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Basic density of radiata pine in New Zealand: genetic and environmental factors

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Abstract Wood basic density is among the selection criteria for many fast-grown tree species, including *Pinus radiata* D. Don in New Zealand. Basic density was assessed in 23,330 stem cores from 18 trials to study the heritability, the relevance of environmental effects and the magnitude of genotype-by-environment (GxE) interaction. Site differences in annual average temperature dominated variability in this dataset, with lower latitude and altitude (i.e. warmer) sites displaying higher average density. Between highest- and lowest-density sites there was an 18% difference (302.7 vs. 358.4 kg m^{-3}) for the linear mean for cores of rings 1–5 and a 39% difference (329.7 vs. 459.1 kg m^{-3}) for the linear mean of rings 6–10. The estimated heritabilities fluctuated between 0.28 and 0.94 (mean, 0.6); however, basic density displayed little within-site variability (phenotypic coefficient of variation, <8%). Bivariate analyses were used to estimate between-site genetic correlations as an indication of GxE interaction. Only 57 out of the 153 pairs of trials contained enough information to estimate the between-site genetic correlations and, out of those, 15 estimates were not statistically significant. Moderate to high (0.46–0.96) significant genetic correlation estimates indicated that there was little interaction for basic density, suggesting no need to modify the breeding strategy to account for differential performance in this trait. Poor connectedness between trials could be depressing estimates of genetic correlations. This situation should be considered when designing genetic testing schemes, particularly when purposely inducing imbalance as in rolling front strategies.

Keywords Genetic correlation · Genotype-by-environment interaction · Wood properties · Connectedness · *Pinus radiata* · Basic density

Introduction

Predicting the genetic worth of individuals is crucial to tree breeding programs, and it is often based upon data from multiple genetic trials. Forest plantations are deployed in extensive, heterogeneous environments, and the spatial allocation of genetic trials aims to reflect this situation.

Environmental differences affect both the quantity and quality of wood produced. In terms of quantity, potential site productivity is commonly expressed using indices of tree height (e.g. site index) or volume (e.g. 300 index, Kimberley et al. 2005) at a standard age. The environmental effects on wood properties are sometimes mapped as 'quality regions'; as an example, Cown (1992, page 8) divided New Zealand in to three basic density regions: high, medium and low. It is expected that the *average* quantity and quality of wood will rise or fall depending on the productivity index or quality region where the trees are growing.

Nevertheless, one of the main questions during genetic testing is 'Will superior genotypes perform satisfactorily across heterogeneous environments?'. The varying effect of environmental conditions on

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the performance of different genotypes is termed genotype-by-environment (GxE) interaction. GxE interaction can be partitioned into effects due to heterogeneity of variances and to lack of correlation (Muir et al. 1992). Heterogeneous variances—related to changes of scale like site index—are not necessarily a problem. However, changes of ranking depending on testing environment may lead to more complex breeding and/or deployment strategies that require multiple breeding objectives (Howarth et al. 1997; Goddard 1998).

There are two naïve extremes when dealing with GxE interaction: assuming that either there is complete lack of interaction or that the interaction is important for every trait, site and genotype. Most breeding programs will fall between these extremes. In general terms, GxE interaction appears to be relatively important in *part* of the forest estate for growth traits (e.g. stem diameter, height and volume see Johnson and Burdon 1990; Carson 1991; Matheson and Wu 2005), but presents a small magnitude for wood properties (e.g. Kumar 2004; Gapare et al. 2009). Unfortunately, most studies for wood traits rely on a small number of both sites and genotypes or on indirect—and less accurate—assessments, like penetrometer readings to estimate basic density.

Pinus radiata D. Don is the most important temperate plantation species in the Southern hemisphere, covering over 3.7 million ha, mostly in New Zealand, Chile and Australia. The New Zealand radiata pine breeding program started in the 1950s initially focusing on growth, form and health traits—as did many tree breeding strategies around the world. The selection criteria were later extended to encompass traits that relate to the quantity and quality of wood produced (Jayawickrama and Carson 2000; Dungey et al. 2007). One such trait is wood basic density. While for solid wood production basic density should no longer be considered the paramount structural and appearance timber property (Apiolaza 2009) it is still an important trait, particularly for fiber and energy production as well as for 'carbon forestry'.

