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Effect of additive, dominant and epistatic variances on breeding and deployment strategy in Norway spruce

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Genetic variances are important parameters and have a great impact on the determination of optimal breeding strategies of tree species. A large clonal testing program was conducted to estimate additive, dominant and epistatic variances for the development of breeding and deployment strategies in Norway spruce (*Picea abies* (L.) Karst.). The analysis results of genetic variation for growth and wood properties in two clonal trials in central Sweden indicated that the important sources of total genetic variation were both additive and non-additive genetic variances. Additive genetic variation accounted for the majority of total genetic variation for diameter at breast height (DBH) and wood quality traits, whereas non-additive genetic variation was significant only for tree height at an early age. Predicted genetic gain was the highest for clonal deployment based on best tested (replicated) clones (4.7–65.3 per cent), followed by clonal deployment of the best individual trees from a full-sib family trial (3.5–57.7 per cent), and the deployment of seedlings generated by open-pollination (1.9–48.3 per cent).

Introduction

Genetic variance is the variance associated with the genetic differences among trees in a population and is usually partitioned into additive, dominance and epistatic components (Falconer and Mackay, 1996). Additive effects of genes are cumulative over generations and are the main source of genetic variation utilized by most plant breeding programs. However, the interactions between alleles at the same locus (dominance) and, particularly, the interactions of alleles between loci (epistasis) could also play a central role in heterosis, polymorphism and evolution (Yu *et al*., 1997) and can be exploited in an alternative deployment program (Berlin *et al*., 2019). The levels of the additive and non-additive genetic variance in traits important for breeding programs have a great impact on the determination of optimal breeding strategies (White *et al*., 2007). Ignorance of epistasis is particularly common in forest tree breeding because of substantial limitations in the statistical power and experimental methods required to partition the non-additive variance into its components (Foster and Shaw, 1988). Ignorance of non-additive genetic variance might cause bias predictions of breeding values, variance components and genetic parameters estimated (Costa e Silva *et al*., 2004; Baltunis *et al*., 2013). Well-designed genetic trials with full-sib family structure and clonal identification are therefore required to estimate all three variance components and associated heritabilities in the narrow-sense (only additive genetic variance) and in broad-sense (all genetic variance) (Wu, 2018). In addition, number of families and number of clones within a family tested are also essential for accurate estimation (Chen *et al*., 2020). The relative importance of additive and non-additive genetic effects is required information to properly evaluate the potential for genetic gain from various breeding programs and deployment options used in the genetic improvement of forest trees (Costa e Silva *et al*., 2004). However, most studies have mainly focused on additive genetic effects in forest tree breeding programs.

Estimates of genetic gain from clonally replicated trials in conifers were initially started in 1980s (Wu, 2018). But only few reliable estimates for the non-additive variation were reported (Mullin and Park, 1992; Isik *et al*., 2003, 2005; Baltunis *et al*., 2007, 2009, 2013; Weng *et al*., 2008) in which growth traits were the focus rather than wood quality traits (Costa *et al*., 2004; Wu *et al*., 2008; Chen *et al*., 2020). When non-additive genetic variance is significant, total genetic variance and broad-sense heritability are expected to be higher than the corresponding additive genetic variance and narrow-sense heritability

(Wu, 2018). Given that non-additive genetic variation can be exploited in deployment, this could result in larger genetic gain from selection (Mullin and Park, 1992; Chen *et al*., 2020).

Norway spruce (*Picea abies* (L.) Karst.) is one of the most important conifer species in Europe, both from economic and ecological points of view (Högberg *et al*., 2014). The versatility of the wood and its large geographic distribution make it a widely used tree species in the European forest industry (Steffenrem *et al*., 2016). In Sweden, genetic improvement has been carried out on this species for about 60 years and has mainly focused on improving growth and adaptive traits, including tree height, diameter, branch quality, survival and frost damage (Karlsson and Rosvall, 1993) and a large clonal testing program was launched, including more than 18 400 clones tested from the mid-1970s. Benefits from using clones are considerable through intense within-family selection and capturing of non-additive genetic effects (Rosvall *et al*., 2019). A previous study in Norway spruce indicated that selection based on clonal means plus vegetative deployment would be the most effective strategy (Chen *et al*., 2020). In a recent review of deploying clonal forestry of Norway spruce, there was a special call to estimate non-additive genetic variance for growth and wood quality traits in order to assess the benefits and risks of Norway spruce clonal forestry (Wu, 2018; Rosvall *et al*., 2019). Thus, non-additive genetic effects were analysed for height and DBH; however, the scale and importance of dominance and epistatic effects could not be clearly distinguished (Berlin *et al*., 2019) whereas additive and non-additive genetic variances were equally necessary for growth traits (Chen *et al*., 2020). Wood quality traits have recently been incorporated into selective breeding programs (Högberg *et al*., 2014; Chen *et al*., 2016; Wu, 2018). Knowledge of wood quality and genetic correlations of wood quality traits with each other and with growth rate is limited (Steffenrem *et al*., 2009). The negative genetic correlation (adverse relationship) between wood quantity and quality was found (Hannrup *et al*., 2004; Kroon *et al*., 2011; Högberg *et al*., 2014; Chen *et al*., 2015). Breeding to overcome adverse genetic correlations and to effectively utilize non-additive genetic variation is two of the most challenging issues in Norway spruce tree improvement and deployment programmes.

This study, therefore, aims to (1) assess the relative importance of additive, dominance and epistatic genetic effects for growth and wood quality traits; (2) estimate heritabilities and genetic correlations for these traits; (3) furthermore, this study aims to predict genetic gain from clonal deployment of the top of tested and replicated clones (CRC) compared with clonal deployment of the top individual trees (CIN) taken e.g. from a full-sib family trial, and deployment of seedlings only, generated from the open-pollination of the best individuals (SOP) by establishing e.g. a seed orchard. To achieve these aims, two clonal full-sib family progeny tests of Norway spruce in central Sweden were studied.

Materials and methods

Plant material and field trials

Two clonal full-sib family progeny trials of Norway spruce, S209 at Lugnet and S241 at Rådahöjden, were established in 1991 in central Sweden (see Table 1). The two trials comprised 1015 clones representing 167 full-sib families from crossing combinations between 58 parents including 36 acting as females and 37 acting as males. Each family from these two trials had an average of 6.2 clones (1–16 clones) and each clone was represented by 1–10 ramets (average 3.2 ramets). Growth traits were measured at tree ages 6 and 12 for height (Ht_6, Ht_{12}) (m), and 12 and 26 for diameter at breast height (DBH_{12}, DBH_{26}) (cm). A randomized incomplete block design using the single-tree plot was used in the two clonal trials.

Table 1 Details of the two field trials (Lugnet and Rådahöjden).

Details	Lugnet	Rådahöjden
Latitude	59.38°N	60.00°N
Longitude	17.31°N	13.31°N
Altitude	10 m	210 m
Mean annual rainfall	592 mm	849 mm
Mean annual temperature	7°C	5°C
Soil type	Sedimentary clay	Sandy till
Number of measured trees	1215	2021
Number of clones	548	555
Number of families	74 (167*)	165 (167*)
Average number of ramets/clone	2.2	3.6

*The value in parenthesis is the total number of families used in two trials.

