Predicted genetic gain and realised gain in stand volume of *Eucalyptus globulus*

A.N. Callister^{123*}, N. England⁴ and S. Collins⁴

¹Treehouse Consulting, 2 Campbell Rd, Denmark WA 6333, Australia

²Honorary Research Fellow, Department of Forest and Ecosystem Science,
University of Melbourne. Water Street, Creswick VIC 3363, Australia

³Adjunct Research Fellow, Centre of Excellence in Natural Resource Management, The
University of Western Australia, PO Box 5771, Albany, WA 6332, Australia

⁴Elders Forestry Limited, PO Box 1421, Albany WA 6331, Australia

*Corresponding author: andrew@treehouseconsulting.com.au; phone +61 427 482 947

Abstract

Realised gains in stand volume of *Eucalyptus globulus* Labill. families at 7-9 years were compared with gains predicted at 3.5 years. Gain predictions were based on height, diameter at breast height (DBH), sectional area, and stem volume for 153 full-sib families and 18 commercial checks in five-tree line plots on three West Australian sites. Single-site narrow-sense heritability estimates were 0.12-0.24 for height, 0.08-0.12 for DBH, 0.09-0.13 for sectional area and 0.14-0.19 for stem volume. Genetic dominance effects were significant ($p < 0.05$) in most cases and the estimated dominance:additive genetic variance ratio was heterogeneous for height and DBH.

Stand volume was measured for 93 of the same families and checks in 40-tree block plots on four sites. Heritability of stand volume was 0.25-0.76, with an across-sites estimate of 0.41. Dominance effects were statistically absent at two sites. Estimated region-wide additive genetic correlations between selection traits (in line plots) and stand volume (in block plots) ranged from 0.86 to 0.90. Estimated stand volume gain was 23% of the mean for the best 12% of families and 14% of the mean for the best 24% of families. Realised gain was under-estimated by predictions based on height, DBH, and sectional area, which had smaller coefficients of additive variation than did stem volume. It is concluded that although BLUP analysis of early-age height and DBH can provide for

1 indirect selection on *E. globulus* stand volume, analysis of stem volume is required to
2 predict genetic gain at an appropriate scale.

3

4 **Keywords (4-6):** *Eucalytus globulus*; realised genetic gain; stand volume; predicted
5 genetic gain; genetic correlation, best linear unbiased prediction

6

1 **Introduction**

2 A common goal of forest tree improvement is to increase harvest stand volume, usually
3 by indirect selection on stem size early in the rotation. Trials with small plots such as
4 lines or single trees are recommended for this purpose because they allow evaluation of
5 many genetic entries while providing good replication and control of environmental
6 variation (Magnussen 1993). The optimum selection age for pulpwood eucalypts has
7 been estimated at between 3 and 4 years (Greaves et al. 2003). However, such early data
8 from small-plot trials might not provide for an accurate prediction of genetic gains in
9 harvest stand volume. The scale of genetic effects may change through time and with
10 trial design (Foster 1989; Carson et al. 1999) and competition between entries in small
11 plots can either inflate heritability and/or gain estimates (Foster 1989; Vergara et al.
12 2004; Stanger et al. 2011) or cause size-related mortality leading to artificially depressed
13 heritability (Stackpole et al. 2010). Realised gains in stand volume must therefore be
14 determined using trials with large plots ('block plots') which represent the growing
15 environment of a commercial stand (Foster 1992; Foster and Knowe 1995).

16
17 Predicted genetic gains in harvest stand volume are commonly used for projecting yields
18 and wood supply, implementing index selection, and estimating the financial returns from
19 breeding. For softwood species with rotations of 30 years or more, early-rotation genetic
20 gains have been translated into harvest volume increments by incorporating genetic
21 values into empirical growth models (e.g. Hamilton and Rehfeldt 1994; Carson et al.
22 1999; Adams et al. 2006; Gould et al. 2008). A simpler method is to calculate genetic
23 gain as a percentage of the population mean in the selection trait. This approach been
24 applied to *Eucalyptus globulus* Labill. on a 10-year rotation (Sanhueza et al. 2002) and to
25 other species for comparing the predicted gain with realised gain at mid-rotation (Vergara
26 et al. 2004; Weng et al. 2008; Verryen et al. 2009; Ye et al. 2010).

27
28 Early-rotation measurements can be used to predict stand volume gain if genetic
29 correlations are large enough and the traits are expressed at an equivalent genetic scale.
30 There are few published estimates of genetic correlations between predicted and realised
31 gain in forest trees. Ye et al. (2010) studied 15-year old *Pseudotsuga menziesii* var.

1 *menziesii* (Mirb.) Franco gains trials and reported a correlation of 0.77 between predicted
2 and realised gain amongst 20 families. This estimate was based on best linear unbiased
3 predictions (BLUPs) representing predicted breeding values for stem volume and least-
4 square means (LSMs) for stand volume in block-plots. Jansson et al. (1998) used linear
5 mixed models to jointly model *Pinus sylvestris* L family data from single-tree plots and
6 block plots and estimate the correlations between them. They reported additive genetic
7 correlations between stem volume in single-tree plots and stand volume in block plots
8 ranging from 0.63 to 0.99, with an average of about 0.87.

9
10 Predicted and realised gains have been compared using LSM volume of seed orchard
11 entries in block-plots and predictions based on the BLUPs of parents represented in the
12 deployment. In these cases, the seed orchard entries are comprised of seed from a whole
13 orchard which may have been rouged or they represent different classes of genetic gain
14 collected from an orchard. Following this approach, Verry et al. (2009) reported an
15 average 14% realised gain in stem volume of *Eucalyptus grandis* Hill ex Maiden. per
16 generation over three generations compared to a predicted 13% gain. Ye et al. (2010)
17 reported a realised gain in stand volume of *Pseudotsuga menziesii* var. *menziesii* of 17%
18 compared to a predicted 16% from analysis of single-tree plots. Poorer correspondence
19 between predicted and realised gain has also been reported. Weng (2008) found that
20 realised gain in stand volume of *Pinus banksiana* Lamb. following seed orchard rouging
21 was nearly one-third lower than predicted (realised gain of 13% versus predicted gain
22 18%). Similarly, average genetic gains in mean annual increment of *Pinus elliottii*
23 Engelm, var *elliottii* across 38 block-plot trials in southern USA were found to be almost
24 one-fifth lower than predicted for two breeding populations (Vergara et al. 2004).

25
26 *E. globulus* is a highly valued pulpwood plantation species and is the subject of advanced
27 genetic improvement programs. Growth improvement of *E. globulus* is commonly
28 achieved by selection on diameter at breast height (DBH) and height (e.g. Li et al. 2007;
29 Costa e Silva et al. 2009; Callister et al. 2011). We measured stand volume in a series of
30 four block-plot progeny trials of full-sib families in Western Australia after 7-9 years of a
31 10-year pulpwood rotation. The same families were represented in line-plot trials in the

1 same region, which were measured for DBH and height at 3.5 years. The objectives of
2 the study were to (A) quantify the genetic control of stand volume at late-rotation in
3 block-plot trials; (B) estimate the genetic correlations between early-rotation stem size in
4 line plots and late-rotation stand volume in block plots and (C) compare predicted and
5 realised gain estimates using a range of individual-tree traits for the prediction.

7 MATERIALS AND MEASUREMENTS

8 *Breeding Population and Test Sites*

9 This study was based on a second-generation breeding population which was previously
10 described as ‘EGP1’ by Callister et al. (2011). The population consisted of 153 full-sib *E.*
11 *globulus* families representing 86 parents from seven Australian provenances (SE
12 Tasmania, W Tasmania, NE Tasmania, Furneaux Group, King Island, Gippsland, and
13 Otways) and a Portuguese and a Californian land race. The parents were first-generation
14 selections from 84 open-pollinated (OP) families collected from native trees and land-
15 race plus-trees. Two parents were selected from the same OP family in two instances.
16 The parents were mated using an incomplete factorial design which included crosses
17 within and between provenances/land races. There was no reciprocal crossing. Five
18 commercial bulk collections and 13 unselected open-pollinated native families were
19 included as ‘checks’. The trials were established at seven locations in the south west of
20 Western Australia in 1999; three line-plot (or row-plot) trials (L1-3) and four block-plot
21 trials (B1-4; Figure 1; Table 1). The trial sites were within 200 km of each other and had
22 previously been used for agriculture. All full-sib families and checks were represented in
23 the line-plot series and a subset of 86 control-pollinated families and seven checks were
24 established in the block-plot series. From 22 to 86 full-sib families were measured at
25 each block-plot site (Table 1) and a total of 647 block-plots contributed data to this
26 analysis. The representation of genetic entries in block-plot trials was highly unbalanced.
27 For example, four families were each represented by 35 to 64 plots across the four trials,
28 while 30 families were represented by only one plot at one of the trials.

29
30 [INSERT TABLE 1 NEAR HERE] [INSERT FIGURE 1 NEAR HERE]

1
2 The line-plot trials consisted of five replicates of 10 incomplete blocks. Each block
3 contained 18 to 21 five-tree plots to which control-pollinated families and checks were
4 randomly assigned. The block-plot trials were established with incomplete blocks of 16 to
5 20 block-plots. Each block plot was five rows wide and eight planting spaces long (40
6 planting spaces per plot). Tree spacing throughout was 5×2.5 m (800 stems ha⁻¹) except
7 at site B1, where it was 5×2.0 m (1000 stems ha⁻¹). Soils were typical for the region,
8 classified as duplex or gravelly duplex and greater than 3 m deep (Table 1). Anaerobic
9 subsoils occurred within 3 m depth at sites B2 and B4.

