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Genetic analysis and clonal stability of two yellow cypress clonal populations in British Columbia

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Abstract

Genetic analysis of height and form at age 12 years of 697 yellow cypress (*Callitropsis nootkatensis* [D. Don] Oerst.) clones tested across seven sites in coastal British Columbia (BC) were explored in populations: Population 1 – No Pedigree and Population 2 – Reconstructed Pedigree. Genetic variances were statistically significant but generally higher $\hat{\sigma}^2_g$ was observed for Population 2. Height and form were under low to moderate genetic control as indicated by clonal repeatability and estimates were relatively similar between populations. For example, average $\hat{H}^2$ in Population 2 was 0.31 for height (range: 0.18–0.45) and 0.22 for form (range: 0.06–0.32). While average $\hat{H}^2$ in Population 1 was 0.25 for height (range: 0.19–0.35) and 0.18 for form (range: 0.09–0.27). The reconstructed pedigree in Population 2 allowed partitioning the genetic variance ($\hat{\sigma}^2_g$) into component parts of additive ($\hat{\sigma}^2_a$), specific combining ability ($\hat{\sigma}^2_s$), and clone ($\hat{\sigma}^2_c$); however, general lack of structure within the population resulted in variance components to be estimated with little precision for additive and specific combining ability. The majority of genetic variation was associated with clone for both traits. For example, $\hat{\sigma}^2_c$ accounted for 57.6% and 62.5% of the total genetic variance for height and form, respectively. Growth and form responses of clones across test environments were relatively stable and overall type-B genetic correlations were in excess of 0.8 for both traits implying clones selected for production populations should respond favorably across the seed planning zone for yellow cypress in coastal BC.

Key words: Yellow cypress, clones, genetic testing, genotype x environment, clonal repeatability.

Introduction

Long-term tree improvement programs aim to increase the population mean breeding value of a few key traits through breeding and selection of superior genotypes. These programs are based on classical recurrent selection for general combining and focus on exploiting the additive portion of the genetic variance. In British Columbia (BC), Canada, tree breeding programs strive to improve economic and adaptive traits (e.g., disease resistance, growth rate, and wood quality) while maintaining acceptable levels of genetic variability (YANCHUCK, 2001).

Yellow cypress (*Callitropsis nootkatensis* [D. Don] Oerst.) is an ecologically and commercially important coastal conifer inhabiting sea level to montane coastal forests of the Pacific Northwest from Alaska to northern Washington, with more southerly populations occupying disjunct and higher elevation sites, and several outliers in the interior rainforests (BURNS and HONKALA, 1990). Yellow cypress has a commercial rotation length of 60 years and its wood has excellent characteristics for milling and is suitable for: furniture, molding, paneling,

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flooring, utility poles and many more end-uses (BURNS and HONKALA, 1990). It is well-adapted to cool snowy habitat and has significant phenotypic plasticity, a long reproductive cycle, ability to be cloned, indeterminate growth, and high inbreeding rates (EL-KASSABY et al., 1991; HAWKINS et al., 2001; THOMPSON et al., 2008). However, the use of zygotic seedlings as a mode of propagation in yellow cypress faces a number of obstacles for reforestation as well as breeding programs: 1) seed production from natural stands and orchards is inconsistent and requires the occurrence of favorable environmental conditions during the pollination, fertilization, and seed and cone development stages, a period that spans two consecutive years (EL-KASSABY et al., 1991; ANDERSON et al., 2002; HAK and RUSSELL, 2004), 2) bulk seed-cone collections are often generated from an unknown number of parents with unknown parental contribution, 3) first- and second-year seed-cones are difficult to distinguish, and thus, operational cone collections often contain developmentally immature seed (OWENS and MOLDER, 1974; OWENS and MOLDER, 1975; OWENS and MOLDER, 1977), and 4) mature seed require prolonged seed pre-treatment (i.e., stratification) for breaking dormancy with variable germination among seed donors, thus reducing seedling recovery (PAWUK, 1993). Yellow cypress clonality occurs naturally (THOMPSON et al., 2008) and operational nursery techniques for producing rooted cuttings have been developed (RUSSELL, 1992). Therefore, vegetative propagation of yellow cypress is considered a viable option for production of planting stock for reforestation (KARLSSON and RUSSELL, 1990; RUSSELL, 1992).

