the pure species populations. Estimates of h^2 for disease severity on the adult foliage of the F_1 hybrids were consistent with the pure species estimates. Estimates of h^2 based on the open-pollinated progeny (h^2_{op}) collected from the *E. globulus* males were comparable to those obtained from controlled crossing (Table 5). In both *E. globulus* and *E. nitens* the h^2_{op} estimates for juvenile foliage were larger than the h^2 estimates, whereas those obtained from adult foliage were smaller. However, the standard errors of the h^2 estimates would suggest that these differences were not significant.

Genetic correlations between disease severity scores and growth traits are given in Table 6. The percent severity in the adult canopy was highly genetically correlated with severity in the juvenile canopy in all populations (0.52 to 0.74, see

Table 5. Components of variance, proportion of dominance variation (d^2), and individual narrow-sense heritability estimates (h^2) and their approximate standard errors (SE) for *Mycosphaerella* spp. resistance in *E. globulus*, *E. nitens*, and their F_1 hybrid.

	Components of variance						
Cross type	Additive	Incomplete block	Plot	Family (SCA)	Residual	d^2	h^2±SE
E. globulus pooled							
Juvenile	0.008	0.008	0.011	0.001	0.047	0.060	0.119±0.024
Adult	0.017	0.006	0.010	0.002	0.046	0.107	0.227±0.032
E. globulus (King Island),							
Juvenile	0.006	0.009	0.003	0.003	0.040	0.231	0.115±0.058
Adult	0.024	0.001	0.008	0.001	0.037	0.057	0.343±0.101
E. globulus (Taranna)							
Juvenile	0.009	0.010	0.012	0.000	0.054	0.000	0.120±0.042
Adult	0.020	0.012	0.011	0.003	0.046	0.150	0.250±0.060
E. globulus open pollinated							
Juvenile	0.019	0.003	0.013	0.001	0.034	ne	0.177±0.072
Adult	0.020	0.004	0.011	0.004	0.030	ne	0.192±0.076
E. nitens							
Juvenile	0.005	0.022	0.004	0.000	0.032	0.000	0.122±0.052
Adult	0.026	0.006	0.023	0.002	0.083	0.059	0.194±0.065
E. nitens open pollinated							
Juvenile	0.018	0.016	0.004	0.001	0.031	ne	0.208±0.058
Adult	0.008	0.011	0.000	0.000	0.117	ne	0.004±0.053
E. globulus × *E. nitens*							
Juvenile	0.044	0.022	0.008	0.001	0.034	0.046	0.506±0.010
Adult	0.011	0.004	0.012	0.002	0.037	0.129	0.177±0.057

Note: ne, not estimable. The phenotypic variance used to calculate d^2 and h^2 did not include the variance component due to incomplete blocks.

Table 6). In the *E. globulus* population, neither the adult nor juvenile severity was genetically correlated with tree size at the presumed time of infection, as genetic correlations between damage and height or DBH in 1992 were low (Table 6). Growth, measured as the increment in DBH, height, or volume over the summer season in which the infection occurred, was consistently positively genetically correlated with severity in the juvenile canopy. This correlation indicated that at the genetic level, increased *Mycosphaerella* damage was associated with an increase in subsequent growth. While contrary to what may be expected, this appears to be due to the delayed phase change in *E. globulus* being genetically associated with both increased growth ($r_g = 0.61$ between HTPC and sectional area at age 4 years; P. Volker, unpublished data) and increased damage to the juvenile canopy ($r_g = 0.54$, Table 6). In contrast, correlations of incomplete block effects between damage to the juvenile foliage and subsequent growth traits, which reflect an environmental correlation, were consistently negative (–0.26 to –0.38) as would be expected if disease was having a direct deleterious effect on growth.