10
11 Climate information for each trial site was obtained by spatial interpolation of available
12 information from nearby weather stations using ‘Data Drill’ (Jeffrey et al. 2001). The
13 climate at all sites could be described as ‘mediterranean’ with hot, dry summers and cool,
14 wet winters. However, rainfall across the region in the years following trial
15 establishment was less than the thirty-year average and ranged from an average 461
16 mm.year⁻¹ at site L2 to 694 mm.year⁻¹ at site B3 (Table 1). Pan evaporation was 2.3 to
17 3.3 times greater than rainfall (Table 1).

18 19 *Data measurement and augmentation*

20 Every tree in the line-plot trials was assessed for total height and diameter at 1.3 m
21 (breast height; DBH) at 3.5 years. On trees with two stems (incidence less than 4%), the
22 DBH of both stems was measured and entered as the square root of the sum of the
23 squares. Sectional area at breast height was calculated from DBH assuming each stem
24 was circular. Block-plots were measured at 9 years at sites B1, B3, and B4 and at 7 years
25 at site B2. DBH of all trees was measured. At site B2, the height of all plot trees in the
26 18 central planting positions was measured. At B1, B3 and B4, heights of a subsample of
27 nine trees per plot were measured, comprising the six trees in the plot centre and the three
28 largest remaining trees (total n=4650). The following fixed-effects model was fitted to
29 these data using Genstat Version 13 (VSN International, UK):

$$[1] \text{HT}_{ijk} = \mu + \text{DBH}_{ijk} + \text{Fam}_i + \text{Block/Site}_j + \text{Fam}_i.\text{DBH}_{ijk} + \text{Block/Site}_j.\text{DBH}_{ijk} + e_{ijk},$$

where HT_{ijk} and DBH_{ijk} are height and DBH of the k^{th} tree from the i^{th} family in the j^{th} incomplete block, Fam_i and Block/Site_j are direct effects of the i^{th} family and the j^{th} incomplete block (nested within site) on height, $\text{Fam}_i.\text{DBH}_{ijk}$ and $\text{Block/Site}_j.\text{DBH}_{ijk}$ are effects of the i^{th} family and the j^{th} incomplete block (nested within site) on the slope of relationship between HT and DBH, and e_{ijk} is random error. Model [1] explained 75% of observed variation in height and all fitted effects were significant ($p < 0.001$). Residual plots confirmed that the DBH-HT relationship was linear and residuals were homoscedastic and normally distributed. The unmeasured heights of 14,746 trees at sites B1, B3 and B4 were then predicted using their DBH measurements and the coefficients estimated in fitting Model [1].

Total over-bark stem volume of each measured tree in each trial was calculated using the following volume function which had been parameterized with data from West Australian *E. globulus* trees ranging from 30 dm³ to 400 dm³ in volume:

$$[2] \quad V_{\text{over-bark}} = 0.1248(\text{DBH}_{\text{over-bark}})^{1.3651} H^{1.1779},$$

where $V_{\text{over-bark}}$ is expressed in dm³, $\text{DBH}_{\text{over-bark}}$ is measured in cm and tree height, H , is measured in m.

Trees in the central 18 planting positions of each block-plot were identified at sites B2, B3, and B4 and formed the basis for all subsequent analyses at those sites. Trees on the periphery of block-plots were excluded to remove potential competition from adjacent plots. The within-plot positions of trees were poorly identified during the measurement at site B1, so all trees at site B1 contributed to subsequent analyses. Stand volume (m³ ha⁻¹) was calculated for each block-plot by summing tree volumes and dividing by the plot area.

1 ANALYSES AND RESULTS

2 *The general statistical framework of genetic analyses*

3 Genetic analyses were conducted using ASReml version 3.0 (Gilmour et al. 2009) within
4 the framework of the general linear mixed model:

5

$$6 \quad [3] \quad \mathbf{y} = \mathbf{X}\mathbf{b} + \mathbf{Z}\mathbf{u} + \mathbf{e}$$

7

8 where $\mathbf{y}$ is the vector of observed values for the response variable, sorted by site for
9 multi-site analyses; $\mathbf{b}$ and $\mathbf{u}$ are vectors of fixed and random effects, respectively, $\mathbf{Z}$ and
10 $\mathbf{X}$ are incidence matrices relating observations to model effects and $\mathbf{e}$ is the vector of
11 residuals. The distribution of random effects was assumed to be multivariate normal with
12 means and (co)variances defined by:

13

$$14 \quad [4] \quad \begin{bmatrix} \mathbf{u} \\ \mathbf{e} \end{bmatrix} \sim N \left(\begin{bmatrix} \mathbf{0} \\ \mathbf{0} \end{bmatrix}, \begin{bmatrix} \mathbf{G} & \mathbf{0} \\ \mathbf{0} & \mathbf{R} \end{bmatrix} \right)$$

15

16 where $\mathbf{G}$ and $\mathbf{R}$ are the (co)variance matrices relating to $\mathbf{u}$ and $\mathbf{e}$, respectively, and $\mathbf{0}$ is a
17 null matrix. Each of the j effects in $\mathbf{u}$ were assumed to be mutually independent with
18 (co)variance $\mathbf{G}_i$, so that $\mathbf{G} = \oplus_{i=1}^j \mathbf{G}_i$, where $\oplus$ denotes the direct sum operation.

19 Standard errors of functions of variance components were calculated in ASReml using an
20 approximation of a first-order Taylor series expansion (Gilmour et al. 2009).

21

22 *Single-site analyses of stem size in line-plot trials*

23 Methods

24 Height, DBH, sectional area, and stem volume data from line-plot trials were first
25 analysed using univariate, single-site mixed models to understand the genetic architecture
26 for selection (line-plot) traits. We expected the variance functions for DBH and sectional
27 area to be almost identical, but that sectional area might be expressed on a larger scale.
28 In each model, the terms in $\mathbf{b}$ comprised fixed effects for the mean, replicates, and
29 checks. The random terms in $\mathbf{u}$ represented provenance effects of parents with the design

matrices for the maternal and paternal provenances overlaid upon each other, general combining ability (GCA) given by overlaying the design matrices for the maternal and paternal parents, specific combining ability (SCA) given by family-specific effects, plot effects, trial design effects of factors blocks, rows, and columns, and a term for additional within-plot variance of checks. Residuals were generally decomposed into spatially dependent (ξ) and spatially independent (η) components according to:

$\mathbf{R} = \sigma_{\xi}^2 [AR1(\rho_{col}) \otimes AR1(\rho_{row})] + \sigma_{\eta}^2 \mathbf{I}_n$, where σ_{ξ}^2 and σ_{η}^2 are spatially dependent and spatially independent residual variances, respectively, $\otimes$ is the Kronecker product and $AR1(\rho)$ represents a first-order autoregressive correlation matrix (Cullis and Gleeson 1991; Dutkowski et al. 2006). No variance was explained by the spatial autocorrelation process for DBH and sectional area at site L1. The significance of SCA effects was determined with one-tailed likelihood ratio tests (LRTs) with $\chi_{0.5}^2$ (Stram and Lee 1994). Additive genetic variance (σ_A^2) was estimated as $4\hat{\sigma}_{GCA}^2$, where $\hat{\sigma}_{GCA}^2$ is the estimated GCA variance, dominance genetic variance (σ_D^2) was estimated as $4\hat{\sigma}_{SCA}^2$, where $\hat{\sigma}_{SCA}^2$ is the estimated SCA variance, and phenotypic variance (σ_P^2) was estimated as $2\hat{\sigma}_{GCA}^2 + \hat{\sigma}_{SCA}^2 + \hat{\sigma}_{plot}^2 + \hat{\sigma}_{block}^2 + \hat{\sigma}_{row}^2 + \hat{\sigma}_{col}^2 + \hat{\sigma}_{\eta}^2$, where $\hat{\sigma}_{plot}^2$ is the variance due to plots, $\hat{\sigma}_{block}^2$ is the variance due to incomplete blocks, $\hat{\sigma}_{row}^2$ is the variance due to trial rows, and $\hat{\sigma}_{col}^2$ is the variance due to trial columns. Coefficients of additive genetic variation (CV_A) were estimated for each trait and site by $\hat{CV}_A = \hat{\sigma}_A / \bar{X}$, where $\bar{X}$ is the mean. Narrow-sense heritabilities (h^2) and dominance proportions (d^2) were estimated for each trait at each site by $\hat{h}^2 = \hat{\sigma}_A^2 / \hat{\sigma}_P^2$ and $\hat{d}^2 = \hat{\sigma}_D^2 / \hat{\sigma}_P^2$.

Results

$\hat{CV}_A$ was smallest for DBH (mean 0.057), followed by height (mean 0.073), sectional area (mean 0.108), and stem volume (mean 0.141; Tables 2 and 3). Single-site $\hat{h}^2$ ranged from 0.12 to 0.24 for height, 0.08 to 0.12 for DBH, 0.09 to 0.13 for sectional area and from 0.14 to 0.19 for stem volume (Tables 2 and 3). Estimated dominance genetic effects were considerably smaller at site L2, where it was only significant for height ($p < 0.05$;

Table 2). Although $\hat{h}^2$ and $\hat{d}^2$ were nearly equivalent for DBH and sectional area, the quadratic relationship between the traits resulted in CV_A for sectional area that were nearly double those for DBH. Similarly, $\hat{h}^2$ and $\hat{d}^2$ for stem volume were generally intermediate between those for height and DBH, while CV_A for stem volume were more than double the average CV_A for height and DBH.