Two different clonal strategies have been implemented for yellow cypress improvement in British Columbia. The British Columbia Forest Service established cloned seedlings from partial diallels with the objectives of providing elite clones for use on all public lands in BC and for identifying individuals for advanced generation breeding. In this study, we report on an alternative, yet simpler, strategy developed by Western Forest Products Inc. (WFP) whereby clones from wind-pollinated, natural stands' bulk seed collections were used for testing and the identifying of superior clones for deployment on their forest lands (see MASSAH et al., 2010 for details). While the former strategy controls the pedigree, thus providing greater deployment flexibility and accurate assessment of genetic gain and diversity as well as the estimation of clonal breeding values, the latter can be less expensive in that costs associated with breeding are avoided; however, it lacks pedigree control, thus genetic gain and diversity estimates are generally inaccurate and subjective, and clonal breeding values cannot be estimated.

Deployment of well-tested clones is theoretically superior to family forestry as the former captures the total extent of genetic variation while the latter only deals with the additive variance component, resulting in higher potential genetic gain (LIBBY and RAUTER, 1984; MATHESON and LINDGREN, 1985; MULLIN et al., 1992; SHELBOURNE, 1992; O'NEILL et al., 2005; BALTUNIS et al., 2007; BALTUNIS et al., 2009). However, prediction of the genetic performance of clones needs to be done efficiently and optimally in order to select the best genotypes for deployment. The more information incorporated in the prediction of genetic effects, such as performance of relatives, the more confidence one should expect from predictions. For example, YE et al. (2007) reported that multiple-generation data such as including first-generation information in analyses of second-generation progeny data was beneficial in improving ranking and reliability of predicted breeding values. BALTUNIS et al. (2009) also reported that prediction of the genetic merit of clones was improved by incorporating information from relatives from seedling progeny trials in the clonal analyses.

Many factors can contribute to errors associated with predicting breeding values (BV) or clonal values (CV) for clonal forestry programs including misidentification of parents and offspring (GELDERMANN et al., 1986; ADAMS et al., 1988; ERICSSON, 1999), ignoring genetic effects in the model (LU et al., 1999), or ignoring genotype by environment (GxE) interactions (COMSTOCK and MOLL, 1963). In addition, epigenetic, non-genetic, or non-random environmental factors can confound estimates of genetic variance. C-effects are non-genetic effects that are common to relatives of a family or members of a group such as clones (LERNER, 1958). If C-effects are present, then the total genetic variation associated with clones will be overestimated (LIBBY and JUND, 1962).

ERICSSON (1999) demonstrated that increasing rates of pedigree error results in under- and over-estimating the additive and dominance genetic variances, respectively, which in turn affect heritability estimates. Using inaccurately defined relationships or falsely assuming no relatedness among tested individuals can lead to biased estimates of genetic variance components, heritability, and breeding values (NAMKOONG, 1966; SQUILLACE, 1974; ASKEW and EL-KASSABY, 1994). Furthermore, models that ignore genetic effects, such as specific combining ability (SCA) can upwardly bias estimates of additive genetic variance, heritability, and parental breeding values and should be avoided (LU et al., 1999). Accounting for relatedness of genotypes through correct pedigree increases the accuracy and precision of the genetic merit (BV or CV) of tested genotypes. Recently, low-input breeding strategies have been proposed in forest trees using reconstructed pedigrees and are referred to as "breeding without breeding (BwB)" (EL-KASSABY and LSTIBUREK, 2009). BWB involves the use of DNA markers to reconstruct a pedigree based on naturally occurring half- and full-sib families resulting from mating among selected parents (EL-KASSABY and LSTIBUREK, 2009).

Genetic variance, heritability, and breeding values or clonal values may be upwardly biased when they are based on a single trial because they are potentially confounded with genotype by environment (GxE) (COMSTOCK and MOLL, 1963). Therefore, tree breeders are concerned with testing genotypes across a range of sites in order to obtain unbiased estimates of genetic effects and to measure the stability of genotypes across different environments. The assessment of GxE is essential for formulating breeding and deployment strategies (WHITE et al., 1993; WHITE et al., 2007). The patterns of

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response across environments may involve either change in the ranking of genotypes and/or alterations in scale (LYNCH and WALSH, 1998). The presence of GxE at the clonal level is of particular concern for clonal forestry programs when the relative ranks of genotypes change markedly across environments. Raymond and NAMKOONG (1990) suggested two general approaches in dealing with GxE. The first approach characterizes the planting area with respect to the interactive behavior of site categories, and then specific genotypes are deployed to particular sites, thus maximizing yield over the total range of planting sites. The second approach identifies stable genotypes (i.e., generalists) that perform well over all environments.

Genetic improvement of yellow cypress is in relatively early stages in British Columbia, and to our knowledge, there have been no published genetic parameters for yellow cypress. Therefore, the objectives of the paper were to 1) determine the genetic variation for height and form at age 12 years for two yellow cypress clonal populations: Population 1 – No Pedigree and Population 2 – Reconstructed Pedigree, and 2) determine the genetic stability of yellow cypress clones and the importance of GxE for height and form across multiple sites.