Non-destructive testing tools used to assess wood density on standing trees were Pilodyn 6 J Forest (Proceq, Schwerzenbach, Switzerland) and Micro-drill Resistograph IML-RESI PD300 (Instrumenta Mechanic Labor, Germany). Pilodyn 6J Forest was used to measure pilodyn penetration depth (Pilo). When measuring, bark was not removed. The measurement was conducted at approximate 1.3 m above the ground and on the same side for all trees. Pilodyn penetration has repeatedly been observed to show high magnitudes of correlation with wood density in Norway spruce (Högberg *et al*., 2014; Chen *et al*., 2015). Besides, the resistograph (Resi) was used for drilling trees bark to bark at a given feed speed in one mutually perpendicular direction at height of ca 1.3 m. The resistograph's resistance traces were then transferred to the PD Tools Pro program and exported as text files. Custom software available as a web URL https://forestquality.shinyapps.io/ResiProcessor/ was used to process the resistance traces and extract the over-bark and under-bark diameter, together with the mean resistance value of the under-bark portion of the trace (Downes *et al*., 2018). A measure of bark thickness (BkTh*)* was also provided from the resistograph data profile (Figure 1).

Also, the wood stiffness of standing trees was indirectly assessed by first determining the acoustic velocity (AV, ms^{-1}) measured using the Hitman ST300 tool (Fibergen, Christchurch, New Zealand). Two probes were inserted into a tree stem, separated vertically at a distance of 0.7–1.3 m and orientated at an angle of 450 to the stem with the tips facing each other. The acoustic velocity was calculated:

$$AV = \frac{s}{t} \quad (1)$$

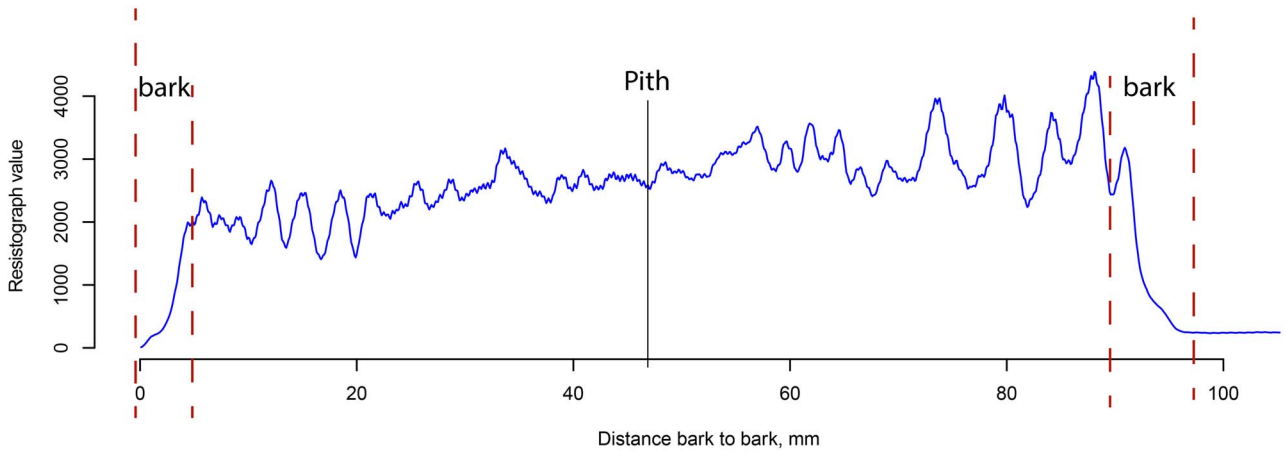

Figure 1 The profile of resistograph measurement (a proxy of wood density) from bark to bark. The distances between two red dash lines on the two sides are the bark thickness; the average of both sides' bark thickness is used as bark thickness for the tree.

where the distance between probes is *s* and the transit time is *t*. In the second step, the AV measurement was then combined with Pilo measurements so that the modulus of elasticity (MOE) could be determined as the indirect measure of wood stiffness by using the following equation (Chen *et al.*, 2015):

$$\text{MOE} = \frac{1}{\text{Pilo}}\ 10\ 000\ \text{AV}^2 \tag{2}$$

Finally, the spiral grain angle (GA) was measured for each tree at 1.3 m height using the wedge grain angle gauge (Hannrup *et al.*, 2004; Fries *et al.*, 2014). Measurements from two opposite sides (northwest and southeast) of the stem were taken and the mean value was used as the phenotypic GA value thus eliminating measurement errors due to leaning stems.

Statistical analysis

Statistical analysis

Cross-site analyses were conducted for traits measured in both trials and single site analyses were conducted for those traits only measured in one trial (Table 1) in ASReml 4.0 (Gilmour *et al.*, 2014). The analysis of variance components for across site was undertaken according to the following three general mixed linear models:

$$y = X\beta + Z_a a + Z_{as} as + b(s) + e \tag{3}$$

$$y = X\beta + Z_a a + Z_{as} as + Z_f f + Z_{fs} fs + Z_{b(s)} b(s) + e \tag{4}$$

$$y = X\beta + Z_a a + Z_{as} as + Z_f f + Z_{fs} fs + Z_c c + Z_{cs} cs + Z_{b(s)} b(s) + e \tag{5}$$

where y is the vector of observations for traits, β is the vector of fixed effects (i.e. mean and site), a is the vector of random additive genetic effects, as is the vector of random additive genetic effects by site interaction effects, f is the vector of random effects of full-sib families (specific combining ability, SCA), fs is the vector of random effects of SCA by site interaction, c is the vector of random effects of clones within full-sib families, cs is the vector of random effects of clones within full-sib family by site interaction and $b(s)$ is the vector of random effects of the block within site. X, Za, Z_{as}, Z_f, Z_{fs}, Z_c, Z_{cs} and $Z_{b(s)}$ are the known incidence matrices relating to the observations in y to effects in b, a, as, f, fs, c, cs and $b(s)$, respectively. The variances associated with the random effects a, as, f, fs, c and cs are referred to as $A\sigma_a^2$, $\sigma_{as}^2 I_s \otimes A$, $\sigma_f^2 I_f$, $\sigma_{fs}^2 I_{fs}$, $\sigma_c^2 I_c$ and $\sigma_{cs}^2 I_{cs}$, respectively. The random effects of $b(s)$ have a heterogeneity of variance structure ($\sigma_{b1}^2 I_{b1} \bigoplus \sigma_{b2}^2 I_{b2}$), where σ_{b1}^2 and σ_{b2}^2 are block variances for sites 1 and 2, respectively. e is the vector of random residual terms ~N(0, R), with a heterogeneity of variance structure between two sites ($R = \sigma_{e1}^2 I_1 \bigoplus \sigma_{e2}^2 I_2$), where $\bigoplus$ is the direct sum and σ_{e1}^2 and σ_{e2}^2 are the residual variances for sites 1 and 2, respectively. σ^{2e} is the average of the two-residual variances. $\boldsymbol{0}$ is the null matrix. If a trait was only measured in one site, then the random effect by site interaction term will be dropped and the block within site term will be replaced by block effect. Similar to the univariate model (5), a bivariate model was used to estimate variance and covariance components. However, due to the complication of model fitting, the variances of the block within site and residual effects for each site are homogeneous. In the bivariate model, unstructured (US) covariance structures were used to fit for the a, as, f, fs, c and cs and residual effects. Diagnal (DIAG) covariance structure was used to fit the block within site effects. If any of the variance components are in boundary, then the terms will be dropped in a specific bivariate model.