[INSERT TABLES 2 AND 3 NEAR HERE]

The mean $\hat{\sigma}_D^2 : \hat{\sigma}_A^2$ was 1.31 for height, 0.72 for DBH, 0.57 for sectional area, and 0.64 for stem volume (Tables 2 and 3). SCA effects did not suggest the presence of between-provenance heterosis. Provenance variance ($\hat{\sigma}_{PROV}^2$) was relatively small for all traits, as indicated by the mean ratio $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ of 0.15 (Tables 2 and 3). No provenance variance was found for height and stem volume at site L2, and in most cases the standard error was larger than the provenance variance estimate (Tables 2 and 3).

Multi-site analyses of stem size in line-plot trials

Methods

Multi-site models were fitted to selection trait data to determine inter-site correlations and heterogeneity in genetic variance ratios. These results were used to determine the best model for data standardisation and to assist the interpretation of results from region-wide analyses. Data for multi-site analyses were adjusted for spatial trends by subtracting from $\mathbf{y}$ the row and column effects and spatial residuals. Five models (Lms-1 to Lms-5) were then fitted to full-sib family data for each trait (Table 4). They contained effects of site means and replicates in $\mathbf{b}$; parent provenances (overlaid design matrices), GCA (overlaid design matrices), SCA, blocks and plots in $\mathbf{u}$. Genetic correlations were constrained to be uniform between pairs of sites (as in Li et al. 2007; Costa e Silva et al. 2009). Further constraints on genetic (co)variances were varied between models to test the hypotheses that inter-site SCA or GCA correlations were 1 using LRTs with $\chi_{0.5}^2$ and to test heterogeneity in the ratios $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ and $\hat{\sigma}_{SCA}^2 : \hat{\sigma}_{GCA}^2$ using LRTs with χ_2^2

(Table 4). There was insufficient provenance variance at site L2 to fit this term with inter-site correlation.

[INSERT TABLE 4 NEAR HERE]

Results

Uniform among-sites correlation estimates in GCA ranged from 0.71 for sectional area to 0.87 for height and they were significantly different from 1 in each case (Table 5). On the other hand, uniform correlations in SCA were only significantly different to 1 for height (r_{SCA} 0.63; Table 5). Ratios of $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ were not significantly heterogeneous among sites for any trait ($p > 0.05$) and uniform estimates ranged from 0.04 for height to 0.15 for sectional area (Table 5). Ratios of $\hat{\sigma}_{SCA}^2 : \hat{\sigma}_{GCA}^2$ were significantly heterogeneous for height, DBH and sectional area due to smaller SCA variance at site L2 (Tables 2, 3, and 5). Nevertheless, $\hat{\sigma}_{SCA}^2 : \hat{\sigma}_{GCA}^2$ was not significantly heterogeneous for stem volume ($p > 0.05$), for which the uniform estimate was 0.55 (Table 5).

[INSERT TABLE 5 NEAR HERE]

Region-wide analyses of stem size in line-plot trials

Methods

The region-wide analysis of each selection trait provided the breeding values for use in selection and genetic-gain estimation. Site additive standard deviations from Model Lms-5 (Table 4) were used to standardise the spatially adjusted data for scale effects. The variance constraints in Lms-5 made it as close as possible to the region-wide model, in which the standardised data were used. Re-scaled data (x_{adj}) were also centred to the respective trial mean ($\bar{x}_i$): $x_{adj} = (x - \bar{x}_i) / \hat{\sigma}_{A_i}$. This standardisation procedure is convenient because back-transformed breeding values (as a percentage of the population mean) are obtained by simply multiplying BLUPs by CV_A .

The region-wide models of height, DBH, sectional area, and stem volume each contained effects of replicates within sites, and checks in **b**. Effects of parent provenances,

provenance×site, GCA, GCA×site, SCA, SCA×site, blocks-in-sites, plots-in-sites, and additional within-plot effects for checks were included in **u** (using overlaid design matrices for provenance, GCA and their site interaction effects). Residual variances were heterogeneous. Average narrow-sense heritabilities (h_{ave}^2) and dominance proportions (d_{ave}^2) were estimated for each trait by:

$$\hat{h}_{ave}^2 = 4\hat{\sigma}_{GCA}^2 / (2\hat{\sigma}_{GCA}^2 + \hat{\sigma}_{GCA \times Site}^2 + \hat{\sigma}_{SCA}^2 + \hat{\sigma}_{SCA \times Site}^2 + \sum_{i=1}^3 (\hat{\sigma}_{block_i}^2 + \hat{\sigma}_{plot_i}^2 + \hat{\sigma}_{error_i}^2)) / 3$$

$$\text{and } \hat{d}_{ave}^2 = 4\hat{\sigma}_{SCA}^2 / (2\hat{\sigma}_{GCA}^2 + \hat{\sigma}_{GCA \times Site}^2 + \hat{\sigma}_{SCA}^2 + \hat{\sigma}_{SCA \times Site}^2 + \sum_{i=1}^3 (\hat{\sigma}_{block_i}^2 + \hat{\sigma}_{plot_i}^2 + \hat{\sigma}_{error_i}^2)) / 3$$

where genetic variances are estimated across sites, $\hat{\sigma}_{block_i}^2$ is block variance of the i^{th} site, $\hat{\sigma}_{plot_i}^2$ is plot variance of the i^{th} site, and $\hat{\sigma}_{error_i}^2$ is the residual variance of the i^{th} site.

Results

Region-wide average heritability estimates ($\hat{h}_{ave}^2$) were 0.18 for height, 0.07 for DBH and sectional area, and 0.14 for stem volume (Table 6). Region-wide $\hat{\sigma}_D^2 : \hat{\sigma}_A^2$ ranged from 0.38 for stem volume to 0.81 for DBH. Provenance effects were small at the region-wide scale, with $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ ranging from 0.10 for height to 0.22 for DBH (Table 6).

[INSERT TABLE 6 NEAR HERE]

Survival in block-plots

Methods and Results

Variable survival in block-plots was examined for genetic effects and to provide a basis for removing stand volume data that were significantly impacted by poor survival. The proportion of surviving trees within each block-plot ranged from 23% to 100% with a mean of 87%. It was assumed to follow a binomial distribution and modeled, site by site, with a generalized linear mixed model using a logit link function:

$$[5] \quad \eta_{ijkl} = \log[\pi/(1 - \pi)] = \mu + B_k + GCA_i + GCA_j + SCA_{ij} + e_{ijkl}$$

where η_{ijk} is the link function, π is the proportion of surviving trees, μ is the conditional mean, B_k is the fixed effect of the k^{th} incomplete block, GCA_i and GCA_j are the random effects of the i^{th} maternal parent and j^{th} paternal parent with overlaid design matrices, SCA_{ij} is the random effect of specific combining ability between parents i and j , and e_{ijk} is the random residual with $N(0, \sigma_e^2 \mathbf{I})$. Data were excluded from plots in two drainage lines which cut across site B4.

There was no detectable variance associated with parental or family genetic effects at any of the block-plot sites. We therefore considered variable survival to be a nuisance in the analysis of stand volume, and we sought to remove its effect by imposing site-specific survival thresholds for the inclusion of stand volume data. A fixed-effects model was fitted to data from each site separately using Genstat version 13 (VSN International, UK) to determine the significance of survival to plot stand volume:

$$[6] \quad V_{nkl} = \mu + S_n + F_k + B_l + e_{nkl}$$

where V_{nkl} is the stand volume of the n^{th} plot, μ is the mean, S_n is the survival of the n^{th} plot, F_k is the random effect of the k^{th} family, B_l is the random effect of the l^{th} block, and e_{nkl} is random error.

Survival was a significant determinant of stand volume at each site when all measured plots were considered ($p < 0.05$). Data from plots with the poorest survival were then excluded and the remaining data were re-analysed with Model [6]. If survival remained a significant effect in the remaining data, plots with the next-poorest survival were excluded and the remaining data were re-analysed. This process was repeated until survival was not a significant effect in the analysis of remaining data ($p < 0.05$). The lowest plot survival in the remaining data at the completion of this process was considered to be the survival threshold for the site.

Survival thresholds were found to be 63% at B1 and 78% at B2, B3, and B4. Stand volume data from 5% to 11% of measured plots at each site were excluded from further analyses because their survival was poorer than these thresholds.

Single-site analyses of stand volume in block-plot trials

Methods

The analysis of stand volume commenced at the single-site level to understand the genetic architecture of this trait. Trial means and checks were included as fixed effects in **b**, while random effects of the parent provenance, GCA, SCA, and incomplete block effects were generally included in **u**. Design matrices were overlaid for provenance and GCA effects. SCA was not fitted at site B2, where only 22 families were measured. An additional random term was fitted to within-plot variance of checks at sites B1, and B3, the only trials in which checks were represented sufficiently to estimate this term.

Residuals at sites B1, B3 and B4 were decomposed into spatially dependent (ξ) and spatially independent (η) components using the same first-order autoregressive structure as for the line-plot trials (AR1 $\times$ AR1), except that in this case the experimental units were plots rather than trees. Spatial analysis was not possible at site B2, where measured plots were widely scattered amongst unmeasured plots.