A thorough genetic testing system requires high connectedness (pedigree relationships) among trials, leading to a more precise estimate of genetic correlation, a more accurate comparison of estimated breeding values between trials and higher accuracy of selection (Kennedy and Trus 1993). Unfortunately, poor connectedness is not unusual in tree breeding, where often there are few parents in common among trials, even when tracing back the pedigree. This may be due to technical problems (propagation difficulties, differential site mortality, etc), limited resources, or simply oversight. In spite of connectedness issues, trials are frequently incorporated in genetic evaluations that attempt to compare genetic material *among* trials (e.g. Baltunis et al. 2009, for stem diameter). The implicit assumption is that GxE interaction is negligible; however, there is evidence to suggest that this interaction can be significant for stem diameter in New Zealand (e.g. Johnson and Burdon 1990; McDonald 2009).

In this research, the combined analysis of stem core data for basic density from over 23,000 trees distributed in 18 trials is presented. Then the variation of additive genetic control, the relevance of environmental effects and the magnitude of the interaction between genotypes and environment are reviewed. Finally, the role poor connectedness plays in our understanding of genetic parameters is discussed.

Materials and methods

This study considered 17 progeny trials across the New Zealand forest estate and one trial (D) in New South Wales (Australia), including a range of mating designs and field designs, as well as of environmental conditions (Table 1) and ages of assessment. Until recently, the New Zealand breeding program focused most testing in the Central North Island; only one of the trials (B) in Table 1 is located in the South Island, as shown in Fig. 1.

Basic density—oven-dry weight divided by green volume expressed in kg m^{-3}—was calculated for 5-mm diameter stem cores at breast height (1.3 m). A total of 23,330 trees were assessed, ranging from 246 trees in trial B to 3,000 trees in trials P and Q. There are 768 parents in the dataset (695 with progeny data), most of them representing the New Zealand land race, with the exception of parents in trial J, which contains Guadalupe Island hybrids. The stem cores include different numbers of rings (see Table 2), with more than half of the trials including rings 6–10, although there are cores covering rings 1–5, 1–7 and 1–8.

The genetic analyses considered two stages:

First, univariate analyses were run considering all genetic (additive and, when appropriate, dominance effects) and experimental design features (replicates, sets and plots). All effects, except for the overall mean, were considered as random.

For most trials the only significant ($p < 0.05$) random effect was additive genetic. Furthermore, dropping additional significant effects (in trials that presented them) changed heritability estimates by less than 2%. Therefore, univariate analyses were simplified from a general model including a fixed overall mean, and

Table 1 Establishment year, location (Latitude South and Longitude East), and environmental data from NIWA for the trials

Trial	Year	Latitude	Longitude	Altitude	Temperature	Rainfall
A	1987	36°21′49″	174°07′39″	100	14.5	1,202
B	1990	45°59′38″	170°11′43″	27	10.6	759
C	1993	39°13′56″	176°51′51″	451	11.1	1,553
D	1993	33°28′42″	149°01′31″	800	12.4	842
E	1995	38°16′52″	176°43′30″	332	12.9	1,762
F	1995	38°08′31″	176°34′14″	117	12.9	2,161
G	1988	36°21′39″	174°06′15″	81	15.0	1,283
H	1988	38°16′25″	175°52′09″	372	12.2	1,689
I	1988	37°53′17″	176°23′18″	98	13.8	1,839
J	2000	38°09′07″	176°36′41″	85	13.3	2,111
K	1989	36°21′39″	174°06′15″	81	15.6	1,259
L	1981	37°58′35″	176°32′22″	280	13.4	2,156
M	1985	38°13′39″	176°08′01″	678	10.7	1,561
N	1985	38°14′20″	175°59′40″	495	11.2	1,564
O	1987	38°37′30″	176°20′40″	565	10.9	1,358
P	1968	38°16′27″	176°41′15″	415	12.7	1,724
Q	1968	38°16′27″	176°41′15″	415	12.5	1,634
R	1969	38°45′54″	176°15′43″	700	10.6	1,720