In the *E. nitens* population, there was a general negative correlation between disease on both the adult and juvenile foliage and tree size at the presumed time of infection. The genetic correlations between disease severity on the juvenile foliage and subsequent growth were inconsistent, but negative for adult foliage (Table 6). Within the *E. nitens* × *E. globulus* F_1 hybrid population, severity of disease on both the juvenile and adult foliage were positively correlated with all the growth traits measured. Tree size at the time of infection (DBH and height at 2 years) appears to have had a profound effect on disease severity, with faster growing hybrids having higher scores for both juvenile and adult foliage (Table 6). A larger juvenile canopy (larger HTPC) was strongly genetically correlated with severity on both the juvenile and adult foliage. This contrasts with both *E. globulus* and *E. nitens*, where high severity in the adult canopy was generally associated with a smaller juvenile canopy (HTPC).

Correlations of parental breeding values

Parental breeding values (BVs) for disease severity on either juvenile or adult foliage were similar for *E. globulus* (Pearson's correlation coefficient, $r = 0.69$, see Table 7), consistent with the high genetic correlation given in Table 6. Likewise, parental BVs estimated from controlled-cross progeny and open-pollinated progeny were highly correlated in both *E. globulus* and *E. nitens* (Pearson's correlations of 0.73–0.75 and 0.50–0.73 respectively, Table 7). The distinction between *E. globulus* parents from the King Island provenance (which generally had lower BVs, corresponding to lower disease severity) and parents from Taranna for these correlations can be seen in Fig. 2. The correlations between parental BVs calculated from controlled-crossed progeny were poorly correlated with those calculated from parents in hybrid combination for either species (Table 7). Correlations between parental BVs obtained for *E. nitens* in any combination were consistently lower than those obtained for *E. globulus*, perhaps because of the low number of parents (11) and the single provenance origin

Table 6. Genetic correlations between disease severity on juvenile and adult foliage and growth traits prior to infection and incremental growth after initial infection, for *E. nitens*, *E. globulus*, and *E. nitens* × *E. globulus* hybrids.

	E. globulus pooled		*E. nitens*		*E. nitens* × *E. globulus*	
	Juvenile	Adult	Juvenile	Adult	Juvenile	Adult
Damage on adult	0.570	1.000	0.521	1.000	0.735	1.000
Height at 2 years	–0.122	0.302	–0.342	–0.307	0.539	0.386
DBH at 2 years	0.195	0.045	–0.119	–1.000	0.777	0.610
Growth (height 2–3 years)	0.540	0.048	0.368	–0.816	0.616	0.524
Growth (DBH 2–3 years)	0.702	0.256	–0.135	–0.754	0.703	0.560
Growth (vol. 2–3 years)	0.469	0.105	0.012	–0.870	0.738	0.548
HTPC (3 years)	0.538	–0.311	0.617	–0.595	0.717	0.501

Note: *Eucalyptus globulus* pooled estimates included both the King Island (K) and Taranna (T) provenances as well as the interprovenance crosses (both K × T and T × K). DBH, diameter at breast height; HTPC, the height to vegetative phase change (i.e., the height at which juvenile foliage changed to adult foliage).

Table 7. Pearson's correlation coefficients (±SE) between controlled cross estimates of breeding values and those estimated for the same parents in open pollination and in hybrid combination for *E. nitens* and *E. globulus*.

		Controlled cross	Significance
E. nitens			
Juvenile	Hybrid	0.26±0.48	$0.50<P<0.20$
	Open pollinated	0.73±0.26	$0.02<P<0.01$
Adult	Hybrid	0.31±0.39	$0.50<P<0.20$
	Open pollinated	0.50±0.33	$0.10<P<0.05$
Juvenile vs. adult	Controlled cross	0.40±0.31	$0.20<P<0.10$
E. globulus			
Juvenile	Hybrid	0.24±0.27	$0.50<P<0.20$
	Open pollinated	0.75±0.14	$P<0.001$
Adult	Hybrid	0.40±0.25	$0.20<P<0.10$
	Open pollinated	0.73±0.14	$P<0.001$
Juvenile vs. adult	Controlled cross	0.69±0.15	$P<0.001$

Note: Standard errors and significance levels were calculated according to Zar (1984).