Heritability of stand volume in block-plots was estimated by

$$\hat{h}_{STAND}^2 = 4\hat{\sigma}_{GCA}^2 / (2\hat{\sigma}_{GCA}^2 + \hat{\sigma}_{SCA}^2 + \hat{\sigma}_{block}^2 + \hat{\sigma}_{\xi}^2 + \hat{\sigma}_{\eta}^2), \text{ where terms are defined as for line-plot}$$

analyses above. Dominance proportion of stand volume in block plots was estimated by:

$$\hat{d}_{STAND}^2 = 4\hat{\sigma}_{SCA}^2 / (2\hat{\sigma}_{GCA}^2 + \hat{\sigma}_{SCA}^2 + \hat{\sigma}_{block}^2 + \hat{\sigma}_{\xi}^2 + \hat{\sigma}_{\eta}^2) \cdot \hat{h}_{STAND}^2 \text{ and } \hat{d}_{STAND}^2 \text{ differ from narrow-}$$

sense heritability and dominance proportion estimates because among-trees size variation was averaged within plots and did not contribute to phenotypic variance. Although spatial residual variance is not customarily included in the phenotypic variance component (e.g. Ye and Jayawickrama 2008), it was included here to produce estimates of variance components at the whole-of-site scale, which we considered to be appropriate for deployed gain in stand volume.

Results

Mean stand volume was $138 \text{ m}^3\text{ha}^{-1}$, $79 \text{ m}^3\text{ha}^{-1}$, $97 \text{ m}^3\text{ha}^{-1}$, and $92 \text{ m}^3\text{ha}^{-1}$ at sites B1 to B4 respectively, corresponding to mean annual increment of $15.3 \text{ m}^3\text{ha}^{-1}\text{y}^{-1}$, $11.3 \text{ m}^3\text{ha}^{-1}\text{y}^{-1}$, $10.8 \text{ m}^3\text{ha}^{-1}\text{y}^{-1}$, and $10.2 \text{ m}^3\text{ha}^{-1}\text{y}^{-1}$, respectively. At sites B2, B3, and B4 genetic variation in stand volume was overwhelmingly attributed to additive effects (GCA), with nil estimated variance for SCA at sites B3 and B4, and nil or negligible variance for provenance effects (Table 7). However, at site B1 genetic control of stand volume followed a different pattern, whereby estimated provenance variance was nearly two-thirds of $\hat{\sigma}_{GCA}^2$ and $\hat{\sigma}_{SCA}^2$ was large but not significant (Table 7). $\hat{h}_{STAND}^2$ ranged from 0.25 to 0.76 (Table 7), indicating that a substantial proportion of variance in late-rotation stand volume was attributed to additive genetic variance. CV_A for stand volume ranged from 0.138 to 0.217 (mean 0.178).

[INSERT TABLE 7 NEAR HERE]

Multi-site analyses of stand volume in block-plot trials

Methods

Multi-site models were fitted to stand volume to determine inter-site correlations and explore the best way to accommodate the larger provenance and SCA variances at site B1. These results were used to determine the best model for data standardisation and which terms to include in the region-wide and unified analyses. Data for multi-site analyses of stand volume were adjusted for spatial trends at sites B1, B3, and B4 by subtracting spatial residuals from $\mathbf{y}$. Six models (Bms-1 to Bms-6) were then fitted to full-sib family data (Table 8). The models each included site mean effects in $\mathbf{b}$; heterogenous GCA effects (with overlaid design matrices) and block effects in $\mathbf{u}$. The inconsistent partitioning of genetic variance at site B1 was examined by comparing the Akaike information criterion (AIC) of models that included provenance and SCA only at B1 with models that included provenance and SCA variance at each site as a uniform ratio of GCA variance. The model sequence also allowed testing whether a uniform between-site additive genetic correlation was significantly different from 1.

[INSERT TABLE 8 NEAR HERE]

Results

The best multi-site model by AIC included provenance and SCA effects only at site B1 (Model Bms-1; Table 8). The uniform inter-site GCA correlation from this model was 0.44, which was significantly different from 1 ($p < 0.001$, LRT Bms-2 versus Bms-1 using $\chi^2_{0.5}$). The omission of the provenance term at site B1 resulted in only a minor increase of 0.5 in AIC (Model Bms-3; Table 8), so Bms-3 was adopted as the most parsimonious multi-site model to form the basis of the proceeding with further analyses. Model Bms-4, which included provenance and SCA effects as uniform proportions of GCA variance, had an AIC 4.3 greater than model Bms-1, which included provenance and SCA only at site B1 (Table 8). Comparison of models Bms-3 and Bms-6 showed that SCA variance at site B1 was significant ($p = 0.02$, LRT using $\chi^2_{0.5}$) when included with correlated GCA effects in analysis of multi-site data.

Region-wide analysis of stand volume in block-plot trials

Methods

Region-wide analysis of stand volume was conducted to estimate the proportion of phenotypic variance represented by additive genetic variance at a regional scale and as a precursor to the unified GCA analyses. Full-sib family data were spatially adjusted and standardised for scale effects using additive standard deviations from Model Bms-3. The region-wide model included site means in **b** and a single variance for GCA, SCA at site B1, GCA×site interaction, and site-specific block effects in **u**. Site residuals were heterogeneous. Across-sites average heritability in stand volume ($h^2_{STAND_{ave}}$) was

estimated as:
$$\hat{h}^2_{STAND_{ave}} = \frac{4\hat{\sigma}^2_{GCA}}{(2\hat{\sigma}^2_{GCA} + \hat{\sigma}^2_{GCA \times Site} + [\hat{\sigma}^2_{SCA(SiteB1)} + \sum_{i=1}^4 (\hat{\sigma}^2_{block_i} + \hat{\sigma}^2_{error_i})] / 4)}.$$

Results

Additive genetic variance in stand volume was an appreciable proportion of total variance at the regional scale, with $\hat{h}_{STAND_{ave}}^2$ of 0.41 (Table 9). GCA×site variance was more than 50% greater GCA variance, which is expected from the uniform across-sites GCA correlation estimate of 0.44 reported above.

[INSERT TABLE 9 NEAR HERE]

Unified GCA analyses of line- and block-plot data

Methods

Joint analyses of GCA using data from both plot types (‘unified GCA’ analyses) were undertaken to estimate genetic correlations between individual-tree selection traits at age 3.5 years and stand volume at age 7-9 years. Four models were fitted with correlations at the site scale, each including one selection trait and stand volume. Line-plot and block-plot data representing full-sib families were adjusted only for spatial trend, as previously described. The models included all trial means and replicates within line-plot trials in **b**, while **u** contained provenance effects for each line-plot trial, GCA for each trial, SCA for each line-plot trial and at site B1, and block and plot effects at sites where they were present. Provenance variance at line-plot trials was fitted as heterogeneous without inter-site correlation and SCA correlations were heterogeneous between line-plot trials.

Design matrices were overlaid for provenance and GCA effects. GCA was modeled with: (A) uniform correlation in the selection trait between sites, (B) uniform correlation in stand volume between sites, and (C) uniform correlation between sites, between traits.

Four unified GCA models were fitted to estimate genetic correlations at the regional scale. Each model included one selection trait and stand volume. The full-sib family data were adjusted for spatial trend and for scale effects by additive standard deviation, as previously described. All trial means and replicates within line-plots were included in **b**, while **u** contained one variance for provenance effects across line-plot trials, a variance for GCA across each trial type, SCA at each individual line-plot trial and at site B1, and block and plot effects at sites where they were present. SCA correlations were heterogeneous between line-plot trials. Design matrices were overlaid for provenance

and GCA effects. GCA effects were modeled with one inter-trait correlation which was compared with 1 using LRT with $\chi^2_{0.5}$.

Results

The uniform additive genetic correlations between 3.5-year selection traits and late-rotation stand volume at the site scale were 0.65 for height, 0.59 for DBH, 0.58 for sectional area, and 0.65 for stem volume (Table 10). Between-site genetic correlations within traits were similar to those reported above. The additive genetic correlations between 3.5-year selection traits and late-rotation stand volume at the regional scale ranged from 0.86 for height to 0.90 for stem volume (Table 10). Each correlation was significantly different to 1 ($p < 0.05$).

[INSERT TABLE 10 NEAR HERE]

Comparison of predicted and realised genetic gains

Methods

Although the above results demonstrate good genetic correlation between early-age selection traits and late-rotation stand volume, differences in the scale of genetic effects could result in very different estimates of genetic gain. Our final analysis was therefore intended to compare the genetic gain of the best families in selection traits (i.e. predicted gain) with realised gain of the same families in late-rotation stand volume.

BLUPs from region-wide models of line-plot data were used to estimate values for provenance, GCA and SCA in units of additive standard deviation. The predicted genetic value of each family was calculated for height, DBH, sectional area, and stem volume by summing the relevant provenance, GCA, and SCA values and multiplying the result by the mean CV_A of the three line-plot trials (0.073 for height, 0.057 for DBH, 0.108 for sectional area, and 0.141 for stem volume).