According to assumptions defined by Costa e Silva *et al.* (2004), the additive, dominance, epistatic, total genetic, environmental (residual) and phenotypic variances were estimated as follows:

$$\hat{V}_A = \hat{\sigma}_a^2 \tag{6}$$

$$\hat{V}_D = 4\hat{\sigma}_f^2 \tag{7}$$

$$\hat{V}_I = \hat{\sigma}_c^2 - 3\hat{\sigma}_f^2 \tag{8}$$

$$\hat{V}_G = \hat{\sigma}_a^2 + \hat{\sigma}_f^2 + \hat{\sigma}_c^2 \quad (9)$$

$$\hat{V}_E = \left(\hat{\sigma}_{e1}^2 + \hat{\sigma}_{e2}^2\right) * 0.5 \quad (10)$$

$$\hat{V}_P = \hat{\sigma}_a^2 + \hat{\sigma}_{as}^2 + \hat{\sigma}_f^2 + \hat{\sigma}_{fs}^2 + \hat{\sigma}_c^2 + \hat{\sigma}_{cs}^2 + \hat{V}_E \quad (11)$$

Proportions of dominance ($\hat{d}^2$) and epistatic ($\hat{i}^2$) variances, individual narrow-sense ($\hat{h}^2$), broad-sense ($\hat{H}^2$) and clonal mean ($\hat{H}^2_{\bar{c}}$) heritabilities were estimated according to (Costa e Silva *et al*., 2004; Baltunis *et al*., 2009):

$$\hat{d}^2 = \frac{\hat{V}_D}{\hat{V}_P} \quad (12)$$

$$\hat{i}^2 = \frac{\hat{V}_I}{\hat{V}_P} \quad (13)$$

$$\hat{h}^2 = \frac{\hat{V}_A}{\hat{V}_P} \quad (14)$$

$$\hat{H}^2 = \frac{\hat{V}_G}{\hat{V}_P} \quad (15)$$

$$\hat{H}^2_{\bar{c}} = \frac{\hat{V}_G}{\hat{V}_{\bar{c}}} = \frac{\hat{V}_G}{\hat{V}_G + \frac{\hat{V}_E}{r}} \quad (16)$$

where $\hat{V}_{\bar{c}}$ is the variance of clonal means and r is the harmonic mean number of ramets per clone.

Total genetic ($\hat{r}_G$) and additive genetic ($\hat{r}_A$) correlations between traits were calculated from variances and covariances using the mixed model equation, and the following formula were used for calculation (Isik *et al*., 2017):

$$\hat{r}_G = \frac{\hat{Cov}_G(x,y)}{\left[\hat{V}_G(x) \cdot \hat{V}_G(y)\right]^{1/2}} \quad (17)$$

$$\hat{r}_A = \frac{\hat{Cov}_A(x,y)}{\left[\hat{V}_A(x).\hat{V}_A(y)\right]^{1/2}} \quad (18)$$

where $\hat{Cov}_G(x,y)$ and $\hat{Cov}_A(x,y)$ are the total genetic covariance component and additive genetic covariance between traits x and y.

Expected genetic gains (ΔG) were estimated for a number of deployment options based on various forward selection schemes under selection intensities ranging from 1 to 20 per cent ($i = 2.665$–1.400). The deployment strategies considered were (1) deployment of seedlings generated by the open-pollination of the best individual trees (assuming no pollen contamination), which is based on only the additive portion of the genetic variance (SOP), (2) clonal deployment of the best individual trees from a full-sib family field trial with cutting propagation without replicated clonal testing (CIN) and (3) clonal deployment of the best clones by replicated clonal testing with cutting propagation (CRC). Genetic gains were calculated as follows:

$$\Delta G_{h^2} = i\hat{h}^2\sqrt{\hat{V}_P} \quad (19)$$

$$\Delta G_{H^2} = i\hat{H}^2\sqrt{\hat{V}_P} \quad (20)$$

$$\Delta G_{H^2_{\bar{c}}} = i\hat{H}^2_{\bar{c}}\sqrt{\hat{V}_{\bar{c}}} \quad (21)$$

All gains were expressed as the percentage gain over the mean of the trait:

$$\%\Delta G = \frac{\Delta G}{\bar{y}_i} \cdot 100 \quad (22)$$

where $\bar{y}_i$ is the population mean for trait i.

Delta method was used to estimate standard error for all genetic parameters by ASReml standard alone version v4.1, except standard error of genetic gain that was estimated by ASReml R v4.1 since, in the current version, ASReml standard alone is not allowed to estimate standard error for a complicated genetic parameter, such as genetic gain.

Results

Summary of traits measured in two trials

In total, 1016 genotypes with an average of 3.2 ramets were measured in two trials. Eighty-eight genotypes within seventy-two families are overlapped between the two sites Lugnet and Rådahöjden. The basic summary of the measured traits is shown in Table 2. The mean values of growth traits in Rådahöjden were higher than those in Lugnet. For example, the mean value of Ht_6 was 233.5 cm in Rådahöjden, which is higher than 125.7 cm in Lugnet. The coefficient of variation (CV) varied from 5.9 per cent for AV_{26} in Lugnet to 46.0 per cent for GA_{26} in Lugnet. The CV of growth traits is higher than wood quality traits, except GA27 and MOE26. Phenotypic correlation and the distribution of each trait are shown in Supplementary Figure S1. The distribution for all traits is approximately normal, the growth traits showed slight two peaks, and thus, the heterogeneous variances for block and residuals effects were fitted in the model for those traits when we estimated genetic variances.

Comparison of variance components of different models

To detect the G × E interaction and dissect non-additive effects including dominance and epistatic effects, three models including additive only (A), additive and dominance only (AD), and additive, dominance, epistatic effects (ADE full model) were performed for all traits. The results are shown in Table 3 and Supplementary Table S1. Based on Akaike information criterion (AIC), only the full model of traits Ht_6, Ht_{12} and DBH_{12} showed the smallest AIC value. Other traits, the additive model only showed the smallest AIC value.

Partitioning of genetic variances and genetic parameters

Single site and across sites estimates of variance components and genetic parameters for wood and growth traits for the data from two clonal progeny trials are presented in Table 4. Additive genetic variation accounted for the majority of the total

Table 2 Number of trees measured (*n*), number of genotypes (*N*), minimum, maximum, mean and coefficient of variation (CV) for all traits in two Norway spruce trials (Lugnet and Rådahöjden).