Variables derived from GIS layers correspond to altitude (masl), temperature (annual average, °C), and rainfall (annual, mm)

random replicate, plot and additive effects to the following model:

$$y = Xb + Za + e \quad (1)$$

where y is the vector of phenotypic observations for a single site, b the vector of the fixed effect (overall mean), a the vector of additive genetic effects and e is the vector of residuals. X and Z are incidence matrices linking the phenotypes to the overall mean and additive genetic values vectors, respectively. The expected value and variances were $E[y] = Xb$, $\mathrm{Var}[a] = G = \sigma_a^2 A$ and $\mathrm{Var}[e] = R = \sigma_e^2 I$ for A the numerator relationship matrix and I an identity matrix. The residuals were assumed to be identically and independently normally distributed.

In a second stage, all pairs of trials were run as bivariate analyses, fitting only overall mean and additive

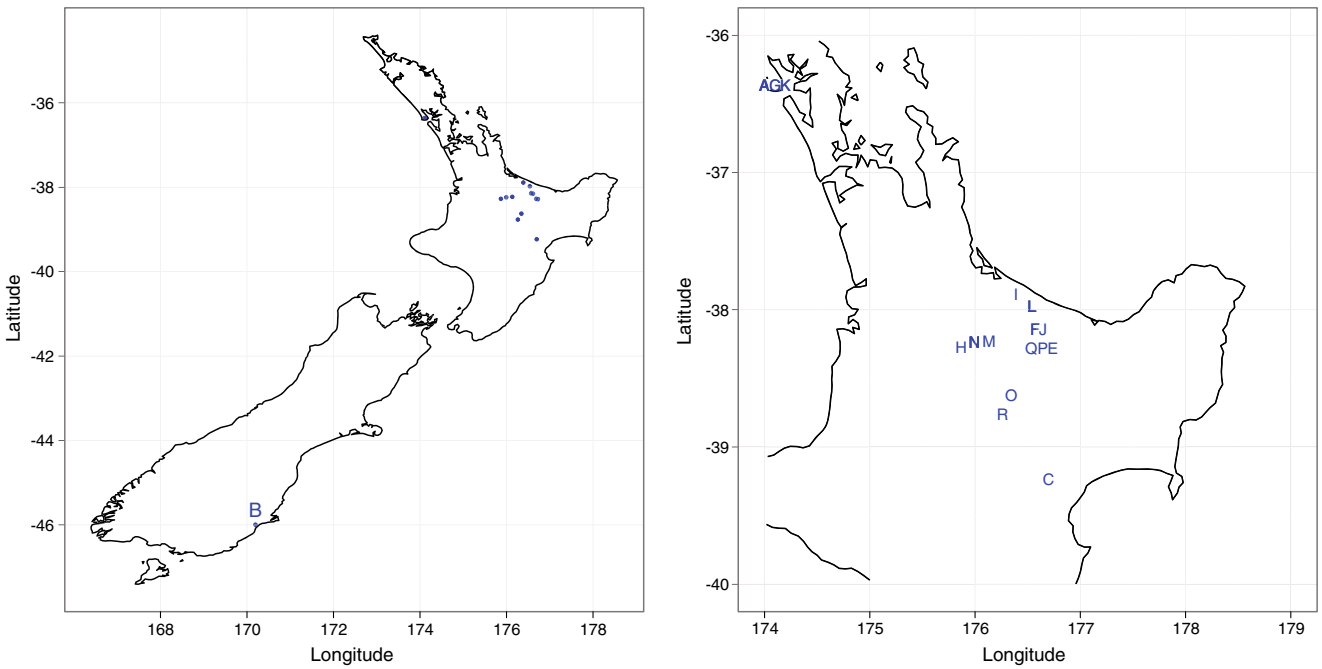

Fig. 1 Locations of trials in New Zealand, where letters correspond to trial codes in Table 1. Notice the poor coverage in the South Island (only trial B) and the absence of trial D located in Australia

Table 2 Descriptive statistics for phenotypic wood basic density, including range of rings sampled (rings), number of extracted cores (cores), mean trial density (mean, kg m^{-3}), standard deviation (SD) and coefficient of variation (CV, %)