Genetic entries in block-plot trials were considered (perhaps counterintuitively) to be fixed effects represented by least-square means (LSMs) for stand volume. This

distinction from our preceding analyses of random genetic effects is important because
 LSMs are less conservative than BLUPs and they will produce larger estimates of genetic
 gain when sample sizes are relatively small (as is the case here). The statistical literature
 does not provide a consistent view on how to judge an effect to be considered fixed or
 random, or even how the terms are defined (Gelman 2005). McCulloch et al. (2011 ;
 Sect 1.6b) provides the following guidance: “*In endeavoring to decide whether a set of
 effects is fixed or random, the context of the data, the manner in which they were
 gathered and the environment from which they came are the determining factors... are
 the levels of the factor going to be considered a random sample from a population of
 values which have a distribution?*” In our case, the families which were entered into
 block-plot trials *could* be considered as random representations of a distribution
 representing all possible full-sib *E. globulus* families, and this formed the basis of the
 preceding analyses. However, for estimating genetic gain, we considered the “context of
 the data” differently. We considered a hypothetical scenario in which the full range of
 families was tested in line-plots, measured at 3.5 years and represented by random
 effects, or BLUPs. In this scenario, the best-ranked families were then deployed
 commercially (mimicked by the block-plots in our experiment), prompting the question
 ‘*how well did we predict the realised genetic gain in our commercial stands?*’ In this
 case, once those best families were selected for deployment they were no longer
 considered to be drawn randomly from a distribution but rather they represented a
 particular treatment with a fixed effect. It should be also noted that the experimental
 errors in the block-plot analyses were independent of the errors adjusted for when making
 BLUP estimates in line-plots. Treating families as fixed effects is comparable to an
 assessment of inventory plots in which ‘selected’ and ‘average’ families were established.
 Stand volume data were adjusted for spatial trend (as described above) and then divided
 by the respective site mean. This simple transformation largely overcame site
 productivity differences and the intuitive representation of stand volume as a percent of
 the site mean is useful for making comparisons with predicted levels of genetic gain.
 Representation of better- and poorer-performing families across sites was sufficiently
 balanced for bias to be negligible. The statistical model contained fixed effects for

genetic entries and random effects for incomplete blocks. Families and checks with only one or two block-plots were excluded from realised gain estimation as their LSMs were considered to be too imprecisely estimated. The LSMs of the remaining 42 full-sib families and four checks were compared with their predicted gain in height, DBH, sectional area, and stem volume. The predicted and realised genetic gain of the best 12%, 24%, and 50% of the 42 full-sib families were calculated as:

$$[7] \text{ Gain} = \frac{\sum_{i=1}^n x_i}{n} - \frac{\sum_{j=1}^{42} x_j}{42},$$

where x_i is the genetic value (either BLUP or LSM) of the i^{th} family in an elite group of n families on the basis of the selection variable (height, DBH, sectional area, or stem volume), and x_j is the genetic value (either BLUP or LSM) of the j^{th} family in the entire group ($i \subseteq j$).

Results

Realised genetic gain was around 23% for the best 5 families, 14% for the best 10 families, and 7% for the best half of the group of 42 full-sib families (Table 11).

Predicted genetic gains varied with CV_A of the selection trait, from 4.7% DBH gain to 14.6% stem volume gain in the best 5 families (Table 11). This scale effect resulted in under-predictions of genetic gain when using height, DBH, or sectional area (Table 11). Genetic gain predictions were closest to realised gain estimates for stem volume; realised gain was 8 percentage points greater than predicted for the best 5 families, 1.2 percentage points greater than predicted for the best 10 families, and 0.3 percentage points smaller than predicted for the best 21 families. The correlation between realised gain and predicted gain in stem volume of individual full-sib families and checks was 0.82 (Figure 2). Agreement between predicted and realized gain was better for families and checks with 10 or more block-plots (Figure 2), suggesting that some of the imprecision was due to small sample size of block-plots.

Three checks were represented by more than 10 block-plots and realised gain for these entries ranged from -5.4% to -1.5% of the population mean (Figure 2). Two of these

1 entries were bulk collections from open-pollinated seed orchards and one was a collection
2 from a native stand which had performed well in previous trials.

3
4 Although we stand by our use of LSM estimates for realised gain, some readers will no
5 doubt be interested in the gains estimated if we had treated families as random effects.
6 As expected, we found that treating families as random resulted in smaller estimates of
7 gain, and that this reduction was dependent on the family sample size. Gain estimates for
8 families represented by more than 10 plots differed by less than 1.5% when treating
9 families as random. Gain estimates for families represented by 3 to 8 plots differed by up
10 to 13.3%. The BLUP-based realised gain was 17.4% for the best 5 families, 10.2% for
11 the best 10 families, and 5.6% for the best 21 families ranked by 3.5-year stem volume.

12
13 [INSERT TABLE 11 NEAR HERE]

14
15 [INSERT FIGURE 2 NEAR HERE]

16 17 18 **DISCUSSION**

19 Single-site $\hat{h}^2$ and $\hat{d}^2$ for DBH were comparable to previous estimates for full-sib *E.*
20 *globulus* in Australia (Volker 2002; Li et al. 2007) and DBH $\hat{h}^2$ was similar to the
21 average value of 0.12 from 40 Portuguese sites reported by Araujo et al. (2012). Single-
22 site $\hat{h}^2$ and $\hat{d}^2$ for height were within the range reported by Li et al. (2007). Previous
23 reports of genetic parameters for sectional area and stem volume are lacking for full-sib
24 *E. globulus* families. Inter-site r_{GCA} estimates in selection traits between 0.71 and 0.87
25 suggest that additive genetic $\times$ site interaction is a not a major concern for the population
26 in this region, even though it may be statistically significant. Previous estimates of
27 additive genetic correlation in full-sib *E. globulus* DBH have been reported between 0.60
28 and 0.83 (Volker 2002; Li et al. 2007; Costa e Silva et al. 2009; Araujo et al. 2012).

1 The genetic architecture of our population in Western Australia appears broadly similar
2 to that of another full-sib *E. globulus* population studied by Li et al. (2007) across sites
3 ranging from Tasmania and Victoria to Western Australia. Our estimate of 1.31 for a
4 uniform $\hat{\sigma}_D^2 : \hat{\sigma}_A^2$ for height was similar to the value of 1.20 reported by Li et al. (2007)
5 although our estimate of 0.64 for DBH was smaller than their reported value of 1.00.
6 Like Li et al. (2007), we found that heterogeneity in $\hat{\sigma}_D^2 : \hat{\sigma}_A^2$ was more significant for
7 height than for DBH and we also found that SCA effects were more site-dependent for
8 height than for DBH. The $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ estimates that we obtained were far smaller than
9 similar statistics reported by Li et al. (2007), who used the sub-race classification of
10 Dutkowski and Potts (1999). This suggests that some of the variance attributed to
11 additive effects in our analyses may have been distributed to subrace effects if we had
12 used the Dutkowski and Potts (1999) classification rather than coarser geographical
13 provenances and land races.

14
15 We found that stand volume in block-plots was under substantial additive genetic control,
16 with region-wide additive variance representing 41% of the phenotypic variance (given
17 by $\hat{h}_{STAND_{ave}}^2$). This result is crucial to commercial *E. globulus* improvement programs
18 which aim to increase harvest yield. CV_A for stand volume in our study (mean 0.18)
19 corresponded with those of Jansson (2007), who reported CV_A between 0.14 and 0.24
20 (mean 0.17) for fifteen block-plot family trials of *Pinus sylvestris*. $\hat{h}_{STAND}^2$ of stand
21 volume at sites B2 and B4 were larger than expected for $\hat{h}^2$ of a growth trait, due
22 probably to the lack of between-trees variance in the denominator. Unusually large
23 heritability estimates for stand volume were also calculated by Jansson et al. (1998), who
24 reported a mean of 0.59 from full-sib families of *Pinus sylvestris* at five block-plot trials.

25
26 Although the genetic correlations between early-age selection traits and stand volume are
27 of critical importance for effective yield improvement, they have rarely been quantified
28 due to the cost of implementing block-plot family trials. The uniform between-site r_{GCA}
29 estimates of 0.59 to 0.65 that we found between selection traits and stand volume (Table

10) are at the low end of the range reported for the same traits in *Pinus sylvestris* (0.53 to 0.99; Jansson et al. 1998). The larger r_{GCA} estimates of 0.86 to 0.90 at the regional scale (Table 10) are more meaningful measures of the inter-trait correlations because they relate to the breeding value estimates of stem size and of stand volume across the region.

Our inter-trait r_{GCA} results suggest that height or DBH of line-plot entries were nearly as good as stem volume for indirect selection on harvest stand volume. On the other hand, predicted gains based on height were about half the realised gain and those based on DBH were about a third of the realised gain. CV_A of selection traits determines the scale of predicted genetic gain from parent selection. For example, sectional area had a larger $\hat{CV}_A$ than did DBH and it produced a less biased estimate of stand volume gain.

Nevertheless, we found that only stem volume was expressed on a scale suitable for predicting stand volume gain. *E. globulus* breeders have published variances and heritability estimates for DBH (e.g. Costa e Silva et al. 2009; Stackpole et al. 2010; Araujo et al. 2012) and sometimes for DBH and height (e.g. Lopez et al. 2002; Li et al. 2007), but rarely for stem volume (c.f. Sanhueza et al. 2002). Our findings prompt a call for publication of genetic parameters (including CV_A) for individual stem volume, as it appears to be expressed on a scale more similar to that of stand volume.

Most of the published studies comparing predicted and realised gain have confounded the issues of genetic correlation and scale by using a handful of seed orchard entries to represent different levels of realised gain. In two cases volume gains have met or surpassed predicted values (e.g. Verryen et al. 2009; Ye et al. 2010), and the correlation of 0.77 between predicted and realised gains for families reported by Ye et al. (2010) is similar to our correlation of 0.82 (see Figure 2). In other cases, predicted gains have not been achieved in comparable gains trials (e.g. Vergara et al. 2004; Weng et al. 2008).

One reason that realised gains might not meet predicted gains is that between-tree competition in intimate genetic mixtures is not representative of block plantings where individuals compete with genetically similar material. This complication may be particularly evident amongst clones (e.g. Sharma et al. 2008; Stanger et al. 2011) and its

1 effects were perhaps not apparent in our study due to the early assessment of line-plot
2 trials just before canopy closure and the onset of stronger competition. Another potential
3 problem with early-age predictions of gain is that percentage gain can decrease over time
4 when absolute gain is unchanging (e.g. see the gain predictions of Carson et al. 1999).
5 The trial sites in our study may not have expressed this phenomenon because they were
6 strongly water-limited and would not have grown much in the last half of the rotation.