	Site Lugnet					Site Rådahöjden				
Trait	*n*	*N*	Min-Max	Mean	CV (%)	*n*	*N*	Min-Max	Mean	CV (%)
Ht_6 (cm)	1214	547	37–240	125.7	26.7	2014	555	30–372	233.5	21.4
Ht_{12} (cm)	1214	547	130–700	482.9	18.6	2012	555	140–810	568.0	17.2
DBH_{12} (cm)	1214	547	7–97	54.7	24.9	2012	555	10–122	71.0	22.8
DBH_{26} (cm)	1214	547	33–239	118.0	24.3	2020	555	16–305	136.4	24.9
$Pilo_{26}$ (mm)	1204	545	12.0–26.5	19.0	13.1	1415	552	10–28	18.8	12.5
AV_{26}	1192	545	3.0–5.2	4.3	5.9	1415	552	3.1–5.3	4.3	6.8
MOE_{26} (Gpa)	1185	544	4.48–15.96	10.0	18.2	1415	552	5.1–18.0	9.9	20.0
$BkTh_{27}$ (mm)	1205	548	3.15–9.00	5.5	15.3	NA	0	NA	NA	NA
$Resi_{27}$	1203	548	1198.4–2741.1	1963.3	13.1	NA	0	NA	NA	NA
GA_{27} (∘)	1140	529	−12.5-65	24.9	46.0	NA	0	NA	NA	NA

NA represents that the trait was not measured in that trial. Note: ht6 and ht12 represent that tree height was measured at tree age 6 and 12, respectively; DBH_{12} and DBH_{26} represent diameter at breast height measured at tree ages 12 and 26, respectively; $Pilo_{26}$, AV_{26}, $BkTH_{27}$ and $Resi_{27}$ represent that Pilodyn penetration, acoustic velocity, bark thickness and resistograph, measured at tree ages 26, 26, 27 and 27, respectively. MOE_{26} represents modulus of elasticity predicted based on AV26 and Pilo26 measured at tree age 26.

genetic variation associated with Ht_6, DBH_{12}, DBH_{26} and wood traits (53.3–99.4 per cent), whereas non-additive genetic variation contributed low to moderate portion to the total genetic variation (0.6–46.7 per cent). Among them, dominance genetic variation contributed a lower or zero portion to the total genetic variation (0–32.5 per cent).

Non-additive genetic variation appeared to be more important only for Ht_{12} (61.8 per cent) in which dominance contributed considerably (69.9 per cent) and the relative importance between dominance and epistasis varied with age. For wood traits, dominance ranged from zero to 32.5 per cent while epistasis was less than 16.5 per cent. Partitioning of genetic variance indicated that both additive and non-additive genetic variances were important sources of total genetic variances for most of the traits, especially for growth traits.

For tree height, the proportion of phenotypic variance due to dominance variance showed an increasing trend (0.00–0.14) by increasing age but epistatic variance decreased (0.03 to −0.01) with age. The total non-additive genetic variance in the model was higher than the additive genetic variance for Ht_{12}whereas for Ht_6 and DBH, this relationship was the reverse. For the wood traits, the results showed zero levels of dominance for Pilo, GA and BkTh; low for Resi (10.2 per cent) and MOE (13.2 per cent); and moderate for AV (32.5 per cent). In addition, epistatic genetic variances for wood traits estimated from the data were very low and especially negative for Resi.

Overall, clonal mean heritability ($H^2_{\bar{c}}$) estimates were always the largest in magnitude as compared with broad-sense (H^2) and narrow-sense (h^2) heritabilities for studied traits. Most wood traits including Resi, Pilo, MOE and GA also showed higher heritability than growth traits. Estimates of h^2 for all traits were low to high (0.08–0.51) while H^2 ranged from 0.15 to 0.61. Clonal mean heritability estimates ($H^2_{\bar{c}}$) were however systematically higher (0.28–0.78) than h^2 and H^2 for all observed traits and their small standard errors indicated the high precision of estimates (Table 4).

Trait–trait correlations

Estimates of the total genetic and additive genetic correlations among observed traits in both single trials and across trials are presented in Table 5. Total genetic correlations among growth traits were generally comparable to additive genetic correlations but had lower standard errors. Therefore, additive and total genetic correlations are henceforth reported and discussed together as genetic correlations. Both total genetic and additive genetic correlation among tree height and DBH growth traits were very high (0.72–0.95). Genetic correlations between BkTh and growth were highly positive (0.79–1.00) but both correlations between BkTh and wood traits were close to zero (−0.19 to 0.31).

Wood density measured indirectly by Resi and Pilo showed unfavourable and moderate genetic correlations with DBH (−0.42 to −0.63 for Resi and 0.38–0.51 for Pilo). However, wood density using Resi measurements had unfavourable and moderate correlation with height (−0.30 to −0.44), but relatively low correlations between Pilo and height (0.02–0.25). The estimate of the total and additive genetic correlation between Resi and Pilo was strongly negative (both values at −0.88). Besides, MOE had low positive (large standard error) to moderate negative (unfavourable) genetic correlations with growth traits but high correlations with Resi and Pilo. However, grain angle had very weak genetic correlations with the most traits (−0.03 to 0.32) except for AV and MOE (−0.30 to −0.60).

Response for different selection schemes

Genetic gains for DBH were calculated assuming equal selection intensities ($i = 1$–20 per cent) for each deployment strategy (Figure 2). Regarding the predicted genetic gain, clonal deployment of the best tested and replicated clones (CRC) offered the greatest gains, followed by clonal deployment of the best individuals (CIN) from full-sib family, and then the open-pollinated seedling deployment (SOP). For example, when selecting the top 5 per cent of tested and replicated clones

Table 3 Variance components of three different models and their standard errors (in parenthesis) and Akaike information criterion (AIC).

Trait	Model	α_a^2	α_{as}^2	α_f^2	α_{fs}^2	α_c^2	σ_{cs}^2	AIC
Ht_6	A	441.1 (87.0)	201.9 (72.4)					27053.4
	AD	453.0 (85.6)	157.7 (73.4)	0 (NA)	31.3 (23.4)			27055.1
	ADE	197.3 (74.7)	45.1 (47.9)	0 (NA)	36.4 (24.9)	51.2 (89.8)	193.9 (88.2)	**27044.0***
Ht_{12}	A	2536.9 (386.1)	631.2 (265.2)					32027.6
	AD	2211.6 (390.0)	635.8 (268.3)	254.0 (122.7)	0 (NA)			32024.4
	ADE	617.6 (345.1)	402.9 (231.5)	279.0 (127.4)	0 (NA)	719.9 (409.0)	445.8 (405.2)	**32015.4***
DBH_{12}	A	47.0 (9.3)	18.6 (7.3)					20241.1
	AD	45.3 (9.6)	13.7 (7.9)	0.8 (3.6)	4.3 (4.1)			20241.1
	ADE	24.5 (9.7)	4.6 (6.6)	0.9 (3.8)	5.5 (4.5)	5.7 (10.2)	14.7 (10.1)	**20237.9***
DBH_{26}	A	157.6 (34.9)	72.1 (28.5)					**25045.0***
	AD	153.8 (36.2)	57.1 (31.0)	1.7 (15.0)	1.7 (15.0)			25046.7
	ADE	136.3 (38.6)	39.3 (30.2)	0.9 (14.7)	15.4 (17.2)	0 (NA)	28.3 (26.9)	25049.6
$Pilo_{26}$	A	1.7 (0.2)	0.03 (0.08)					**6529.5***
	AD	1.7 (0.2)	0 (NA)	0 (NA)	0.02 (0.05)			6533.4
	ADE	1.4 (0.4)	0 (NA)	0 (NA)	0.02 (0.05)	0.2 (0.3)	0.04 (0.2)	6536.7
AV_{26}	A	0.02 (0.003)	0.005 (0.002)	0.020 (0.07)				**-4657.2***
	AD	0.02 (0.003)	0.005 (0.002)	0.001 (0.001)	0 (NA)			-4655.6
	ADE	0.01 (0.004)	0.004 (0.002)	0.002 (0.001)	0 (NA)	0.007 (0.003)	0 (NA)	-4656.9
MOE_{26}	A	1.3 (0.1)	0.1 (0.08)					**5135.6***
	AD	1.3 (0.2)	0.1 (0.08)	0.03 (0.04)	0 (NA)			5138.9
	ADE	0.9 (0.3)	0.1 (0.07)	0.04 (0.04)	0 (NA)	0.2 (0.20)	0 (NA)	5141.9
$BkTh_{27}$	A	0.12 (0.03)						**565.1***
	AD	0.12 (0.03)		0 (NA)				567.1
	ADE	0.11 (0.04)		0 (NA)		0.02 (0.03)		568.7
$Resi_{27}$	A	24085.0 (3095.8)						**14090.0***
	AD	23493.7 (3254.0)		611.9 (1154.5)				14091.6
	ADE	23493.7 (3254.0)		611.9 (1154.5)		0 (NA)		14093.6
GA_{27}	A	88.8 (8.7)						**6269.2***
	AD	88.8 (8.7)		0 (NA)				6271.2
	ADE	66.8 (18.3)		0 (NA)		13.2 (10.7)		6272.0