Material and Methods

Genetic material, experimental design and phenotypic measurements

The genetic material in this study came from a series of yellow cypress clonal trials managed by Western Forest Products Inc. (WFP) in British Columbia, Canada (*Table 1*). The populations were previously described by Massah et al. (2010), but briefly, WFP collected seed from 33 different natural seedlots across Vancouver Island, British Columbia. Generally, seeds from 29 or more parent trees were collected from non-neighboring trees and bulked within each seedlot (MASSAH et al., 2010). Therefore, the genetic identity (pedigree) of the seedlings produced from these seedlots was unknown. For this study, seedlings from five of the seedlots were phenotypically selected in the nursery after the first growing season (at age one) based on height and developed into hedges to produce vegetatively propagated rooted cuttings for use in field trials keeping track of clonal identity. Over two series of trials, seven yellow cypress clonal trials were established (*Table 1*). All trials were randomized complete block designs with three single tree plots per clone per replicate with three replicates (9 ramets per clone per trial). Rows and columns (X and Y coordinates) were included as a source of incomplete blocking to account for microsite variation across the trials.

In total, 697 yellow cypress clones from the five bulked seedlots were tested across the seven trials. Within each trial, clones were divided into two populations. Population 1 contained 289 clones from two seedlots (Seedlots 3 and 4) and were not genotyped. The number of clones from Population 1 (No Pedigree) tested at each trial ranged from 65 (trial 2) to 157 (trial 7). For Population 2, seeds from 30, 29 and 36 parent trees were collected from non-neighboring trees and bulked for Seedlots 1, 2, and 5, respectively (MASSAH et al., 2010). A total of 408 clones from these three seedlots were genotyped for pedigree reconstruction using SSR molecular markers (MASSAH et al., 2010). The number of clones from Population 2 (Reconstructed Pedigree) tested at each trial ranged from 87 (trial 2) to 233 (trial 7). Assuming the grouping structure based on molecular markers was correct, the reconstructed pedigree allowed clones from the three bulked seedlots to be assigned to parent and full-sib family groups within parent (*Table 2*). The reconstructed pedigree identified a total of 57 half-sib families (seed trees) with 19, 21 and 17 seed trees in seedlots 1, 2, and 5, respectively (MASSAH, 2008). Not all clones were tested across trials; Table 3

Table 1. – Details and geographic information for seven yellow cypress clonal trials located on Vancouver Island, British Columbia.

Series	Trial	Trial ID	Location	Latitude (N)	Longitude (W)	Elevation (m)
1	1	PML1	Port McNeill	50°35'	127°13'	130
	2	MRH1	Mahatta River	50°28'	127°38'	565
	3	JRH1	Jordan River	48°30'	124°08'	755
	4	JRL1	Jordan River	48°30'	124°17'	160
2	5	PML3	Port McNeill	50°38'	127°23'	180
	6	JRL3	Jordan River	48°25'	124°01'	110
	7	JRH3	Jordan River	48°30'	124°10'	740

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Table 2. – Structure of yellow cypress clones from two populations tested at seven trials: Population 1 – no pedigree, and Population 2 – reconstructed pedigree based on molecular markers.

Series	Trial	*Population 1 – No Pedigree*	*Population 2 – Reconstructed Pedigree*					
		# Clones	# Clones	# Seed trees (HS)	Average clones per HS	# Full-sib (FS)	Average clones per FS	Average FS per HS
1	1	136	191	49	3.9	98	1.9	2.0
	2	65	87	40	2.2	60	1.5	1.5
	3	68	88	40	2.2	62	1.4	1.6
	4	135	191	49	3.9	98	1.9	2.0
2	5	78	118	45	2.6	76	1.6	1.7
	6	82	126	47	2.7	79	1.6	1.7
	7	157	233	54	4.3	115	2.0	2.1

Table 3. – Number of clones in each trial (bold, diagonal) and number of clones in common between pairs of trials (above diagonal).

a. Population 1 – No pedigree

	PML1 (1)	MRH1 (2)	JRH1 (3)	JRL1 (4)	PML3 (5)	JRL3 (6)	JRH3 (7)
PML1 (1)	**135**	65	67	134			
MRH1 (2)		**65**	65	64			
JRH1 (3)			**68**	66			
JRL1 (4)				**135**			
PML3 (5)					**99**	77	99
JRL3 (6)						**82**	78
JRH3 (7)							**157**

b. Population 2 – Reconstructed Pedigree

	PML1 (1)	MRH1 (2)	JRH1 (3)	JRL1 (4)	PML3 (5)	JRL3 (6)	JRH3 (7)
PML1 (1)	**191**	87	88	191			
MRH1 (2)		**87**	85	87			
JRH1 (3)			**88**	88			
JRL1 (4)				**191**			
PML3 (5)					**118**	116	118
JRL3 (6)						**126**	126
JRH3 (7)							**237**

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shows the number of clones in common, or connectedness, among trials for both populations.