7
8 *E. globulus* can be feasibly reproduced in large full-sib families (Collins and Callister
9 2010), presenting the prospect of single-family deployment to commercial plantations.
10 Such a strategy would be predicated on an assumption that gain predictions from line-plot
11 progeny trials were unbiased and precise. Although we found that region-wide stem
12 volume BLUPs formed unbiased estimates of genetic gain in stand volume, the precision
13 of individual family gain predictions may be insufficient for single-family deployment.
14 For example, there were cases in which predicted gain of a well-represented family was
15 more than 10% below predicted gain (see Figure 2). Seed-based deployment of *E.*
16 *globulus* should therefore continue as family mixtures or as individual families only if a
17 reasonable number of families is represented in each commercial stand. These guidelines
18 will help to counter the risk that the genetic value of particular families is over-predicted.

19
20 We found that variable survival in block plots was not under measurable genetic control
21 and we removed its effect by setting thresholds for data exclusion. Another option was to
22 apply a model combining genetic effects and stocking (e.g. Jannsson et al. 1998), but we
23 found that too few plots were significantly affected by mortality to adequately
24 parameterize such a model. Stand volume of *E. globulus* in the trial region has
25 previously been found to be insensitive to stocking rates above about 600 stems ha⁻¹
26 (White et al. 2009). This is consistent with the survival thresholds that we applied, which
27 corresponded to around 630 stems ha⁻¹.

28
29 A number of outstanding issues could be addressed in further studies with improved
30 designs. We had too few block-plot sites to adequately understand the GCA×site
31 interaction in stand volume, and our study was limited to sites that were relatively poor

for first-rotation *E. globulus*. Nevertheless, such sites were representative of a large portion of the West Australian estate in the first rotation and they are likely to be more typical of second-rotation sites due to water and nutrient depletion in the first rotation (Mendham et al. 2011). An improved study design may feature adjacent line-plot and block-plot family trials along productivity gradients and across regions, to better quantify the effects of site and plot type on genetic correlations and the scale of genetic variation. Results from a large experiment such as this may be suitable for developing a gain-prediction tool based on integrated selection-age measurements and growth models. On the other hand, our results do not suggest that a growth model is needed for genetic gain prediction within a more limited environment. We were satisfied that the gain predictions of the best 25% and 50% of families based on stem volume were unbiased, and the gain under-prediction for the best 12% of families could be due to random variation at the family level considering that only five families were represented in that stratum. Our study was too small to form any reliable conclusions about the degree of non-additive genetic control of stand volume. An experiment that aimed to reliably define SCA effects on stand volume would need to be extremely large, considering that the estimation of the dominance variance requires about 20 times as much data as for additive variance, for equivalent accuracy (Miszta 1997).

Conclusions

We have found that late-rotation stand volume was under substantial additive genetic control, with $\hat{h}_{STAND_{ave}}^2$ of 0.41 at the regional scale. Our results suggest that BLUP analysis of early-rotation height, DBH, or stem volume from line-plot progeny data provides adequately for indirect selection on stand volume in *E. globulus* and that predicted gains in stem volume are reasonable measures of stand volume gain. Predicted gains in height or DBH were found to under-estimate stand volume gains due to scale differences. Therefore, we recommend expressing *E. globulus* progeny trial data as stem volume if unbiased genetic gain predictions are required and we encourage the publication of genetic parameters for stem volume in addition to DBH and height.

1

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8

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1 **Table 1.** Summary of site conditions and other particulars for three line-plot (L) trials and
2 four block-plot (B) trials established 1999

Trial code	L1	L2	L3	B1	B2	B3	B4
Trees per plot	5	5	5	40	40	40	40
Locality	Rocky Gully	South Stirling	Manypeak s	Bridget own	Frankl and	Napie r	Manyp eaks
Latitude	34° 31'	34° 26'	34° 43'	34° 6'	34° 21'	34° 46'	34° 45'
Longitude	117° 13'	118° 30'	118° 10'	116° 25'	117° 12'	117° 58'	118° 12'
Soil Description:	Gravelly duplex	Gravelly duplex	Duplex	Gravell y duplex	Gravel ly duplex anaero bic subsoi ls	Grav elly duple x	Duplex anaero bic subsoil s
Average Annual Rainfall (mm) ¹	605	461	598	592	503	694	598
Average Evapn/Rainfall	2.3	3.3	2.5	2.2	2.8	2.1	2.5
CP Families in this study	153	143	148	49	22	86	41
Measurement age (years)	3.5	3.5	3.5	9	7	9	9
Mean±SD Height (m)	8.8±1.6	7.3±1.4	10.2±1.8	15.8±2. 9	12.2± 2.0	14.6± 2.3	14.3±1. 7
Mean±SD DBH (cm)	10.6±1.7	8.2±1.8	12.0±2.3	18.6±4. 6	13.4± 3.0	15.2± 3.8	15.1±3. 1

3 ¹From planting to measurement age

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Table 2. Estimates of variance components and functions from single-site analyses of tree height and diameter at breast height in three line-plot trials (L1-L3) at age 3.5 years. Estimated standard errors are in parentheses.

		Tree Height (m)						Diameter at Breast			
		L1		L2		L3		L1		L2	
Mean		8.77		7.30		10.23		10.55		8.21	
Genetic variances	$\hat{\sigma}_{PROV}^2$	0.024	(0.022)	0.000	(0.000)	0.011	(0.021)	0.013	(0.015)	0.007	(0.007)
	$\hat{\sigma}_{GCA}^2$	0.056	(0.024)	0.094	(0.023)	0.166	(0.050)	0.070	(0.023)	0.090	(0.023)
	$\hat{\sigma}_{SCA}^2$	0.151	(0.035)***	0.031	(0.018)*	0.154	(0.047)***	0.050	(0.024)**	0.012	(0.007)
Trial design variances	$\hat{\sigma}_{block}^2$	-	(-)	-	(-)	-	(-)	0.020	(0.011)	-	(-)
	$\hat{\sigma}_{row}^2$	0.020	(0.015)	-	(-)	-	(-)	0.070	(0.025)	-	(-)
	$\hat{\sigma}_{col}^2$	-	(-)	-	(-)	-	(-)	0.020	(0.014)	0.039	(0.014)
	$\hat{\sigma}_{plot}^2$	0.022	(0.022)	0.066	(0.024)	0.050	(0.038)	-	(-)	-	(-)
Residual variances and autocorrelations	$\hat{\sigma}_{checks}^2$	1.018	(0.119)	0.597	(0.115)	1.173	(0.184)	1.329	(0.173)	0.418	(0.173)
	$\hat{\sigma}_{\eta}^2$	1.509	(0.040)	1.411	(0.038)	2.233	(0.063)	2.345	(0.057)	2.827	(0.057)
	$\hat{\sigma}_{\xi}^2$	0.312	(0.116)	0.121	(0.044)	0.520	(0.155)	-	(-)	0.097	(0.097)
	$\hat{\rho}_{col}$	0.91	(0.03)	0.92	(0.04)	0.87	(0.05)	-	(-)	0.96	(0.05)
	$\hat{\rho}_{row}$	0.98	(0.01)	0.96	(0.03)	0.98	(0.01)	-	(-)	0.97	(0.01)
	$\hat{\sigma}_{\eta}^2$	0.12	(0.05)	0.22	(0.05)	0.24	(0.07)	0.11	(0.03)	0.12	(0.03)
Genetic parameters	$\hat{d}^2$	0.33	(0.07)	0.07	(0.04)	0.22	(0.07)	0.08	(0.04)	0.02	(0.04)
	$\hat{\sigma}_D^2 : \hat{\sigma}_A^2$	2.69	(1.48)	0.33	(0.23)	0.93	(0.46)	0.71	(0.47)	0.13	(0.47)
	$\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$	0.43	(0.44)	0.0	(0.00)	0.06	(0.13)	0.19	(0.23)	0.08	(0.23)
	$C\hat{V}_A$	0.054	(0.035)	0.084	(0.042)	0.080	(0.044)	0.050	(0.029)	0.073	(0.029)
	$\hat{h}^2$	0.12	(0.05)	0.22	(0.05)	0.24	(0.07)	0.11	(0.03)	0.12	(0.03)

$\hat{\sigma}_{PROV}^2, \hat{\sigma}_{GCA}^2, \hat{\sigma}_{SCA}^2, \hat{\sigma}_{block}^2, \hat{\sigma}_{row}^2, \hat{\sigma}_{col}^2, \hat{\sigma}_{plot}^2, \hat{\sigma}_{checks}^2, \hat{\sigma}_{\eta}^2, \hat{\sigma}_{\xi}^2$ = estimated provenance, GCA, SCA, incomplete block, row, column, plot, additional checks residual, spatially independent residual, and spatial residual variances, respectively; $\hat{\rho}_{col}, \hat{\rho}_{row}$ = estimated residual autocorrelation coefficients in the column and row directions, respectively; $\hat{h}^2, \hat{d}^2, \hat{\sigma}_D^2 : \hat{\sigma}_A^2, \hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2, C\hat{V}_A$ = estimated narrow-sense heritability, dominance proportion, ratio of dominance variance to additive variance, ratio of provenance variance to GCA variance, and coefficient of additive variation, respectively. Significance of $\hat{\sigma}_{SCA}^2$ by LRT using $\chi_{0.5}^2$ $p < 0.05$ *, $p < 0.01$ **, $p < 0.001$ ***. Only non-zero variances are shown for trial design factors.