Bold and * represent that the fitness of model is better than the other two models. Models A, AD and ADE represent models of equations (3) (additive only), 4 (additive + dominance) and 5 (additive + dominance + epistasis), respectively.

for clonal deployment (CRC), genetic gains were estimated to reach 9.8–10.6 per cent for DBH from ages 12 to 26 and 8.9–9.6 per cent for tree height from age six to 12 (Table 6). When clonally deploying the best 5 per cent individuals (CIN), gains at 7.0–7.4 per cent for DBH and 6.4–7.0 per cent for height could be expected. Finally, deploying the offspring of the best 5 per cent selected individuals by open pollination (SOP) would result in gains of 5.5–7.3 per cent for DBH and 2.7–5.1 per cent for height. CRC deployment thus showed a relative superiority of 40.0–43.2% per cent over CIN and 45.2–78.2% per cent over SOP for DBH at the same selection intensity. In addition, genetic gains for wood quality traits including BkTh but excluding GA reached from 4.7 to 17.8 per cent in CRC deployment, 3.5–13.8 per cent in CIN deployment and 1.9–11.2 per cent in SOP deployment. According to the results, the CRC strategy offered a relative improvement of 13.2–37.7 per cent over CIN and 22.4–147.4 per cent over SOP deployment in terms of genetic gains for wood traits.

Discussion

Partitioning of genetic variances and genetic parameters

Most tree breeding programs only use additive variation in their breeding and deployment program if selection is based on phenotypes or predicted breeding values, and propagation is based on open-pollinated seed orchards only. This is because non-additive genetic components cannot be used for genetic advance in such scenario (Isik *et al*., 2017). Phenotypic variance comprises (1) additive variance: heritable from parents to progeny; (2) dominance and epistasis variance: only heritable with the same

Table 4 Additive, dominance and epistatic genetic variances, genetic parameters and their standard errors (in parenthesis) for observed traits in Rådahöjden, Lugnet[(a)] and across two sites.

	Ht_6	Ht_{12}	DBH_{12}	DBH_{26}	$Pilo_{26}$	AV_{26}	MOE_{26}	$BkTh_{27}$[a]	$Resi_{27}$[a]	GA_{27}[a]
$\hat{V}_A$	197.3 (74.6)	617.7 (344.4)	24.5 (9.7)	136.3 (38.7)	1.4 (0.4)	0.01 (0.004)	0.95 (0.28)	0.11 (0.04)	23 494 (3255.6)	66.8 (18.3)
$\hat{V}_D$	0 (0)	1116.1 (509.3)	3.5 (15.1)	3.5 (59.1)	0 (0)	0.006 (0.004)	0.16 (0.17)	0 (0)	2447.5 (4623.3)	0 (0)
$\hat{V}_I$	51.2 (89.4)	−117.2 (551.6)	3.1 (15.3)	−2.7 (44.3)	0.2 (0.3)	0.003 (0.004)	0.06 (0.19)	0.02 (0.03)	−1835.7 (3467.5)	13.2(10.7)
$\hat{V}_G$	248.5 (86.6)	1616.6 (400)	31.1 (10)	137.2 (37.1)	1.6 (0.3)	0.019 (0.003)	1.17 (0.16)	0.12 (0.03)	24106.0 (3107.6)	80.0 (10.0)
$\hat{V}_P$	1695.4 (54.3)	7913.4 (253.9)	200.1 (6.5)	873.6 (28.5)	4.9 (0.2)	0.067 (0.003)	3.09 (0.15)	0.58 (0.03)	53410.0 (2914.2)	130.6 (10.1)
$\hat{V}_{\bar{C}}$	890 (48.5)	4167.8 (228.6)	101.0 (5.8)	424.4 (25.3)	2.7 (0.2)	0.037 (0.002)	1.85 (0.15)	0.33 (0.02)	37322.0 (2914)	102.8 (9.9)
d^2	0 (0)	0.14 (0.06)	0.02 (0.08)	0.00 (0.07)	0 (0)	0.09 (0.06)	0.05 (0.06)	0 (0)	0.05 (0.09)	0 (0)
i^2	0.03 (0.05)	−0.01 (0.07)	0.02 (0.08)	0.00 (0.05)	0.03 (0.06)	0.04 (0.06)	0.02 (0.06)	0.03 (0.05)	−0.03 (0.06)	0.10 (0.09)
h^2	0.12 (0.04)	0.08 (0.04)	0.12 (0.05)	0.16 (0.04)	0.29 (0.07)	0.15 (0.06)	0.31 (0.08)	0.18 (0.06)	0.44 (0.04)	0.51 (0.11)
H^2	0.15 (0.05)	0.20 (0.05)	0.16 (0.05)	0.16 (0.04)	0.32 (0.05)	0.28 (0.04)	0.38 (0.04)	0.21 (0.04)	0.45 (0.04)	0.61 (0.03)
$H^2_{\bar{C}}$	0.28 (0.09)	0.39 (0.09)	0.31 (0.09)	0.32 (0.08)	0.60 (0.08)	0.51 (0.06)	0.63 (0.05)	0.38 (0.06)	0.65 (0.04)	0.78 (0.03)
Partitioning of genetic components of variances (%)										
$\hat{V}_A$	79.4 (32.8)	38.2 (20.2)	78.8 (31.8)	99.4 (10.8)	89.5 (17.8)	53.3 (16.7)	81.2 (15.6)	85.6 (22.5)	97.5 (4.8)	83.5 (14.6)
$\hat{V}_D$	0.0 (0.0)	69.9 (34.1)	11.1 (48.4)	2.6 (43.0)	0.0 (0.0)	32.5 (21.5)	13.4 (14.8)	0.0 (0.0)	10.2 (19.2)	0.0 (0.0)
$\hat{V}_I$	20.6 (31.8)	−7.2 (34.9)	10.1 (48.3)	−2.0 (32.2)	10.5 (17.8)	14.2 (20.3)	5.4 (16.8)	14.4 (22.5)	−7.6 (14.4)	16.5 (14.6)

Table 5 Total genetic (above), additive genetic (below) correlations between growth and wood traits and their standard errors (in parenthesis) in Rådahöjden, Lugnet[(a)] and cross sites.