Trial	Rings	Cores	Mean	SD	CV
A	6–10	1129	459.1	34.2	7.5
B	6–10	246	363.5	26.9	7.4
C	1–5	1524	343.0	20.5	6.0
D	1–8	2562	352.8	20.8	5.9
E	1–7	590	345.4	22.1	6.4
F	1–7	656	351.7	18.5	5.3
G	6–10	1054	448.4	35.0	7.8
H	6–10	642	329.7	21.2	6.4
I	1–10	288	344.3	24.4	7.1
J	1–5	2032	358.4	25.7	7.2
K	6–10	639	429.3	35.0	8.1
L	6–10	885	383.9	27.4	7.1
M	6–10	1745	365.6	27.8	7.6
N	6–10	1282	379.9	28.2	7.4
O	6–10	1631	356.9	24.2	6.8
P	1–5	3000	302.7	19.0	6.3
Q	6–10	425	375.9	29.9	8.0
R	1–5	3000	311.1	18.9	6.1

genetic effects. Equation 1 was expanded to accommodate two traits (stacking up the vectors), in such a way that b, a and e now contain the values for both trials. The variances were then $\mathrm{Var}[a] = G = G_0 \otimes A$ and $\mathrm{Var}[e] = R = R_0 \otimes I$, where $\otimes$ represents the Kronecker matrix product and:

$$G_0 = \begin{bmatrix} \sigma^2_{a_1} & \sigma_{a_{12}} \\ \sigma_{a_{12}} & \sigma^2_{a_2} \end{bmatrix} \quad R_0 = \begin{bmatrix} \sigma^2_{e_1} & \sigma_{e_{12}} \\ \sigma_{e_{12}} & \sigma^2_{e_2} \end{bmatrix} \tag{2}$$

Heritabilities (h^2) and genetic correlations (r_{12}) were estimated using the standard formulas:

$$\hat{h}^2 = \frac{\hat{\sigma}^2_a}{\hat{\sigma}^2_a + \hat{\sigma}^2_e} \quad r_{12} = \frac{\hat{\sigma}_{a_{12}}}{\sqrt{\hat{\sigma}^2_{a_1}\hat{\sigma}^2_{a_2}}}$$

All analyses were performed using asreml-r, which is an implementation of ASReml (Gilmour et al. 2002) for the R statistical software system (R Development Core Team 2008). An R script fitted all 18 single-site univariate analyses, as well as the $18 (18 - 1)/2 = 153$ bivariate analyses for all pairs of trials. The statistical significance of all covariance components was tested using a likelihood ratio test, while standard errors for heritabilities and genetic correlations were approximated using a Taylor series (Gilmour et al. 2002).

GPS trial coordinates were matched to New Zealand's National Institute of Water & Atmospheric Research climate data GIS layers, to link genetic performance and parameters with climatic descriptors. Climate data for trial D (in Australia) was obtained from ANUCLIM (Houlder et al. 2001).

Finally, the relationship between mean basic density and environmental factors was modeled using multiple linear regression. The mean basic density for each trial presented in Table 2 was treated as the response variable, while centered (i.e. expressed as deviation from the mean) temperature and rainfall from Table 1 were used as predictors. Centered regressions are easier to interpret, as the main effects and the intercept are interpreted based on deviations from the mean of the data (Gelman and Hill 2007, page 55). A dummy variable—coding for rings 1–5 and 6–10—was used to test for differences of intercept and slope between ring groups. The regression model was fitted using R.

Results

Table 2 provides phenotypic descriptive statistics for the trials. Basic density ranged from 302.7 kg m^{-3} in trial P to 459.1 kg m^{-3} in trial A. However, the difference was accentuated by including different sets of rings in the samples, as basic density increases from pith to bark. A simple way to consider ring differences is to take the average of the ring numbers included in the sample and then compare trials with similar ring average. For example, a sample including rings 1–5 has an average of three while a sample including rings 6–10 has an average of eight. Note that these are linear averages derived from increment cores, not the true cross-sectional area-weighted averages; i.e. the averages are biased toward lower values.

When considering the ten trials with average ring 8, basic density ranged from 329.7 to 459.1 kg m^{-3} (a range of 129.4 kg m^{-3}). In trials with average ring 3–4 the range was smaller from 302.7 to 358.4 kg m^{-3} (a difference of 55.7 kg m^{-3}). Phenotypic variability was also related to ring average (or age), with the coefficient of variation ranging between 5.3% and 7.2% for trials with average ring 3–4 while ranging between 6.4% and 8.1% for older samples.