Table 3. Variance components and functions from single-site analyses of sectional area and stem volume in three line-plot trials (L1-L3) at age 3.5 years. Estimated standard errors are in parentheses.

		Sectional area (cm ²)			Stem Volume (cm ³)	
		L1	L2	L3	L1	L2
Mean		89.8	55.5	116.7	42.0	24.7
Genetic variances	$\hat{\sigma}_{PROV}^2$	3.1 (3.6)	0.7 (1.8)	5.4 (6.1)	1.67 (1.53)	0.00 (0.00)
	$\hat{\sigma}_{GCA}^2$	17.3 (5.5)	14.6 (4.1)	29.4 (11.4)	5.99 (1.88)	4.82 (1.11)
	$\hat{\sigma}_{SCA}^2$	12.9 (5.8)**	0.7 (3.1) ^{NS}	27.1 (15.0)*	6.03 (2.00)***	0.97 (0.81)
Trial design variances	$\hat{\sigma}_{block}^2$	5.7 (2.8)	- (-)	9.3 (11.0)	- (-)	- (-)
	$\hat{\sigma}_{row}^2$	18.8 (6.5)	- (-)	- (-)	3.72 (1.62)	- (-)
	$\hat{\sigma}_{col}^2$	5.6 (3.4)	6.9 (3.2)		- (-)	0.86 (0.51)
	$\hat{\sigma}_{plot}^2$	- (-)	- (-)	- (-)	- (-)	- (-)
Residual variances and autocorrelations	$\hat{\sigma}_{checks}^2$	269.1 (39.4)	48.3 (27.6)	521.4 (96.4)	78.96 (10.71)	10.85 (5.81)
	$\hat{\sigma}_{\eta}^2$	561.2 (13.6)	427.7 (10.9)	1246.8 (33.7)	145.9 (3.62)	88.53 (2.34)
	$\hat{\sigma}_{\xi}^2$	- (-)	30.0 (28.3)	212.9 (61.7)	18.09 (7.44)	4.86 (1.41)
	$\hat{\rho}_{col}$	- (-)	0.98 (0.02)	0.89 (0.04)	0.92 (0.03)	0.84 (0.01)
	$\hat{\rho}_{row}$	- (-)	0.97 (0.03)	0.97 (0.01)	0.97 (0.02)	0.94 (0.01)
Genetic parameters	$\hat{h}^2$	0.11 (0.03)	0.13 (0.03)	0.09 (0.03)	0.14 (0.04)	0.19 (0.04)
	$\hat{d}^2$	0.08 (0.04)	0.01 (0.03)	0.08 (0.04)	0.14 (0.05)	0.04 (0.03)
	$\hat{\sigma}_D^2 : \hat{\sigma}_A^2$	0.75 (0.47)	0.05 (0.22)	0.92 (0.73)	1.01 (0.53)	0.20 (0.11)
	$\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$	0.18 (0.23)	0.05 (0.13)	0.18 (0.23)	0.28 (0.28)	0.0 (0.00)
	CV_A	0.093 (0.052)	0.138 (0.073)	0.093 (0.058)	0.117 (0.065)	0.178 (0.03)

$\hat{\sigma}_{PROV}^2, \hat{\sigma}_{GCA}^2, \hat{\sigma}_{SCA}^2, \hat{\sigma}_{block}^2, \hat{\sigma}_{row}^2, \hat{\sigma}_{col}^2, \hat{\sigma}_{plot}^2, \hat{\sigma}_{checks}^2, \hat{\sigma}_{\eta}^2, \hat{\sigma}_{\xi}^2$ = estimated provenance, GCA, SCA, incomplete block, row, column, plot, additional checks residual, spatially independent residual, and spatial residual variances, respectively; $\hat{\rho}_{col}, \hat{\rho}_{row}$ = estimated residual autocorrelation coefficients in the column and row directions, respectively; $\hat{h}^2, \hat{d}^2, \hat{\sigma}_D^2 : \hat{\sigma}_A^2, \hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2, CV_A$ = estimated narrow-sense heritability, dominance proportion, ratio of dominance variance to additive variance, ratio of provenance variance to GCA variance, and coefficient of additive variation, respectively. Significance of $\hat{\sigma}_{SCA}^2$ by LRT using $\chi_{0.5}^2$ $p < 0.05$ *, $p < 0.01$ **, $p < 0.001$ ***. Only non-zero variances are shown for trial design factors.

Table 4. Constraints applied to multi-site models to estimate inter-site genetic correlations (Lms-1), to facilitate the testing of site $\times$ SCA interaction (Lms-1, -2), site $\times$ GCA interaction (Lms-1, -3 if $r_{SCA} \neq 1$ or Lms-2, -3 if $r_{SCA} = 1$), heterogeneity in $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ (Lms-1, -4), and heterogeneity in $\hat{\sigma}_{SCA}^2 : \hat{\sigma}_{GCA}^2$ (Lms-4, -5).

Model	$\hat{\sigma}_{error}^2$	$\hat{\sigma}_{GCA}^2$	$\hat{\sigma}_{PROV}^2$	$\hat{\sigma}_{SCA}^2$	r_{GCA}	r_{PROV}	r_{SCA}
Lms-1	H	H	H	H	k_1	0	k_2
Lms-2	H	H	H	H	k_1	0	1
Lms-3	H	H	H	H	1	0	k_2 or 1
Lms-4	H	H	$\alpha \hat{\sigma}_{GCA}^2$	H	k_1	0	k_2
Lms-5	H	H	$\alpha \hat{\sigma}_{GCA}^2$	$\beta \hat{\sigma}_{GCA}^2$	k_1	0	k_2

H - heterogeneous variances across sites; k_1, k_2 - uniform across-site correlations

Table 5. Selected results from multi-site models of height, DBH, sectional area and stem volume; uniform inter-site correlation in GCA (k_1), uniform inter-site correlation in SCA (k_2), and constants in the ratios $\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ and $\hat{\sigma}_{SCA}^2 : \hat{\sigma}_{GCA}^2$ (α and β , respectively).

Standard errors of correlations are in parentheses. Correlations were tested for significance against $H_0: k_i = 1$ using LRT with $\chi_{0.5}^2$. Ratio constants α and β were tested for heterogeneity using LRT with χ_2^2 .

Trait	$r_{GCA}(k_1)$		$r_{SCA}(k_2)$		$\hat{\sigma}_{PROV}^2 / \hat{\sigma}_{GCA}^2 (\alpha)$	$\hat{\sigma}_{SCA}^2 / \hat{\sigma}_{GCA}^2 (\beta)$
Height	0.87	(0.06)**	0.63	(0.11)***	0.04 ^{NS}	1.31***
DBH	0.73	(0.10)***	1.00	(bounded)	0.14 ^{NS}	0.64*
Sectional area	0.71	(0.09)***	1.00	(bounded)	0.15 ^{NS}	0.52*
Stem Volume	0.80	(0.07)***	0.87	(0.19) ^{NS}	0.13 ^{NS}	0.55 ^{NS}

p<0.05 *, p<0.01 **, p<0.001 ***

Table 6. Genetic and residual variance components and variance functions from region-wide analyses of tree height, and DBH, sectional area and stem volume in line-plot trials at age 3.5 years using standardised data. Estimated standard errors are in parentheses.

	Tree Height	DBH	Sectional area	Stem Vol
$\hat{\sigma}_{PROV}^2$	0.028 (0.04)	0.034 (0.04)	0.031 (0.04)	0.028 (0.03)
$\hat{\sigma}_{PROV \times Site}^2$	0.000 (n/a)	0.003 (0.01)	0.006 (0.01)	0.004 (0.01)
$\hat{\sigma}_{GCA}^2$	0.265 (0.07)	0.159 (0.05)	0.167 (0.05)	0.209 (0.05)
$\hat{\sigma}_{GCA \times Site}^2$	0.028 (0.01)	0.094 (0.03)	0.085 (0.03)	0.039 (0.02)
$\hat{\sigma}_{SCA}^2$	0.153 (0.05)	0.129 (0.04)	0.095 (0.04)	0.079 (0.03)
$\hat{\sigma}_{SCA \times Site}^2$	0.167 (0.04)	0.013 (0.04)	0.032 (0.04)	0.081 (0.03)
$\hat{\sigma}_{error(site_L1)}^2$	4.464 (0.10)	7.521 (0.18)	7.035 (0.17)	5.078 (0.12)
$\hat{\sigma}_{error(site_L2)}^2$	6.237 (0.17)	9.933 (0.24)	9.502 (0.23)	5.983 (0.15)
$\hat{\sigma}_{error(site_L3)}^2$	4.281 (0.11)	8.957 (0.23)	8.569 (0.22)	5.520 (0.14)
$\hat{h}_{ave}^2$	0.18 (0.04)	0.07 (0.02)	0.07 (0.02)	0.14 (0.03)
$\hat{d}_{ave}^2$	0.10 (0.03)	0.06 (0.02)	0.04 (0.02)	0.05 (0.02)
$\hat{\sigma}_D^2 : \hat{\sigma}_A^2$	0.58 (0.26)	0.81 (0.43)	0.57 (0.33)	0.38 (0.20)
$\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$	0.10 (0.15)	0.22 (0.26)	0.19 (0.23)	0.13 (0.16)

$\hat{\sigma}_{PROV}^2, \hat{\sigma}_{PROV \times Site}^2, \hat{\sigma}_{GCA}^2, \hat{\sigma}_{GCA \times Site}^2, \hat{\sigma}_{SCA}^2, \hat{\sigma}_{SCA \times Site}^2$ = estimated provenance, provenance $\times$ site, GCA, GCA $\times$ site, SCA, SCA $\times$ site variances, respectively; $\hat{\sigma}_{error(site_L1)}^2, \hat{\sigma}_{error(site_L2)}^2, \hat{\sigma}_{error(site_L3)}^2$ = estimated residual variances for sites L1, L2, and L3, respectively; $\hat{h}^2, \hat{d}^2, \hat{\sigma}_D^2 : \hat{\sigma}_A^2, \hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$ = estimated narrow-sense heritability, dominance proportion, ratio of dominance variance to additive variance, and ratio of provenance variance to GCA variance, respectively.