LAMBETH (1980) demonstrated that early selection near 20% of the rotation age was efficient based on juvenile-mature correlations. Therefore, assuming a rotation age of 60 years for yellow cypress, height was measured at the end of the 12th growing season to the nearest cm and tree form was assessed on a categorical 1–4 scale with the following definitions:

1 = Superior form – single stem, compact (short branches or branches down-swept close to bole),

2 = Good form – single stem, heavier branches, some up-sweep but none >50% stem height,

3 = Poor form – multi-stem with one stem dominant, one or more >50% stem height, and

4 = Cull – multiple stem without dominance.

Statistical analyses and genetic parameters

A series of analyses were conducted in ASReml (GILMOUR et al., 2009) in order to estimate the variances and genetic parameters associated with height and form measured at age 12 for each of the two populations. First, a univariate mixed-effects individual-tree model was used on a single-site basis for each trait.

Univariate, single-site analyses:

$$y_{ijklmnopg} = \mu = R_i + S_j + RS_{ij} + P_k + row_{o(i)} + col_{p(i)} + add_{l(k2)} + sca_{m(k2)} + clone_{n(k)} + rclone_{in(k)} + error_{ijklmnopq} \quad [1]$$

where $y_{ijklmnopg}$ is the phenotypic measure of height or form, μ is the overall phenotypic mean, R_i is the fixed effect of the i^{th} replicate,

S_i is the fixed effect of the j^{th} seedlot,

RS_{ij} is the fixed effect of the interaction between replicate and seedlot,

P_k is the fixed effect of the k^{th} population,

$row_{o(i)}$ the random effect of the o^{th} row within replicate ~ $N(0, \hat{\sigma}^2_{row})$,

$col_{p(i)}$ is the random effect of the p^{th} column within replicate ~ $N(0, \hat{\sigma}^2_{col})$,

$add_{l(k2)}$ is the random additive genetic effect in Population 2 (Reconstructed Pedigree) ~ $N(0, \hat{\sigma}^2_a)$,

$sca_{m(k2)}$ is the random genetic effect of specific combining ability in Population 2 ~ $N(0, \hat{\sigma}^2_s)$,

$clone_{n(k)}$ is the random genetic effect of clone within population, where in Population 2, ~ $N(0, \hat{\sigma}^2_c)$ and in Population 1 (No Pedigree) $clone_{n(k)}$ represents the total genetic variance ~ $N(0, \hat{\sigma}^2_g)$,

$rclone_{in(k)}$ is the random effect of replicate by clone within population interaction ~ $N(0, \hat{\sigma}^2_{rg(k1)})$ and $N(0, \hat{\sigma}^2_{rg(k2)})$ for Populations 1 and 2, respectively,

and $error_{ijklmnopq}$ is the random within population residual effect ~ $N(0, \hat{\sigma}^2_{e(k1)})$ and $N(0, \hat{\sigma}^2_{e(k2)})$ for Populations 1 and 2, respectively.

The total genetic and phenotypic variances were estimated for both populations at each site:

$\hat{\sigma}^2_g$ = total genetic variance associated with clones for Population 1,

$\hat{\sigma}^2_P = \hat{\sigma}^2_g + \hat{\sigma}^2_{rg(k1)} + \hat{\sigma}^2_{e(k1)}$ = phenotypic variance for Population 1,

$\hat{\sigma}^2_g = \hat{\sigma}^2_a + \hat{\sigma}^2_s + \hat{\sigma}^2_c$ = total genetic variance associated with clones for Population 2,

$\hat{\sigma}^2_P = \hat{\sigma}^2_a + \hat{\sigma}^2_s + \hat{\sigma}^2_c + \hat{\sigma}^2_{rg(k2)} + \hat{\sigma}^2_{e(k2)}$ = phenotypic variance for Population 2.

It should be noted that with an individual-tree model the additive genetic variance ($\hat{\sigma}^2_a$) is estimated directly, and therefore, the clone term in the model for Population 2 does not estimate the variance associated with clones within full-sib family ($\hat{\sigma}^2_{c(FS)}$), but rather $\hat{\sigma}^2_c = \hat{\sigma}^2_{c(FS)} - 0.5\hat{\sigma}^2_a$ (e.g., COSTA E SILVA et al., 2004).