	Ht_6	Ht_{12}	DBH_{12}	DBH_{26}	$Pilo_{26}$	AV_{26}	MOE_{26}	$BkTh_{27}$[a]	$Resi_{27}$[a]	GA_{27}[a]
Ht_6		0.90 (0.03)	0.83 (0.02)	0.72 (0.04)	0.13 (0.10)	0.28 (0.13)	0.04 (0.11)	0.79 (0.12)	−0.34 (0.14)	0.01 (0.12)
Ht_{12}	0.95 (0.07)		0.85 (0.06)	0.80 (0.08)	0.25 (0.13)	0.23 (0.11)	0.06 (0.10)	1.00 (0.12)	−0.30 (0.14)	−0.03 (0.10)
DBH_{12}	0.85 (0.07)	0.83 (0.09)		0.92 (0.04)	0.38 (0.11)	0.09 (0.13)	−0.11 (0.11)	0.87 (0.11)	−0.63 (0.12)	0.09 (0.12)
DBH_{26}	0.78 (0.09)	0.80 (0.11)	0.92 (0.04)		0.51 (0.08)	−0.10 (0.13)	−0.35 (0.09)	0.86 (0.09)	−0.42 (0.11)	0.14 (0.13)
$Pilo_{26}$	0.02 (0.29)	0.25 (0.13)	0.38 (0.11)	0.51 (0.08)		−0.44 (0.09)	−0.94 (0.03)	0.31 (0.12)	−0.88 (0.05)	0.19 (0.09)
AV_{26}	0.46 (0.39)	0.32 (0.36)	0.04 (0.32)	−0.11 (0.16)	−0.63 (0.16)		0.81 (0.04)	−0.11 (0.13)	0.37 (0.14)	−0.33 (0.09)
MOE_{26}	0.09 (0.30)	0.11 (0.31)	−0.15 (0.26)	−0.39 (0.11)	−0.94 (0.03)	0.86 (0.06)		−0.19 (0.12)	0.71 (0.07)	−0.30 (0.08)
$BkTh_{27}$[a]	0.81 (0.11)	1.00 (0.12)	0.87 (0.11)	0.86 (0.09)	0.31 (0.12)	−0.02 (0.25)	−0.08 (0.21)		0.10 (0.13)	0.06 (0.11)
$Resi_{27}$[a]	−0.34 (0.14)	−0.44 (0.18)	−0.56 (0.1)	−0.43 (0.12)	−0.88 (0.05)	0.37 (0.14)	0.74 (0.08)	0.10 (0.13)		−0.03 (0.09)
GA_{27}[a]	0.12 (0.23)	0.03 (0.24)	0.16 (0.22)	0.14 (0.13)	0.32 (0.17)	−0.60 (0.17)	−0.52 (0.15)	0.15 (0.21)	−0.03 (0.09)	

control pollinated families or the same genotypes used and (3) non-heritable environmental variation. The separation of the genetic variance into its additive, dominance and epistatic components can only be realized in clonal trials with family structure or when various inbred materials are used (Costa e Silva *et al*., 2004; Wu 1996). The genetic model using clones assumes that epistasis reflects primarily interactions involving groups of more than two or three loci (Mullin and Park, 1992) and it was assumed that such interactions would capture the most of the total interaction variance (Costa e Silva *et al*., 2004; Wu 1996). Recently, genomic relationship matrices for additive, dominance and epistatic effects were calculated to estimate the additive, dominance and epistatic variances using marker data such as exome capture and SNPs array (Gamal El-Dien *et al*., 2016; Tan *et al*., 2018; Chen *et al*., 2019). However, a more common and traditional method to estimate all genetic variances are clonal field trials with full-sib family structure.

In this study, two large clonal trials with full-sib family structures were used to partition the total genetic variance into additive, dominance and epistatic variances. The large proportion of the total genetic variance for growth trait DBH and wood traits was explained by additive genetic variance while non-additive genetic variance appeared to be more important for tree height. Epistatic variance estimates was 20.6 per cent

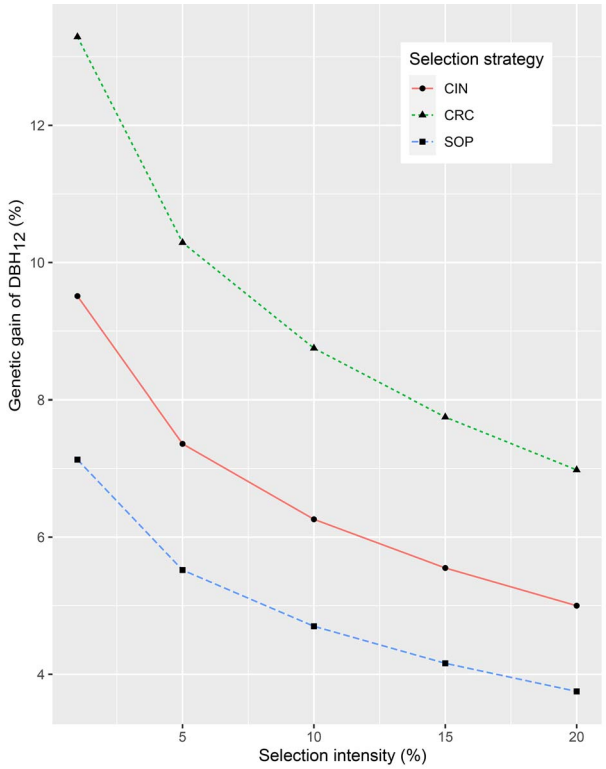

Figure 2 Predicted genetic gain of DBH_{12} (current measurement age for early selection in Norway spruce breeding) from three deployment scenarios (SOP-seedling deployment from open-pollinated progeny, CIN-clonal deployment of the best individual trees from a full-sib family and CRC-clonal deployment from the best clones tested).

of the total genetic variation for tree height at tree age six. These estimates were close to the recent report on the same species (46.5–55.2 per cent) (Chen *et al*., 2020). However, the previous report of Chen *et al*. (2019) did not show any first-order epistatic variance for tree height using genomic-based relationship matrices to estimate epistatic effects in Norway spruce. This may indicate that the epistatic effect of tree height in Norway spruce may be from high-order multi-locus interaction (i.e. among QTLs ≥ 3), but this needs more markers and large population size for further verification. In addition, estimates of dominance variances for growth were close to previous studies on other conifer species, such as black spruce (Mullin and Park, 1992, 1994), white spruce (Weng *et al*., 2008) and hardwood species such as *Eucalyptus globulus* (Costa e Silva *et al*., 2004). In contrast, Lenz *et al*. (2020) did not observe significant dominance effect for growth and wood quality traits in two Norway spruce full-sib family trials. In this study, non-additive components were important or more important for height and DBH at early ages ($\leq$12-year-old). This was similar to a study of white spruce (Weng *et al*., 2008). For the studied wood traits, most estimates of non-additive variance proportions were very low except for AV. Similarly, wood density indirectly measured by Pilo in *E. globulus* showed dominance and epistatic variance estimates to be close to zero (Costa e Silva *et al*., 2004). However, considerable estimates of dominance variance, but with close-to-zero epistatic variance estimates for Pilo were reported

Table 6 Genetic gain (ΔG) and their standard error (in parenthesis) of three deployments for different traits based on selection intensity ($i = 2.063$, e.g. 5% population selected) across two sites[(a)].