Genetic parameter estimates and connectedness between trials are summarized in Table 3. In the lower triangle, the table displays the number of common parents across pairs of trials, considering both female and male parents, as well as controls. Any pair with less than five parents in common was either linked only by controls or only through the pedigree more than one generation ago (i.e. by grandparents). An example of the latter is the relationship between trials P, Q and R (which correspond to the so-called 268 series) and trial L (which contains parents that are progeny

Table 3 Summary of connectedness and estimates of genetic parameters for the prediction of breeding values for basic density

Trials	Parents	Rings	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	P	Q	R
A	64	6–10	**0.45** (0.10)	–	–	–	–	–	–	–	–	–	–	–	–	–	0.70 (0.11)	–	–	–
B	21	6–10	0	**0.28** (0.13)	–	–	–	–	–	–	–	–	–	–	–	–	*0.82* (0.64)	–	–	–
C	194	1–5	0	0	**0.69** (0.07)	0.96 (0.03)	–	–	–	–	–	0.46 (0.20)	–	0.87 (0.24)	0.79 (0.26)	0.74 (0.21)	–	0.99 (0.10)	–	–
D	170	1–8	0	0	170	**0.64** (0.06)	–	–	–	–	–	0.82 (0.12)	–	*0.01* (0.46)	0.98 (0.10)	0.91 (0.14)	–	0.97 (0.09)	0.96 (0.11)	–
E	47	1–7	0	0	12	9	**0.58** (0.13)	0.95 (0.05)	–	–	–	0.99 (0.11)	–	*−0.16* (0.47)	0.86 (0.17)	0.93 (0.43)	–	*0.51* (0.38)	–	0.74 (0.25)
F	43	1–7	0	0	11	8	43	**0.47** (0.12)	–	–	–	–	–	0.66 0.43	*0.53* (0.29)	–	–	*0.46* (0.42)	–	*0.57* (0.34)
G	56	6–10	0	0	0	0	0	0	**0.42** (0.11)	0.68 (0.16)	0.73 (0.20)	–	–	–	–	–	–	–	–	–
H	57	6–10	0	0	0	0	0	0	38	**0.71** (0.16)	0.91 (0.13)	–	–	–	–	–	–	–	–	–
I	26	1–10	0	0	0	0	0	0	20	25	**0.91** (0.27)	–	–	–	–	–	–	–	–	–
J	88	1–5	0	0	28	21	5	4	0	0	0	**0.94** (0.09)	–	*0.55* (0.50)	–	0.81 (0.25)	–	0.55 (0.23)	0.87 (0.17)	*0.46* (0.25)
K	33	6–10	0	0	0	0	0	0	0	0	0	0	**0.30** (0.12)	–	–	–	–	–	–	–
L	54	6–10	0	0	13	9	8	6	0	0	0	2	0	**0.41** 0.12	*0.68* (0.48)	*0.38* (0.53)	–	*0.02* (0.50)	*0.82* (0.48)	*−0.03* (0.45)
M	152	6–10	0	0	27	23	29	27	0	0	0	12	0	0	**0.47** (0.08)	–	–	0.67 (0.14)	0.89 (0.15)	0.66 (0.13)
N	148	6–10	0	0	27	23	29	27	0	0	0	12	0	0	145	**0.48** (0.08)	–	0.81 (0.34)	0.69 (0.21)	0.74 (0.35)
O	85	6–10	63	4	0	0	0	0	0	0	0	0	0	0	0	0	**0.60** (0.11)	–	–	–
P	122	1–5	0	0	20	18	13	11	0	0	0	9	0	0	49	49	0	**0.69** (0.09)	0.71 (0.13)	0.94 (0.03)
Q	51	6–10	0	0	23	20	11	10	0	0	0	9	0	0	37	38	0	42	**0.81** (0.20)	0.65 (0.14)
R	121	1–5	0	0	20	18	13	11	0	0	0	9	0	0	49	49	0	120	42	**0.86** (0.10)

Number of parents in each trial (Parents), number of common parents across trials (below diagonal), heritability (and its standard error) in the bold diagonal, and genetic correlations between sites (and their standard errors) above diagonal. Correlations in italics are non-significant ($p > 0.05$)

of the 268 series). Most pairs of trials show low levels of connectedness, which will influence the ability to estimate GxE interaction across the breeding program.