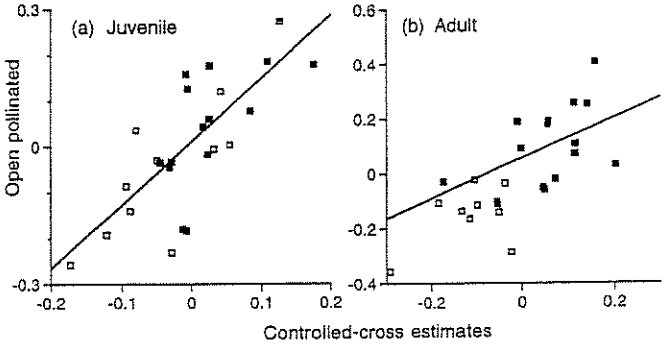
and therefore limited variability of the *E. nitens* parents (Toorongo).

Discussion

There was a significant difference in the severity of damage from *Mycosphaerella* spp. lesions between the two provenances of *E. globulus*, King Island and Taranna. King Island was less damaged, and this is consistent with there having been more intense natural selection for resistance to *Mycosphaerella* disease in that area. Warm, wet weather is conducive to *Mycosphaerella* outbreaks (Park 1988; Carnegie et al. 1994). The wetter maritime climate on King Island (1015 mm annual rainfall) would suggest a general climatic regime more favourable to *Mycosphaerella* spp. than that of Taranna (892 mm annual rainfall, Fig. 3). The lower susceptibility of *E. globulus* provenances from areas with high summer rainfall was noted by Carnegie et al. (1994). *Eucalyptus bicostata* (Mansfield provenance) has previously been found to have very high disease severity when compared with *E. globulus* (Carnegie et al. 1994). The *E. bicostata* in the trial studied here had the lowest disease severity on juvenile foliage but on adult foliage, had similar damage levels to *E. globulus*. The relative resistance of *E. bicostata* in the West Ridgley trial appears to conflict with the severe disease on *E. bicostata* noted by Carnegie et al. (1994). However, the unknown origin of material in this experiment and the low number of families means that the two results can not really be compared. *Eucalyptus bicostata* is widespread and occurs in areas with both winter and summer rainfall regimes. Provenance variation in this subspecies has not been comprehensively tested in Australia, so it is not possible to generalize about its susceptibility relative to *E. globulus*.

Disease severity was higher on interspecific hybrids than on parental populations, both on the juvenile (*E. nitens* × *E. globulus*) and adult (*E. bicostata* × *E. globulus*) foliage. Interspecific hybrids performed at least as badly as the worst performing parent in terms of the levels of damage and were consistently more damaged than the predicted midparent value. In contrast, the disease severity on the interprovenance hybrids of *E. globulus* was intermediate to those on both the Taranna and King Island provenances and was not significantly

Fig. 2. The correlation of breeding values of *E. globulus* parents in open-pollinated and controlled-cross combination for (*a*) juvenile and (*b*) adult foliage. Breeding value estimates included differences between the Taranna (■) and King Island (□) provenances of *E. globulus*. Corresponding Pearson's correlation coefficients are given in Table 7.

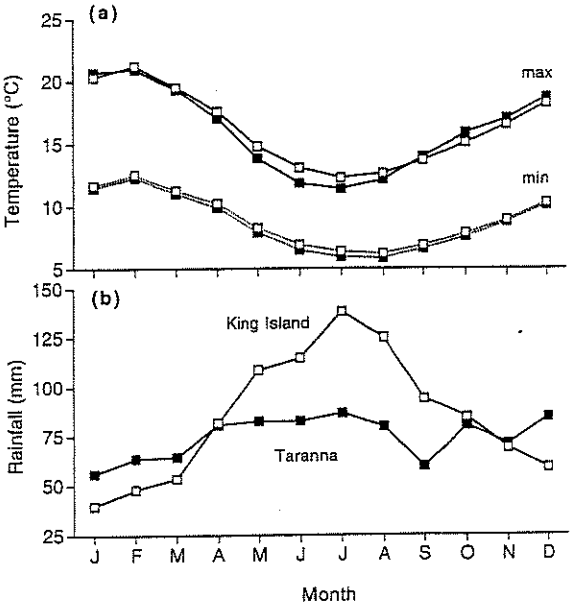


different from the midparent values. The tendency for greater susceptibility of interspecific hybrids is consistent with other observations, suggesting that interspecific hybrids may be more susceptible to pests (Whitham 1989; Fritz et al. 1994; Strauss 1994).