Table 7. Variance components and functions from single-site analyses of stand volume in four block-plot trials. Estimated standard errors are in parentheses.

	Site B1	Site B2	Site B3	Site B4
Mean (m ³ ha ⁻¹)	138	79	97	92
$\hat{\sigma}_{PROV}^2$	60 (79)	0 (0)	0 (0)	5 (18)
$\hat{\sigma}_{GCA}^2$	96 (114)	74 (38)	45 (19)	98 (38)
$\hat{\sigma}_{SCA}^2$	324 (213) ^{NS}	n/f (n/a)	0 (0)	0 (0)
$\hat{\sigma}_{block}^2$	273 (188)	128 (66)	0 (0)	0 (0)
$\hat{\sigma}_{checks}^2$	302 (355)	n/f (n/a)	30 (55)	n/f (n/a)
$\hat{\sigma}_{\eta}^2$	305 (78)	115 (25)	83 (34)	105 (28)
$\hat{\sigma}_{\xi}^2$	438 (250)	n/f (n/a)	320 (86)	277 (87)
$\hat{\rho}_{col}$	0.76 (0.18)	n/f (n/a)	0.68 (0.11)	0.68 (0.14)
$\hat{\rho}_{row}$	0.83 (0.12)	n/f (n/a)	0.71 (0.11)	0.81 (0.07)
$\hat{h}_{STAND}^2$	0.25 (0.29)	0.76 (0.28)	0.36 (0.14)	0.68 (0.21)
$\hat{d}_{STAND}^2$	0.85 (0.55)	n/f (n/a)	0.00 (0.00)	0.00 (0.00)
$\hat{\sigma}_D^2 : \hat{\sigma}_A^2$	3.39 (5.67)	n/f (n/a)	0.00 (0.00)	0.00 (0.00)
$\hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2$	0.63 (1.25)	0.00 (0.00)	0.00 (0.00)	0.05 (0.19)
$C\hat{V}_A$	0.142 (0.155)	0.217 (0.156)	0.138 (0.089)	0.215 (0.135)

$\hat{\sigma}_{PROV}^2, \hat{\sigma}_{GCA}^2, \hat{\sigma}_{SCA}^2, \hat{\sigma}_{block}^2, \hat{\sigma}_{checks}^2, \hat{\sigma}_{\eta}^2, \hat{\sigma}_{\xi}^2$ = estimated provenance, GCA, SCA, incomplete block, additional checks residual, spatially independent residual, and spatial residual variances, respectively; $\hat{\rho}_{col}$, $\hat{\rho}_{row}$ = estimated residual autocorrelation coefficients in the column and row directions, respectively; $\hat{h}_{STAND}^2, \hat{d}_{STAND}^2, \hat{\sigma}_D^2 : \hat{\sigma}_A^2, \hat{\sigma}_{PROV}^2 : \hat{\sigma}_{GCA}^2, C\hat{V}_A$ = estimated narrow-sense heritability of stand volume, dominance proportion of stand volume, ratio of dominance variance to additive variance, ratio of provenance variance to GCA variance, and coefficient of additive variation, respectively. Significance of $\hat{\sigma}_{SCA}^2$ at B1 tested by LRT using $\chi_{0.5}^2$; n/f: not fitted.

Table 8. Constraints applied to models to compare fitting provenance and SCA effects at only Site B1 (Bms-1 to -3) with fitting them as a uniform proportion of $\hat{\sigma}_{GCA}^2$ (Bms-4, Bms-5); to estimate across-sites genetic correlations (Bms-1, Bms-4); to test site $\times$ GCA interaction (Bms-1,-2); to test SCA at site B1 (Bms-3,-6); to test SCA as a uniform proportion of $\hat{\sigma}_{GCA}^2$ (Bms-5, -6); to test provenance at site B1 (Bms-1, -3), and to test provenance as a uniform proportion of $\hat{\sigma}_{GCA}^2$ (Bms-4, -5). The log-likelihood and change in AIC (relative to Model 1) are presented for each model.

Model	$\hat{\sigma}_{error}^2$	$\hat{\sigma}_{GCA}^2$	r_{GCA}	$\hat{\sigma}_{prov}^2$	$\hat{\sigma}_{SCA}^2$	Log-L	ΔAIC
Bms-1	H	H	k_1	at B1	at B1	-1546.2	0.0
Bms-2	H	H	1	at B1	at B1	-1588.2	82.0
Bms-3	H	H	k_1	0	at B1	-1547.4	0.5
Bms-4	H	H	k_1	$\alpha \hat{\sigma}_{GCA}^2$	$\beta \hat{\sigma}_{GCA}^2$	-1548.4	4.3
Bms-5	H	H	k_1	0	$\beta \hat{\sigma}_{GCA}^2$	-1548.4	2.4
Bms-6	H	H	k_1	0	0	-1549.6	2.8

Table 9. Variance components and $\hat{h}_{STAND_{ave}}^2$ from region-wide analysis of standardised stand volume in four block-plot trials. Estimated standard errors are in parentheses.

σ^2 component	Stand Volume
$\hat{\sigma}_{GCA}^2$	0.098 (0.043)
$\hat{\sigma}_{GCA \times Site}^2$	0.152 (0.039)
$\hat{\sigma}_{SCA(site_B1)}^2$	0.450 (0.210)
$\hat{\sigma}_{block(site_B1)}^2$	0.249 (0.121)
$\hat{\sigma}_{block(site_B2)}^2$	0.523 (0.272)
$\hat{\sigma}_{error(site_B1)}^2$	0.287 (0.044)
$\hat{\sigma}_{error(site_B2)}^2$	0.499 (0.104)
$\hat{\sigma}_{error(site_B3)}^2$	0.242 (0.035)
$\hat{\sigma}_{error(site_B4)}^2$	0.158 (0.019)
$\hat{h}_{STAND_{ave}}^2$	0.41 (0.16)

$\hat{\sigma}_{GCA}^2, \hat{\sigma}_{GCA \times Site}^2, \hat{\sigma}_{SCA(site_B1)}^2, \hat{\sigma}_{block(site_B1)}^2, \hat{\sigma}_{block(site_B2)}^2$ = estimated GCA, GCA $\times$ site, SCA at site B1, incomplete block at site B1, and incomplete block at site B2 variances, respectively;

$\hat{\sigma}_{error(site_B1)}^2, \hat{\sigma}_{error(site_B2)}^2, \hat{\sigma}_{error(site_B3)}^2, \hat{\sigma}_{error(site_B4)}^2$ = estimated residual variances for sites B1, B2, B3, and B4, respectively;

$\hat{h}_{STAND_{ave}}^2$ = estimated average narrow-sense heritability for stand volume.

1 **Table 10.** Uniform inter-site additive genetic correlations within and between plot types
2 at the site scale and between plot types at the regional scale. Estimated standard errors are
3 in parenthesis.

Traits	Plot	Selection Traits			
		Height	DBH	Sectional	StemVol
<i>Uniform correlations at the site scale</i>					
Between-site	Line-plot	0.88 (0.05)	0.73 (0.09)	0.71 (0.09)	0.82 (0.07)
Between-site stand	Block-plot	0.45 (0.13)	0.44 (0.13)	0.44 (0.13)	0.45 (0.13)
Selection-stand	Line v	0.65 (0.08)	0.59 (0.09)	0.58 (0.09)	0.65 (0.08)
<i>Single correlations between traits at the regional scale</i>					
Selection-stand	Line v	0.86 (0.08)	0.87 (0.09)	0.87 (0.09)	0.90 (0.07)

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5 **Table 11.** Predicted (BLUP-based) genetic gain for top-ranked families from four 3.5-
6 year selection traits in line-plot trials and realised (LSM) gain in stand volume for the
7 same families in late-rotation block-plots. Families were ranked by selection trait and
8 gains are expressed relative to the mean of 42 families which were represented by three
9 or more block-plots.

Selection Trait (CV_A)	Height (0.073)		DBH (0.057)		Sectional area (0.108)		Stem Volume (0.141)	
	Predicted	Realised	Predicted	Realised	Predicted	Realised	Predicted	Realised
Best 5 families (12%)	8.0%	20.5%	4.7%	20.2%	9.1%	20.5%	14.6%	22.6%
Best 10 families (24%)	6.9%	14.1%	4.0%	13.6%	7.7%	13.5%	12.4%	13.6%
Best 21 families (50%)	4.3%	7.7%	2.5%	6.6%	4.9%	7.1%	7.4%	7.1%

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1 **Figures**

2

3 **Figure 1.** Map of the line-plot trial locations (triangles) and block-plot trial locations
4 (circles) relative to population centres in southern West Australia (stars).

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Figure 2. Relationship between realised genetic gain in stand volume of families in block-plots (as a percentage of the site mean) and predicted genetic gain in stem volume in line-plots at 3.5 years (as a percentage of the population mean on a site where $CV_A = 0.141$) for full-sib families (circles) and checks (triangles) represented by more than 10 block plots (solid symbols) and 3 to 10 block-plots (open symbols). The line represents parity.

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Author/s:

Callister, AN;England, N;Collins, S

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