The diagonal of Table 3 shows in boldface estimates of individual-site heritabilities (and their standard errors), which ranged from 0.28 (0.13) to 0.94 (0.09), with most values falling between moderate and high. The additive variances (and heritabilities) were significantly different from zero for all trials.

All possible pairs of sites were then analyzed as bivariate tree models, where density in each site was considered as a different trait. Out of the 153 pairs of trials, only 47 pairs contained enough information to estimate the genetic correlation between them. These correlation estimates (and their standard errors) are displayed in the upper triangle of Table 3. Out of those, 15 estimates for the correlation between traits were not statistically significant.

Some of the between-site genetic correlations include an element of age–age correlation, because they cover different sets of rings (e.g. 1–5 and 6–10). However, the age–age correlations between these sets are expected to be high (see, for example, Apiolaza and Garrick 2001; Bouffier et al. 2008).

As expected, Fig. 2 shows a positive association between number of parents in common across trials and the magnitude of the standard error of the estimated correlation. In addition, the estimated genetic correlation tends to drop when fewer parents can be used.

Figure 3 displays the relationship between the average basic density for a trial and its average annual temperature. There is an increase of basic density for warmer sites; however, there is also a large difference between average rings. That is, samples closer to the pith (triangles) have lower average densities than samples farther away from the pith (circles). There are two trials that show much lower values than expected: H and P, for which it is still needed to find a satisfactory explanation.

The coefficients for the regression lines (and their standard errors) were 387.60 (7.46) for the intercept, 18.36 (4.19) for the slope of temperature and −48.01 (11.22) for the low ring class effect. All these coefficients were statistically significant ($p < 0.01$), with the regression lines for different ring classes showing a different intercept ($387.60 - 48.01 = 339.59$) but sharing a common slope. This model explained 75% of

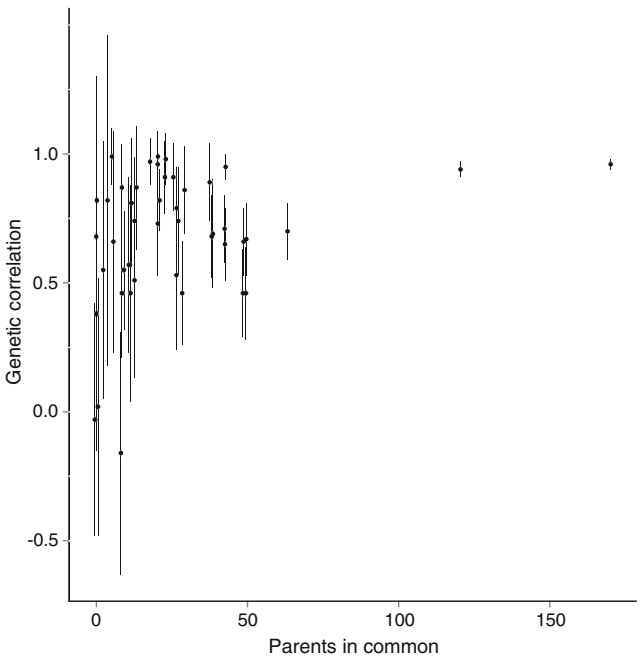

Fig. 2 The magnitude of the estimated genetic correlations between trials and their standard errors (*vertical lines*) are associated to the number of parents in common across trials. The magnitude of GxE, estimated as departure from genetic correlation of +1, can be confounded with the genetic connectedness between trials