Clonal repeatability ($\hat{H}^2$), or individual-tree broad-sense heritability, was estimated on a single-site basis assuming that no bias has risen from extraneous epigenetic effects (C-effects) that may have been introduced in the course of clonal propagation:

$$\hat{H}^2 = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_P}$$

Additionally, repeatability of clonal means ($\hat{H}^2_{\bar{C}}$) was estimated as:

$$\hat{H}^2_{\bar{C}} = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_g + \hat{\sigma}^2_{rg(k1)}/_r + \hat{\sigma}^2_{e(k1)}/_t} =$$

estimate of repeatability of clonal means for Population 1 and

$$\hat{H}^2_{\bar{C}} = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_g + \hat{\sigma}^2_{rg(k2)}/_r + \hat{\sigma}^2_{e(k2)}/_t} =$$

estimate of repeatability of clonal means for Population 2, where r and t are the number of replicates and harmonic mean number of ramets per clone, respectively.

Approximate standard errors for variance components and clonal repeatability estimates were calculated using a Taylor series expansion method in ASReml (GILMOUR et al., 2009).

Variances are not independent of the scale and the mean of the respective traits (SOKAL and ROHLF, 1995). Therefore, the coefficient of genetic variation (CGV) was estimated in order to compare the genetic variance across trials. The CGV expresses the genetic variance relative to the mean of the trait of interest and gives a standardized measure of the genetic variance relative to trait mean. The higher the CGV, the higher is its relative variation.

$$\text{CGV} = \frac{\hat{\sigma}_g}{\bar{x}} \times 100\%$$

where $\hat{\sigma}_g$ is the square root of the total genetic variance and $\bar{x}$ is the population mean. An alternative approach was used to estimate CGV for form which is a subjectively measured trait and bounded by a scale (BURDON, 2008).

$$\text{CGV} = \frac{\hat{\sigma}_g}{\sqrt{(X_{mean} - X_{min})(X_{max} - X_{mean})}} \times 100\%$$

where X_{mean} is the mean and X_{min} and X_{max} are the lower and upper bounds of the scale, respectively.

A series of across-trial analyses were conducted for pairs of trials within Series 1 (Trials 1–4), pairs of trials within Series 2 (Trials 5–7), all four Series 1 trials, all

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three Series 3 trials, and the combined data set from all seven trials (ALL) in order to calculate unbiased estimates of clonal repeatability for height and form and to estimate the stability of clones for height and form across trials.

$$\mathbf{y}_i = \mathbf{X}_i\mathbf{b}_i + \mathbf{Z}_{1i}\mathbf{m}_i + \mathbf{Z}_{2i}\mathbf{n}_i + \mathbf{Z}_{3i}\mathbf{a}_i + \mathbf{Z}_{4i}\mathbf{s}_i + \mathbf{Z}_{5i}\mathbf{c}_i + \mathbf{Z}_{6i}\mathbf{g}_i + \mathbf{Z}_{7i}\mathbf{p}_i + \mathbf{Z}_{8i}\mathbf{q}_i + \mathbf{e}_{k1i} + \mathbf{e}_{k2i}, \quad [2]$$

where $\boldsymbol{y}_i$ is the vector of observations indexed (i) by site, $\mathbf{b}_i$ is the vector of fixed effects (i.e., mean, trial, population, replicates, seedlots), and $\mathbf{X}_i$ is the known incidence matrix relating observations in $\mathbf{y}_i$ to the fixed effects in $\mathbf{b}_i$ where

$$\mathbf{X}_i\mathbf{b}_i = \begin{bmatrix}\mathbf{X}_1 & \mathbf{0}\\ \mathbf{0} & \mathbf{X}_2\end{bmatrix}\begin{bmatrix}\mathbf{b}_1\\ \mathbf{b}_2\end{bmatrix},$$

$\mathbf{m}_i$ is the vector of random row within replicate effects ~ MVN($\mathbf{0}, \mathbf{M}\otimes\mathbf{I}_r$) where

$$\mathbf{M} = \begin{bmatrix}\hat{\sigma}^2_{row1} & 0\\ 0 & \hat{\sigma}^2_{row2}\end{bmatrix},$$

$\mathbf{n}_i$ is the vector of random column within replicate effects ~ MVN($\mathbf{0}, \mathbf{N}\otimes\mathbf{I}_c$) where

$$\mathbf{N} = \begin{bmatrix}\hat{\sigma}^2_{col1} & 0\\ 0 & \hat{\sigma}^2_{col2}\end{bmatrix},$$

$\mathbf{a}_i$ is the vector of random additive genetic effects within Population 2 ~ MVN($\mathbf{0}, \mathbf{D}\otimes\mathbf{A}$) where

$$\mathbf{D} = \begin{bmatrix}\hat{\sigma}^2_{a1} & \hat{\sigma}_{a1a2}\\ \hat{\sigma}_{a1a2} & \hat{\sigma}^2_{a2}\end{bmatrix}$$

and $\mathbf{A}$ is the numerator relationship matrix generated from the reconstructed pedigree for Population 2,