Variables	ΔG (%)		
	SOP	CIN	CRC
Ht_6	5.1 (1.9)	6.4 (2.2)	8.9 (3.0)
Ht_{12}	2.7 (1.5)	7.0 (1.7)	9.6 (2.3)
DBH_{12}	5.5 (2.1)	7.0 (2.2)	9.8 (3.1)
DBH_{26}	7.3 (2.0)	7.4 (1.9)	10.6 (2.7)
$Pilo_{26}$	7.0 (1.7)	7.7 (1.3)	10.6 (1.6)
AV_{26}	1.9 (0.7)	3.5 (0.5)	4.7 (0.6)
MOE_{26}	11.2 (3.0)	13.8 (1.6)	17.8 (1.8)
$BkTh_{27}$[a]	5.3 (1.7)	6.1 (1.4)	8.1 (1.6)
$Resi_{27}$[a]	10.7 (4822.5*)	10.9 (4948.5*)	13.1 (3820.9*)
GA_{27}[a]	48.3 (31.5*)	57.7 (36.4*)	65.3 (24.8*)

SOP = open-pollinated seedling deployment, CIN = clonal deployment of the best tree individuals, CRC = clonal deployment of the best replicated clones. Note that the standard error could be biased when data are not sufficient to estimate genetic parameters in ASReml-R v4.1 compared with ASReml standard alone v4.1.

for Norway spruce (Chen *et al*., 2020). Different populations and genetic structure between two different populations and measured at different age may cause such different results.

Estimates of heritability are critical for understanding the genetic structure of natural forest tree populations as well as breeding populations in tree improvement programs. Broad-sense and narrow-sense heritability are both population-specific, trait-specific and heavily affected by environmental homogeneity, which includes genetic testing (White *et al*., 2007).

The higher broad-sense heritability (H^2) estimates as compared with narrow-sense heritability (h^2) in this study indicated there was considerable non-additive genetic variance exploitable in breeding and deployment programmes for Norway spruce. Values of h^2/H^2 near one imply that the amount of non-additive variance is very small, and clonal values are similar to breeding values thus indicating that any advantages of clonal forestry would be small in comparison to the conventional use of seedling deployment (White *et al*., 2007). In this study, the h^2/H^2 ratios ranged from 0.40 to 1.00 for height and DBH growth traits while they were similar for most wood properties (0.54–0.98). These findings were comparable to the normal range of h^2/H^2 recorded for tree growth traits. In previous studies on Norway spruce, the ratio of h^2/H^2 varied from 0.60 to 0.84 and the author indicated that a valid comparison must use datasets from the same trial with comparable pedigree (Wu, 2018).

Narrow-sense heritability estimates were low to moderate for growth traits (0.08–0.16) and from moderate to high for wood property traits (0.15–0.51) in this study. These results were comparable to observations made in the recent studies of Norway spruce that narrow-sense heritability ranged from 0.03 to 0.40 for growth and from 0.15 to 0.53 for wood properties (i.e. AV, Pilo, Resi, MOE, GA and wood density) (Hannrup *et al*., 2004; Hallingbäck *et al*., 2008; Steffenrem *et al*., 2009; Kroon *et al*., 2011; Högberg *et al*., 2014; Chen *et al*., 2015; Chen *et al*., 2020). Early estimates of broad-sense heritability in Norway spruce from

clone tests in the field ranged from 0.17 to 0.40 for height growth at 1 to 10 years (Roulund *et al*., 1986; Bentzer *et al*., 1989; Lepistö, 1993; Högberg and Karlsson, 1998). In this study, estimates of broad-sense heritability were 0.15–0.20 for height at age 6–12, and 0.16 for DBH at age 12–26. These reports on Norway spruce showed that broad-sense heritabilities of growth traits and wood density were comparable to the results in a previous study (In Chen *et al*., 2020, Resi and Pilo considered as a proxy of wood density), but higher for GA compared with the result (0.43–0.44) in Hannrup *et al*. (2004). Hallingbäck *et al*. (2008) reported that GA had a broad-sense heritability of 0.44–0.63, which was similar to the result of this study (0.61).

Clonal mean heritability estimates ranged from 0.28 to 0.39 for height, 0.31 to 0.32 for DBH and 0.38 to 0.78 for wood traits in this study (Table 4). These heritabilities were lower than estimates for growth in the same species in previous studies (Bentzer *et al*., 1989; Lepistö, 1993) and other species, such as white spruce (Weng *et al*., 2008), black spruce (Mullin and Park, 1994) and loblolly pine (Isik *et al*., 2005; Baltunis *et al*., 2007). However, they were close to the report on Norway spruce in which clonal mean heritability estimates ranged from 0.28 to 0.60 for height, 0.22 to 0.57 for DBH and 0.52 to 0.66 for wood traits (i.e. AV, MOE, Pilo and Resi) (Chen *et al*., 2020). The possible reason is that the new deployment strategy in Norway spruce clonal progeny test series in Sweden used approximately three ramets per clone in each of three or four test trials, compared with several times this number for other species (Mullin and Park, 1994; Weng *et al*., 2008; Baltunis *et al*., 2009). As we expected, clonal mean heritability is always higher than broad-sense heritability for all the traits even there is no non-additive variance. This is because clonal mean heritability is estimated based on smaller residual variance or environment variance (see equation 16, replaced residual variance ($\hat{v}_E$) in estimate of broad-sense heritability by $\frac{\hat{V}_E}{r}$).

Uncertainty of dissecting variance components in the clonal full-sib trials

Partitioning phenotypic variation accurately into additive, dominance, epistatic effects is a complex problem (Carlborg and Haley, 2004; Mackay, 2014), but it is also important for prediction in plant and animal breeding (Jiang *et al*., 2017; Forsberg *et al*., 2017). In this study, we observed small or moderate non-additive effects for growth traits, but none of them was significant, except for tree height at tree age 12 (here, 1.96 times standard error less than variance components was considered as significant, $P < 0.05$). In this study, given the sufficient genetic connection in additive levels between two trials (e.g. 72 overlapped families in the total of 167 families), we think that the reasons of large standard errors for non-additive effects and non-additive by site effects could be that (1) six clones per family on average may not be enough to accurately estimate the non-additive effects. More clones per family would be recommended for improving the estimates of genetic parameters; (2) only 88 genotypes (548 and 555 at each site) were replicated between sites and this is probably the reason why G × E was difficult to be detected for epistatic effects, and (3) the full diallel matings would be more ideal to separate additive from dominance effects than the current sparse partial diallel design. However, if the genomic relationship matrix is available, the genetic parameter estimate could be more accurate for dominance (Chen *et al*., 2019; Thavamanikumar *et al*., 2020). Given these findings in this study, we recommend that larger sample size, particularly within family, would be needed to accurately estimate genetic parameter and their GxE.