Within populations, low to moderate heritabilities (h^2) were obtained for both *E. globulus* and *E. nitens*. Heritabilities obtained for juvenile foliage were usually lower than those for adult foliage, despite mean severity levels being greater on juvenile foliage. The *E. nitens* × *E. globulus* hybrids had, overall, the most severe disease (juvenile foliage 8%) and the highest heritability (0.40). Our heritability estimates for juvenile foliage were generally lower than the 0.32 reported for open-pollinated progenies by Reinoso (1992). The severity of *Mycosphaerella* leaf disease was greater at their sites, with average damage up to 16.4%, whereas at at the present site average disease severity for *E. globulus* was less than 6%. The expression of genetic variation in disease resistance may also be dependent on the level of infection (White and Hodge 1989), and it is possible that larger heritabilities would have been obtained with greater damage on the trees used in the present study. Our heritability estimates may also be lower because of the lower infection levels resulting in susceptible individuals escaping infection. However, this effect would be random and averaged out at the parent, family, and cross-type level. It is unlikely that the heritability estimates of Reinoso (1992) are inflated as a result of the use of open-pollinated progeny, since the present study has shown such estimates to be comparable with those obtained from controlled crossing. In contrast, open-pollinated versus controlled-cross estimates have been shown to be inflated for growth traits (Hodge et al. 1996).

The high correlations obtained between parental breeding values estimated from open pollination and controlled crossing of *E. globulus* clearly indicate that determining the best or worst parents for *Mycosphaerella* damage from open-pollinated progeny would be an acceptable strategy in this instance. This is an important finding, as breeding values calculated from open-pollinated progeny and controlled-cross progeny for growth traits are not always well correlated (see Hodge et al. 1996), and, as controlled crossing is expensive, this would significantly reduce the cost of any breeding pro-

Fig. 3. Average monthly minimum (·····) and maximum (—) temperatures (°C) and rainfall (mm) for the Taranna (■) and King Island (□) provenances of *E. globulus*. All data were obtained from ESOCLIM (H.A. Nix, J.P. Busley, M.F. Hutchinson, and J.P. McMahon; Hutchinson 1991).



gram. Comparable correlations calculated for the *E. nitens* parents were also high, but were not significant for adult foliage. This is most likely due to the relatively low number of parents involved in the half-diallel mating design used and the fact that all parents were from the same provenance (Toorongo). In contrast, the correlation between parental breeding values estimated in hybrid and pure species combinations was always low and not significant. Hence, selecting the best parents from pure species populations for hybrid combination would not necessarily produce the best performing hybrid progeny.

Heritability estimates and breeding values derived from this trial refer to disease severity caused by a complex of two species, *M. cryptica* and *M. molleriana*, although only *M. cryptica* was recorded from *E. nitens* (A.J. Carnegie, unpublished data). These two species generally co-occur on *E. globulus* in southeastern Australia (Carnegie et al. 1994), and thus *E. globulus* selections undertaken for breeding would integrate responses to both pathogens. Parents with breeding values for low disease severity are likely to be resistant to both pathogens, whereas parents with high disease severity may be susceptible to one or other, or both pathogens. How the genetic parameters for disease severity will change with differing contribution of the two pathogens to the disease outbreak and damage is complex and unclear at present. Isolation of the pathogens from the experiment indicates that damage to adult foliage of *E. globulus* was entirely due to *M. cryptica*, whereas both pathogens contributed to damage on the juvenile foliage (A.J. Carnegie, unpublished data). The presence of both pathogens may mask a close genetic association between host and pathogen and could explain the generally lower heritabilities and higher levels of damage recorded on juvenile compared with adult foliage of *E. globulus*. Contribution of each species

of pathogen to damage of juvenile leaves of each cross type is currently being quantified for a subset of these crosses. Nevertheless, positive genetic correlations were observed between disease severity on adult and juvenile foliage in the present study, strong genetic correlations across different sites were observed for disease severity on juvenile foliage by Reinoso (1992), and significant correlations across seasons have been reported at the provenance level by Carnegie et al. (1994). These results suggest a correlated response to selection across disease outbreaks; however, detailed studies with artificial inoculations under greenhouse conditions are required to partition the response to either pathogen alone and together.