$\mathbf{s}_i$ is the vector of random effects of full-sib family (specific combining ability ~ MVN($\mathbf{0}, \mathbf{E}\otimes\mathbf{I}_f$) where

$$\mathbf{E} = \begin{bmatrix}\hat{\sigma}^2_{s1} & \hat{\sigma}_{s1s2}\\ \hat{\sigma}_{s1s2} & \hat{\sigma}^2_{s2}\end{bmatrix}$$

and $\mathbf{I}_f$ is an identity matrix equal to the number of full-sib families in Population 2,

$\mathbf{c}_i$ is the vector of random effects of clones from Population 2 ~ MVN($\mathbf{0}, \mathbf{F}\otimes\mathbf{I}_{cin2}$) where

$$\mathbf{F} = \begin{bmatrix}\hat{\sigma}^2_{c1} & \hat{\sigma}_{c1c2}\\ \hat{\sigma}_{c1s2} & \hat{\sigma}^2_{c2}\end{bmatrix}$$

and $\mathbf{I}_{cin2}$ is an identity matrix equal to then number of clones in Population 2,

$\mathbf{g}_i$ is the vector of random effects of clones from Population 1 ~ MVN($\mathbf{0}, \mathbf{G}\otimes\mathbf{I}_{cin1}$) where

$$\mathbf{G} = \begin{bmatrix}\hat{\sigma}^2_{g1} & \hat{\sigma}_{g1g2}\\ \hat{\sigma}_{g1g2} & \hat{\sigma}^2_{g2}\end{bmatrix}$$

and $\mathbf{I}_{cin1}$ is an identity matrix equal to the number of clones in Population 1,

$\mathbf{p}_i$ is the vector of random interaction effects between replicate and clones in Population 1 ~ MVN($\mathbf{0}, [\mathbf{Q}\otimes\mathbf{I}_p]$) where

$$\mathbf{P} = \begin{bmatrix}\hat{\sigma}^2_{rg1} & \hat{\sigma}_{rg1rg2}\\ \hat{\sigma}_{rg1rg2} & \hat{\sigma}^2_{rg2}\end{bmatrix}$$

and $\mathbf{I}_p$ is an identity matrix equal to the number of replicates,

$\mathbf{q}_i$ is the vector of random interaction effects between replicate and clones in Population 2 ~ MVN($\mathbf{0}, [\mathbf{Q}\otimes\mathbf{I}_p]$) where

$$\mathbf{Q} = \begin{bmatrix}\hat{\sigma}^2_{rg1} & \hat{\sigma}_{rg1rg2}\\ \hat{\sigma}_{rg1rg2} & \hat{\sigma}^2_{rg2}\end{bmatrix},$$

$\mathbf{e}_{k1i}$ is the random vector of residual terms for Population 1 ~ MVN($\mathbf{0}, [\mathbf{R}_1\otimes\mathbf{I}_1]$) where

$$\mathbf{R}_1 = \begin{bmatrix}\hat{\sigma}^2_{e1} & \hat{\sigma}_{e1e2}\\ \hat{\sigma}_{e1e2} & \hat{\sigma}^2_{e2}\end{bmatrix},$$

$\mathbf{e}_{k2i}$ is the random vector of residual terms for Population 2 ~ MVN($\mathbf{0}, [\mathbf{R}_2\otimes\mathbf{I}_2]$) where

$$\mathbf{R}_2 = \begin{bmatrix}\hat{\sigma}^2_{e1} & \hat{\sigma}_{e1e2}\\ \hat{\sigma}_{e1e2} & \hat{\sigma}^2_{e2}\end{bmatrix},$$

$\mathbf{0}$ is the null matrix, $\mathbf{I}_1$ and $\mathbf{I}_2$ are identity matrices equal to the number of observations in Populations 1 and 2, respectively; $\mathbf{I}_r$ and $\mathbf{I}_c$ are identity matrices equal to the number of rows and columns, respectively; $\mathbf{Z}_{1i}$, $\mathbf{Z}_{2i}$, $\mathbf{Z}_{3i}$, $\mathbf{Z}_{4i}$, $\mathbf{Z}_{5i}$, $\mathbf{Z}_{6i}$, $\mathbf{Z}_{7i}$ and $\mathbf{Z}_{8i}$ are the known incidence matrices relating observations in $\mathbf{y}_i$ to effects in $\mathbf{m}_i$, $\mathbf{n}_i$, $\mathbf{a}_i$, $\mathbf{s}_i$, $\mathbf{c}_i$, $\mathbf{g}_i$, $\mathbf{p}_i$ and $\mathbf{q}_i$ respectively.