Trait–trait correlations

In genetic studies, it is necessary to distinguish between two different sources of correlation between traits: genetic and environmental (Falconer and Mackay, 1996). The genetic correlations between traits can be used to predict the effectiveness of direct selection on a measured trait with the primary goal of indirectly improving a correlated target trait that is more difficult or expensive to assess (Isik *et al*., 2017). However, almost all of these estimates were additive genetic correlations (White *et al*., 2007). Total genetic correlation will be more frequently reported as clonal program become more common and it also is important to distinguish the two types of correlation. Estimates of total genetic correlations among traits were generally comparable or slightly different from additive genetic correlations but had lower standard errors. Strong genetic correlation among growth traits in this study were similar to those of other studies, ranging from 0.70 to nearly one (White *et al*., 2007) and agreed also with previous studies on Norway spruce (0.48–0.95) (Roulund *et al*., 1986; Bentzer *et al*., 1989; Hannrup *et al*., 2004; Kroon *et al*., 2011; Chen *et al*., 2015). The observed unfavourable genetic relationships between DBH and Resi, Pilo and MOE were slightly weaker than those observed for the same species (Chen *et al*., 2015) and for *Pinus taeda* (Isik and Li, 2003), but were comparable to observations for other conifer species such as *Pinus contorta* (Hayatgheibi *et al*., 2017) and *Pinus sylvestris* (Fundova *et al*., 2018). In the present study, Resi and Pilo showed a strong negative genetic correlation (−0.88) as expected. In comparison, Pilo showed a strong negative genetic correlation with wood density (−0.62) in Norway spruce (Chen *et al*., 2015) and Scots pine (−0.59 with bark and −0.74 without bark) (Fundova *et al*., 2018) using Siviscan data. Besides, Resi had very high genetic correlation with wood density in *P. taeda* (0.95) (Isik and Li, 2003) and in *P. sylvestris* (0.87–0.96) (Fundova *et al*., 2018). Moreover, the resistograph could provide more detailed measurements of over-bark and under-bark diameter, bark thickness, average resistance of the bark-to-bark (under-bark) trace, average resistance of the outer 50 mm on the entry and exit side of the traces (Downes *et al*., 2018). Thus, the resistograph could be a great tool to reliably assess the relative wood density of standing trees for selection in tree improvement programs.

Both total genetic and additive genetic correlations between grain angle and all remaining traits except Pilo, AV and MOE (−0.60 to 0.32) were very weak and close to zero. The additive genetic correlation between GA and MOE (−0.51) of this study was much higher than that of another report on Norway spruce (Högberg *et al*., 2014). In addition, both total genetic and additive genetic correlation between GA and DBH were all weakly positive and none were significantly different from 0 (Hallingbäck *et al*., 2008).

The negative genetic correlation (adverse relationship) between wood quantity and quality traits (for mostly stem diameter and wood density) is the greatest challenge to conifer breeders. In any case, it will always be important for forest

managers to be aware of the genetic characteristics of the reproductive materials used, both positive and negative aspects (Steffenrem *et al*., 2009). To overcome the adverse genetic correlations, two approaches for breeding programmes are recommended by Wu *et al*. (2008): (1) establishing effective breeding objectives for structural timber products in the short term, and (2) for long-term strategy, by dissecting the genetic basis of the adverse relationship, designing optimal breeding strategies based on the genetic architecture.

Response to different selection schemes

The deployment of elite and adaptable clones across sites is a sound gene resource management strategy for maximizing realized genetic gains. This is one of the most important strategies in tree improvement programs. For this to be possible, a large number of genetically diverse clones that are tested across multiple sites should offer significant total genetic variation and minimal G × E (Baltunis *et al*., 2013). Reliable estimates of all genetic parameters are required for meaningful calculations of genetic gain (Mullin and Park, 1992). Clone testing and deployment in conifer species would possibly bring an additional genetic gain of 5–25 per cent, effectively doubling that obtainable from seedling-based forestry within the same generation (Wu, 2018). The CRC deployment strategy was the most effective as compared with two other deployment strategies in this study, which is in line with the most recent study for the same species (Chen *et al*., 2020). Actually, the top 5 per cent selection of replicated clones was predicted to yield 9.9–10.5 per cent genetic gain for DBH, a relative improvement of 39.4–83.5 per cent over SOP in this study. Similarly, a research on radiate pine found 24 per cent genetic gain of replicated clones, a relative improvement of more than 100 per cent over family forestry (Baltunis *et al*., 2009). The size of the testing population, the number of clones within each family, the ratio of additive to non-additive genetic variances and accuracy of the progeny testing mainly decide variation in terms of estimated extra gains (Wu, 2018).

The report of Chen *et al*. (2020) showed genetic gains of 8–16 per cent for growth and 5–18 per cent for wood quality traits as a result of applying the CRC-strategy and these gains were slightly higher or similar to the results of this study. Genetic gains for tree height were estimated between 10 and 25 per cent from similar clone trials in Germany, Denmark, Norway and Finland (Bentzer, 1993). When using the best 10 per cent of tested and replicated clones, estimated genetic gain for tree height in this study ranged from 7.6 to 8.2 per cent and were smaller than 13–25 per cent in the same species in Sweden reported by (Bentzer *et al*., 1988; Karlsson 1993), and 13–19 per cent in Finland (Lepistö, 1993). Wu (2018) summarized from previous studies on conifer species that an extra genetic gain of 5–25 per cent would be effectively possible from clone testing and deployment. Moreover, Rosvall (2011) suggested considering more flexibility by relaxing the number of ramets planted per clone, in favour of increasing the number of clones to be tested. Thirty to forty clones per family to realize the maximum genetic gain were recommended for different clonal selection scenarios, either selecting the best ten or twenty clones without any co-ancestry restriction or the best single clone from each of the best ten or twenty families (Chen *et al*., 2020). The extra genetic gains from the clonal testing in Norway spruce indicate that clonal deployment is an advantage if superior clones can be identified at early testing stage with an implementation of mass propagation such as mini-cutting, tissue culture. The genomic selection within the elite-family at the seed-forming stage in combination with somatic embryogenesis might be the future of Norway spruce clonal deployment for the increased genetic gain.

Conclusions

This study indicated that both non-additive and additive genetic variances were important sources of total genetic variances for Norway spruce growth and wood quality traits. Additive genetic variation accounted for the majority of the total genetic variation for growth trait and wood traits, whereas non-additive genetic variation is important for growth traits compared with wood quality traits. The relative importance of genetic variances for growth traits varied with age. Wood quality traits (i.e. Resi, Pilo, AV, MOE and GA) had generally higher heritability than growth traits. Total genetic correlations among observed traits were generally comparable to additive genetic correlations. Wood density measured indirectly by Resi and Pilo, and MOE showed moderate and unfavourable genetic correlations with DBH but weak correlations with tree height. The predicted genetic gain of clonal deployment of the best tested and replicated clones was the greatest, followed by clonal deployment of the best individual trees, and then the deployment of seedlings from open pollination of the best individuals. Under selection of the best 5 per cent clones, genetic gains for DBH were estimated to obtain 9.8–10.6 per cent from replicated clonal testing (CRC). This corresponds to a relative improvement of 40.0–43.2 per cent over deployment of the best individual (CIN) and 78.2–255.6 per cent over seed orchard deployment (SOP). For wood traits at the same selection intensity, clonal deployment of replicated clones indicated a relative improvement of 13.2–37.7 per cent over CIN and 22.4–147.4 per cent over SOP.

Data Availability

The datasets generated and/or analysed during the current study are available from the corresponding author and Skogforsk on reasonable request.

Supplementary data

Supplementary data are available at *Forestry* online.

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Conflicts of interest statement

The authors declare that they have no conflict of interest.

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