Previous reports have suggested that a reduction in growth rate of *E. nitens* will not occur unless defoliation of the juvenile crown by *Mycosphaerella* exceeded 25% (Lundquist and Purnell 1987). While the percent damage scores used here differ (see Lundquist and Purnell 1987; Reinoso (1992), our results support this idea. In our trial, growth may well have been reduced by *Mycosphaerella* leaf disease, but genetic correlations between growth and disease severity of the juvenile foliage were predominantly positive, that is, the greater the growth rate the greater the disease severity score. Previous work has suggested that in *E. nitens* at least, trees with juvenile-persistent foliage are the fastest growing (Beadle et al. 1989). Trees with juvenile-persistent foliage also appear to be more susceptible to *Mycosphaerella* disease, possibly as a result of the greater opportunity for disease increase within the larger crown. Genetic correlations with the height to phase change provided support for this interpretation, as delayed transition to adult foliage (i.e., a larger juvenile canopy) was consistently positively correlated with disease severity. However, in the present case, disease does not appear to have been sufficiently severe to counteract the positive effect of the larger juvenile canopy on growth, possibly because of the low levels of severity observed. Indeed, the level of disease severity observed in the present outbreak is low compared with that reported for other sites (Carnegie et al. 1994; C. Reinoso and P. Ades, submitted). For example, Carnegie et al. (1994) report mean disease severity of ca. 30% on juvenile foliage of the King Island provenance of *E. globulus* compared with less than 4% in the present study. It seems that trees with a larger canopy or leaf area index have a greater likelihood of experiencing severe *Mycosphaerella* disease than trees with a small canopy. Reinoso (1992) suggested that one means of increasing resistance to *Mycosphaerella* damage would be to select for early transition to adult foliage. This would reduce the period in which the plantation is highly susceptible to the disease and result in increased growth rate on sites favourable to epidemics. However, this may have an overall negative impact on growth by reducing the more productive juvenile canopy (Beadle et al. 1989). The fact that most tree breeders select for growth in the absence of disease (e.g., Borralho et al. 1992; Jarvis et al. 1995) means that breeders may be indirectly selecting for more susceptible trees, which may have important economic implications when the stock is planted where severe disease is likely.

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References

Beadle, C.L., McLeod, D.E., Turnbull, C.R.A., Ratkowsky, D.A., and McLeod, R. 1989. Juvenile/total foliage ratios in *Eucalyptus nitens* and the growth of stands and individual trees. Trees, **3**: 117–124.

Becker, W.A. 1985. Manual of quantitative genetics. Academic Enterprises, Pullman, Wash.

Borralho, N.M.G., Kanowski, P.J., and Cotterill, P.P. 1992. Genetic control of growth of *Eucalyptus globulus* in Portugal. I. Genetic and phenotypic parameters. Silvae Genet. **41**: 39–45.

Carnegie, A.J., Keane, P.J., Ades, P.K., and Smith, I.W. 1994. Variation in susceptibility of *Eucalyptus globulus* provenances to *Mycosphaerella* leaf disease. Can. J. For. Res. **24**: 1751–1757.

Crous, P.W., Wingfield, M.J., and Park, R.F. 1991. *Mycosphaerella nubilosa,* a synonym of *M. molleriana*. Rev. Plant Pathol. **95**: 628–632.

Davidson, J. 1989. Ethiopia: *Eucalyptus* tree improvement and breeding. Field Document 1. UNDP/FAO project ETH/88/010, Ethiopia. Food and Agriculture Organization, Rome.

Dianese, J.C. 1984. Response of *Eucalyptus* species to field infection by *Puccinia psidii*. Plant Dis. **68**: 314–316.

Dick, M., and Gadgil, P.D. 1983. *Eucalyptus* leaf spots. New Zealand Forest Service, Forest Research Institute, Rotorua. For. Pathol. N.Z. 1.

Eldridge, K., Davidson, J., Harwood, C., and Van Wyk, G. 1993. Eucalypt domestication and breeding. Clarendon Press, Oxford, England.