In order to determine the extent of genotype by environment interaction (GxE) for each trait, type-B genetic correlations ($\hat{r}_B$) were estimated as in BURDON (1977) as a measure of clonal stability across sites

$$\hat{r}_B = \frac{\hat{\sigma}_{g1g2}}{\sqrt{\hat{\sigma}^2_{g1}\hat{\sigma}^2_{g2}}}$$

is the genetic correlation for Population 1 across sites where values closer to unity indicate little GxE.

For Population 2 the stability of clones was also estimated:

$$\hat{r}_B = \frac{\hat{\sigma}_{g1g2}}{\sqrt{\hat{\sigma}^2_{g1}\hat{\sigma}^2_{g2}}}$$

where $\hat{\sigma}^2_{gi}$ was defined above for Population 2 (Model 1) and the genetic covariance across sites ($\hat{\sigma}^2_{g1g2}$) = $\hat{\sigma}^2_{a1a2}$ + $\hat{\sigma}^2_{s1s2}$ + $\hat{\sigma}^2_{c1c2}$.

Clonal repeatability was estimated for Series 1 trials, Series 2 trials, and over all seven trials for both populations using a model similar to Equation [1] with the addition of T_h which is the fixed effect of h^{th} trial, TS_{hj} is the fixed interaction between trial and seedlot, $tclone_{hn(k)}$ is the random effect of the interaction between trial and clone where for Population 1 ~ N($\hat{\sigma}^2_{tg(k1)}$) and Population 2 ~ N($\hat{\sigma}^2_{tg(k2)}$). Additionally, a heterogeneous error structure was used defining a separate residual effect for each trial-population combination.

Clonal repeatability was calculated for Population 1

$$\hat{H}^2 = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_P} = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_g + \hat{\sigma}^2_{tg(k1)} + \hat{\sigma}^2_{rg(k1)} + \hat{\sigma}^2_E}$$

and Population 2

$$\hat{H}^2 = \frac{\hat{\sigma}^2_g}{\hat{\sigma}^2_P} = \frac{\hat{\sigma}^2_a + \hat{\sigma}^2_s + \hat{\sigma}^2_c}{\hat{\sigma}^2_a + \hat{\sigma}^2_s + \hat{\sigma}^2_c + \hat{\sigma}^2_{tg(k2)} + \hat{\sigma}^2_{rg(k2)} + \hat{\sigma}^2_E}$$

where $\hat{\sigma}^2_E$ is a weighted average of the residual variances where the weight is based on the proportion of

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observations in each trial (n_i) to the total observations in all trials in the analysis (N):

$$\hat{\sigma}_E^2 = \frac{\sum n_i \hat{\sigma}_{e_i}^2}{N}.$$

Type-B genetic correlations were estimated for both height and form to give an indication of the stability of clones across trials (YAMADA, 1962) for all pairwise comparisons between trials within a series, Series 1 trials, Series 2 trials, and over all seven trials for both populations:

$$\hat{r}_B = \frac{\hat{\sigma}_g^2}{\hat{\sigma}_g^2 + \hat{\sigma}_{tg}^2}.$$

Bivariate, single-site individual-tree linear mixed-effects models were used to estimate the genetic and

Table 4. – Yellow cypress clonal trial summary statistics for height (cm) and form (scale of 1–4) at age 12.

a. Population 1 – No pedigree

Series	Trial	Trait	Mean	St. Dev.	Minimum	Maximum	N
1	1	Height	246.4	101.0	30.0	590.0	1117
		Form	2.08	0.54	1	4	1150
	2	Height	220.9	86.1	22.0	480.0	512
		Form	2.28	0.65	1	4	534
	3	Height	182.0	63.6	42.0	410.0	436
		Form	2.67	0.69	1	4	493
	4	Height	205.5	74.1	35.0	460.0	1052
		Form	2.08	0.47	1	4	1076
2	5	Height	301.1	86.1	46.0	500.0	601
		Form	2.11	0.46	1	4	608
	6	Height	430.3	96.7	68.0	650.0	675
		Form	2.48	0.70	1	4	692
	7	Height	270.4	72.1	69.0	490.0	1239
		Form	2.20	0.51	1	4	1253

b. Population 2 – Reconstructed Pedigree

Series	Trial	Trait	Mean	St. Dev.	Minimum	Maximum	N
1	1	Height	254.4	94.8	30.0	600.0	1577
		Form	1.99	0.56	1	4	1601
	2	Height	216.8	86.1	24.0	460.0	670
		Form	2.24	0.68	1	4	698
	3	Height	190.1	66.9	36.0	390.0	612
		Form	2.56	0.68	1	4	663
	4	Height	221.3	79.1	40.0	570.0	1489
		Form	1.99	0.49	1	4	1519
2	5	Height	320.7	89.3	32.0	580.0	933
		Form	2.03	0.52	1	4	952
	6	Height	447.3	95.6	92.0	730.0	1013
		Form	2.57	0.75	1	4	1024
	7	Height	283.7	75.1	51.0	490.0	1833
		Form	2.18	0.58	1	4	1850