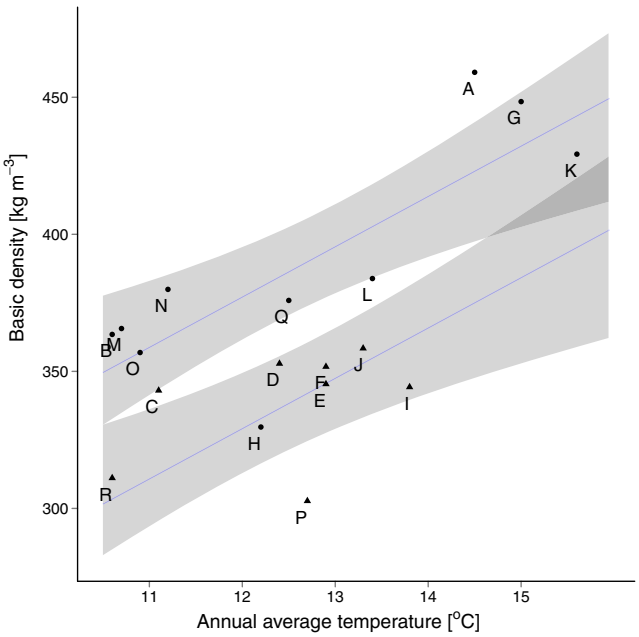

Fig. 3 Relationship between average basic density and average annual temperature for a trial. *Letters* correspond to trial codes in Table 1 and shapes to average ring number sampled in a trial, considering three (*triangles*, rings 1–5) and eight (*circles*, rings 6–10). Notice the increase of basic density with average temperature. *Gray shades* represent the 95% confidence interval for the linear regression lines

the observed variability. Adding annual rainfall did not significantly improve model fit.

Discussion

Variability and genetic control

The high degree of additive genetic control (average $\hat{h}^2 = 0.6$) in these trials supports previous results obtained by Nicholls et al. (1980), Kumar (2004) and Wielinga et al. (2009). In contrast, other researchers have reported lower values of heritability for this species (Zamudio et al. 2002; Li and Wu 2005; Dungey et al. 2006). It is not possible with this data set to disentangle the sources of variability for the estimated heritabilities. The trials represent different environments, genetic backgrounds, sample sizes, ages of assessment and overall quality of site preparation. There was a clear reduction of the standard error of the heritability estimates with sample size (see Table 3), with values stabilizing beyond 1,700 samples per trial. There was no trend between number of samples and the magnitude of estimated heritabilities.

It is easy to see how a readily assessable and highly heritable trait like basic density became the most commonly studied wood trait in breeding programs. It is important to remember, however, that with coefficients of phenotypic variation close to 8% the within-site variability is very small. The narrow genetic variability is dwarfed by environmental differences due to site factors (e.g. mean annual temperature), presenting one of the largest limitations to the operational improvement of basic density. On the other hand, from a purely deployment perspective, forest companies will make their biggest gains from careful site selection.

Estimation of GxE interaction

In forestry, the use of ANOVA with a site × Genetics (usually family or clones) interaction term was traditionally the most commonly used method (e.g. Burdon 1977). The main issues of this approach are that it assumes homogeneous additive variance and identical correlation between all pairs of trials (thus the covariance matrix follows compound symmetry), and it is difficult to frame when using animal model BLUP, although it is relatively simple when using a sire model.

Nevertheless, there are numerous alternative methodologies to study GxE interaction; for example, Freeman (1973), Westcott (1986) and Cooper and DeLacy (1994) presented broad, although by no means exhaustive, reviews. Treating each site as a different trait was suggested by Falconer in 1952 and many recent approaches use multivariate evaluation, assuming that performance in one site is a different, although related, trait to performance in a second site. Fitting this model becomes more difficult with increasing number of sites due to overparameterization and connectedness issues. While there is not much one can do about the latter (except to run pairs of bivariate analyses) the former can be tackled through the use of a factor analytic structure (e.g., Costa e Silva et al. 2006).

Shelbourne (1972) proposed an approximate threshold of 0.7 for genetic correlation to evaluate the practical importance of GxE interaction (when the GxE variance is half the size of the additive variance using an ANOVA approach). Only three of the significant correlations are below that threshold (0.46, 0.65 and 0.68), which suggests that GxE interaction for basic density should not be a major issue in the New Zealand breeding program. Furthermore, the reported genetic correlations (and their corresponding changes of ranking) are an average for the *breeding* population. Focusing on the *deployment* population, with material that on average present better performance, could reduce the importance of GxE interaction for basic density even more.