Falconer, D.S. 1986. Introduction to quantitative genetics. Longman, London.

Fritz, R.S., Nichols-Orians, C.M., and Brunsfeld, S.J. 1994. Interspecific hybridization of plants and resistance to herbivores: hypotheses, genetics and variable responses in a diverse herbivore community. Oecologia, **97**: 106–117.

Griffin, A.R., and Cotterill, P.P. 1988. Genetic variation in growth of outcrossed, selfed and open-pollinated progenies of *Eucalyptus regnans* and some implications for breeding strategy. Silvae Genet. **37**: 124–131.

Groeneveld, E. 1995. REML VCE—a multivariate multimodel restricted maximum likelihood (co)variance component estimation package. Version 3.1 user's guide. Institute of Animal Husbandry and Animal Ethology, Federal Research Center of Agriculture, Mariensee, D-31535, Neustadt, Germany.

Groeneveld, E., Kovac, M., Wang, T., and Fernando, R.L. 1992. Computing algorithms in a general purpose BLUP package for multivariate prediction and estimation. Arch. Tierz. **35**: 399–412.

Hardner, C.M., and Potts, B.M. 1995. Inbreeding depression and changes in variation after selfing in *Eucalyptus globulus*. ssp. *globulus*. Silvae Genet. **44**: 46–54.

Hodge, G.R., Volker, P.W., Potts, B.M., and Owen, J.V. 1996. A comparison of genetic information from open-pollinated and control-pollinated progeny tests in two eucalypt species. Theor. Appl. Genet. **92**: 53–63

Hutchinson, M.F. 1991. The application of thin-plate smoothing splines to continent-wide data assimilation. Bureau of Meteorology, Melbourne. BMRC Res. Rep. 27. pp. 104–113.

Jarvis, S.F., Borralho, N.M.G., and Potts, B.M. 1995. Implementation

of a multivariate BLUP model for genetic evaluation of *Eucalyptus globulus*. *In* Eucalypt Plantations: Improving Fibre Yield And Quality. Proceedings, CRC–IUFRO Conference, 19–24 Feb. 1995, Hobart, Australia. *Edited by* B.M. Potts, N.M.G. Borralho, J.B. Reid, R.N. Cromer, W.N. Tibbits, and C.A. Raymond. CRC for Temperate Hardwood Forestry, Hobart, Australia. pp. 212–216.

Jordan, G.J., Potts, B.M., Kirkpatrick, J.B., and Gardiner, C. 1993. Variation in the *Eucalyptus globulus* complex revisited. Aust. J. Bot. **41**: 763–785.

Kennedy, B.W. 1989. Introduction to linear models in animal breeding. Centre for Genetic Improvement of Livestock, University of Guelph, Vestby, Norway.

Kirkpatrick, J.B. 1975. Geographical variation in *Eucalyptus globulus*. Forest and Timber Bureau. Bull. 47. Australian Government Publishing Service, Canberra.

Lundquist, J.E. 1985. Reduced growth rates of *Eucalyptus nitens* caused by *Mycosphaerella molleriana*. Phytophylactica, **17**: 55 [Abstr.].

Lundquist, J.E., and Purnell, R.C. 1987. Effects of *Mycosphaerella* leaf spot of growth of *Eucalyptus nitens*. Plant Dis. **71**: 1025–1029.

Marks, G.C., and Idczak, R.M. 1977. *Phytophthora cinnamomi* root rot investigations in Victoria: a review with special reference to forestry. For. Comm. Tech. Pap. (U.K.), **26**: 19–36.

Neish, P.G., Drinnan, A.N., and Ladiges, P.Y. 1995. Anatomy of leaf-margin lenticels in *Eucalyptus denticulata* and three other eucalypts. Aust. J. Bot. **43**: 211–221.

Park, R.F. 1988. Epidemiology of *Mycosphaerella nubilosa* and *M. cryptica* on *Eucalyptus* spp. in south-eastern Australia. Trans. Br. Mycol. Soc. **91**: 261–266.

Park, R.F., and Keane, P.J. 1982*a*. Leaf diseases of *Eucalyptus* associated with *Mycosphaerella* species. Trans. Br. Mycol. Soc. **79**: 101–115.

Park, R.F., and Keane, P.J. 1982*b*. Three *Mycosphaerella* species from leaf diseases of *Eucalyptus*. Trans. Br. Mycol. Soc. **79**: 95–100.

Patterson, H.D., and Williams, E.R. 1976. A new class of resolvable incomplete block designs. Biometrika, **63**: 83–92.

Pederick, L.A. 1979. Natural variation in shining gum (*Eucalyptus nitens*). Aust. For. Res. **9**: 41–63.

Potts, B.M., and Jordan, G.J. 1994*a*. Genetic variation in the juvenile leaf morphology of *Eucalyptus globulus* Labill. ssp. *globulus*. For. Genet. **1**: 81–95.

Potts, B.M., and Jordan, G.J. 1994*b*. The spatial pattern and scale of variation in *Eucalyptus globulus* Labill. ssp. *globulus*: variation in seedling abnormalities and early growth. Aust. J. Bot. **44**: 471–492.

Potts, B.M., Volker, P.W., Hodge, G.R., Borralho, N.M.G., Hardner, C.M., and Owen, J.V. 1995. Genetic limitations in the exploitation of base populations of *Eucalyptus globulus* spp. *globulus*. *In* Eucalypt Plantations: Improving Fibre Yield and Quality. Proceedings, CRC–IUFRO Conference, 19–24 Feb., Hobart, Australia. *Edited by* B.M. Potts, N.M.G. Borralho, J.B. Reid, R.N. Cromer, W.N. Tibbits, and C.A. Raymond. CRC for Temperate Hardwood Forestry, Hobart, Australia. pp. 217–221.

Purnell, R.C., and Lundquist, J.E. 1986. Provenance variation of *Eucalyptus nitens* on the eastern Transvaal Highveld in South Africa. S. Afr. For. J. **138**: 23–31.

Reinoso, C. 1992. Variation in *Eucalyptus globulus* in susceptibility to *Mycosphaerella* leaf diseases. Master of Forest Science thesis, University of Melbourne.

SAS Institute Inc. 1992. SAS technical report P-229, SAS/STAT software: changes and enhancements, Release 6.09. SAS Institute Inc., Cary, N.C.

Strauss, S.Y. 1994. Levels of herbivory and parasitism in host hybrid zones. Trees, **9**: 209–213.

Tibbits, W.N. 1986. Eucalypt plantations in Tasmania. Aust. For. **49**: 219–225.

Tibbits, W.N., and Reid, J.B. 1987*a*. Frost resistance in *Eucalyptus nitens* (Deane & Maiden) Maiden: Physiological aspects of hardiness. Aust. J. Bot. **35**: 235–250.

Tibbits, W.N., and Reid, J.B. 1987*b*. Frost resistance in *Eucalyptus nitens* (Deane & Maiden) Maiden: genetic and seasonal aspects of variation. Aust. For. Res. **17**: 29–47.

Volker, P.W., and Orme, P.K. 1988. Provenance trials of *Eucalyptus globulus* and related species in Tasmania. Aust. For. **51**: 257–265.

Volker, P.W., Dean, C.A., Tibbits, W.N., and Ravenwood, I.C. 1990. Genetic parameters and gains expected from selection in *Eucalyptus globulus* in Tasmania. Silvae Genet. **39**: 18–21.

Volker, P.W., Owen, J.V., and Borralho, N.M.G. 1994. Genetic variances and covariances for frost tolerance in *Eucalyptus globulus* and *E. nitens*. Silvae Genet. **43**: 366–372.

White, T.L., and Hodge, G.R. 1989. Predicting breeding values with applications in forest tree improvement. For. Sci. **33**: 173–207.

Whitham, T.G. 1989. Plant hybrid zones as sinks for pests. Science (Washington, D.C.), **244**: 1490–1493.

Wilcox, M.D. 1982. Preliminary selection of suitable provenances of *Eucalyptus regnans* for New Zealand. N.Z. J. For. Sci. **12**: 468–479.

Zar, J.H. 1984. Biostatistical analysis. Prentice-Hall, Inc., Englewood Cliffs, N.J.