Connectedness

This paper highlights the problems created by poor connectedness among trials. In general, any pair of trials with less than 20 parents in common (either directly or via previous generations in the pedigree) had convergence problems in obtaining estimates of genetic correlation. This should be a concern for breeders designing breeding programs, particularly when inducing lower degrees of connectedness as a side-effect of rolling front strategies (see, for example, Borralho and Dutkowski 1998). This result would be further exacerbated by using small trials (e.g. Li et al. 2007).

Simulation work has suggested that as few as four to six families in common would be enough to connect trials for analyses (Johnson 2004). However, this ignores both the biases and large standard errors surrounding the estimation of genetic correlations with too small a sample size (Apiolaza et al. 1999). This problem becomes even more acute when dealing with low-heritability traits (e.g. growth traits).

In addition, the process to generate the composition of paired trials is not necessarily random, with some parents represented in much higher proportions (due to seed availability, survival, unbalanced mating designs, etc.) than others. While in theory BLUP takes

into account selection information to produce unbiased predictions, this assumes *known* genetic parameters (Henderson 1975). In contrast, tree breeding programs are using these trials to *estimate* covariance components, which are in turn used to estimate the magnitude of GxE interaction.

Figure 2 presented a relationship between the estimated genetic correlations and the number of parents in common, where poorly connected trials could underestimate the correlation. In a simulation study Sae-Lim et al. (2010) reported that small sample sizes, exacerbated by particular population structures, were more prone to produce downwardly biased estimates of between-site genetic correlations. In addition, the trials come from different selection series (there are first-, second-generation and Guadalupe hybrids), which would make possible that part of that bias comes from selection effects. If this trend is correct, this would suggest that GxE interaction for basic density could be completely ignored in the New Zealand radiata pine breeding program, as the lowest correlations would be due in part to poor connectedness. To address the estimation problem, testing and sampling procedures will have to be modified, increasing the number of related genetic material in common across trials. The over-reliance on control seedlots of unknown or unclear genetic composition—often problematic in trial analyses—to connect trials is another problem that must be addressed in a breeding program.

Environmental drivers

Cown et al. (1991, page 19) presented clear latitudinal trends for wood basic density in New Zealand, with decreasing average from North to South while Fig. 3 combines latitudinal and altitudinal effects by using temperature. The results from the New Zealand radiata pine breeding program progeny trials support the trends suggested by Harris (1965) and Cown et al. (1991), with some differences explained by the different numbers of rings sampled in each study.

Still one needs to be cautious before claiming the presence of a simple story for environmental drivers. There seems to be a positive association between number of parents in common and genetic correlation; that is, poor correlation (and therefore claims of high interaction) could derive from poor testing practices.

Warmer sites tended to have higher average density. There is still within-trial genetic variability, although lower than 8%, which means that low-density sites could still benefit from using improved material.

Using trial coordinates it was possible to obtain estimates for altitude, temperature, rainfall, wind and radiation. In principle, it would be possible to look for environmental variables that would separate groups of trials with high within-group and low between-group genetic correlations. Nevertheless, the poor connectedness between trials meant that:

1. The estimated correlations involved different sets of parents, and
2. The highly variable number of parents in common between trials (previously discussed) made any conclusions difficult to sustain.

Conclusions

- The degree of genetic control for radiata pine wood density in New Zealand ranges between moderate and high values (mean $\hat{h}^2 = 0.60$). However, the coefficient of variation for this trait is low (less than 8%), limiting the opportunities for increasing basic density.
- Treating the expression in each site as a different trait permitted us to explore the presence of structure in the genetic correlation matrix. However, the gaps in connectedness did not permit fitting more meaningful correlation structures (e.g. factor analytic).
- There was little genotype-by-environment interaction for basic density for radiata pine in New Zealand. Therefore, there would be no need to modify the breeding strategy to account for GxE for this one trait.
- Poor connectedness between trials could be depressing the estimates of genetic correlations. This situation should be considered when designing genetic testing schemes, particularly when inducing imbalance as in rolling front strategies.
- Site differences marked by annual average temperature dominate variability in this dataset. Lower latitude and altitude—that is warmer—sites display higher average basic density. This situation is clearer once age effects of the cores is taken into account.

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