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phenotypic correlations between height and form for Populations 1 and 2 using a similar model to Equation [2] above except now $\mathbf{y}_i$ is the vector of observations indexed (i) by trait. A diagonal structure was used when a variance component was bounded at zero based on results from univariate analyses, otherwise a covariance structure was fitted for replicate by genotype ($\hat{\sigma}_{rg1rg2}$), genetic covariance for Population 1 ($\hat{\sigma}_{g1g2}$) and Population 2 ($\hat{\sigma}_{g1g2} = \hat{\sigma}_{a1a2} + \hat{\sigma}_{s1s2} + \hat{\sigma}_{c1c2}$), and for residual effects between height and form ($\hat{\sigma}_{e1e2}$). Other terms were defined above.

The genetic and phenotypic correlations between height and form were estimated as:

$$r_G = \frac{\hat{\sigma}_{g1g2}}{\sqrt{\hat{\sigma}^2_{g1}\hat{\sigma}^2_{g2}}},$$

and

$$r_P = \frac{\hat{\sigma}_{g1g2}+\hat{\sigma}_{rg1rg2}+\hat{\sigma}_{e1e2}}{\sqrt{\left(\hat{\sigma}^2_{g1}+\hat{\sigma}^2_{rg1}+\hat{\sigma}^2_{e1}\right)\left(\hat{\sigma}^2_{g2}+\hat{\sigma}^2_{rg2}+\hat{\sigma}^2_{e2}\right)}}$$

where $\hat{\sigma}_{g1g2} = \hat{\sigma}_{a1a2} + \hat{\sigma}_{s1s2} + \hat{\sigma}_{c1c2}$, and $\hat{\sigma}^2_{gi} = \hat{\sigma}^2_{ai} + \hat{\sigma}^2_{si} + \hat{\sigma}^2_{ci}$ for Population 2.

Results and Discussion

The overall mean height and form for Population 1 clones were 264.6 cm and 2.23, respectively. Population 2 clones had slightly higher overall mean height (278.7 cm) and lower mean form (2.17). However, substantial variability was observed in both populations across the seven trials (*Table 4*). Across all trials, yellow cypress clones' mean height at age 12 years ranged from 182.0 cm (trial 3, Population 1) to 447.3 cm (trial 6, Population 2). Mean form classes yielded lower variability than that observed for height and ranged from 1.99 (trial 1, Population 2) to 2.67 (trial 3, Population 1) (*Table 4*). Trials in Series 1 produced the lowest age 12 height and this could be attributable to the over-intensive site preparation applied where too much organic material was removed.

The reconstructed pedigree allowed partitioning the genetic variance ($\hat{\sigma}^2_g$) into component parts of additive genetic variance ($\hat{\sigma}^2_a$), specific combining ability ($\hat{\sigma}^2_s$), and clone ($\hat{\sigma}^2_c$); however, there was still a general lack of structure within the population (*Table 2*), causing variance components to be estimated with little precision. The majority of genetic variation was associated with clone for both traits (*Table 5*). For example, averaged over all seven trials $\hat{\sigma}^2_c$ accounted for 57.3% of the total genetic variance for height, while $\hat{\sigma}^2_a$ and $\hat{\sigma}^2_s$ accounted for only 31.3% and 11.4%, respectively. Similarly, for form 48.9%, 37.5%, and 13.6% of $\hat{\sigma}^2_g$ were attributed to $\hat{\sigma}^2_c$, $\hat{\sigma}^2_a$, and $\hat{\sigma}^2_s$, respectively, when averaged over all seven trials. These results were in striking contrast to those obtained for the same species, however, from genetically well-structured progeny trials that involved a series of six cloned diallel tests where 9-year height additive genetic variance averaged 72% of the total genetic variance (J. H. Russell, unpublished data). The main difference between the results of the present study and the unpublished data is the presence of too few individuals from each seed-tree were included in the reconstructed pedigree of Population 2 to allow proper estimation of the additive effects ($\hat{\sigma}^2_a$, BVs, narrow-sense heritability) accurately and precisely (based on high standard errors in the current study). For example, of the clones that were genotyped in Population 2 there was only an average of 2.2-4.3 clones tested per seed-tree (40-54 seed-trees tested per trial) at each of the seven trials, as well as 1.4-2.1 clones per full-sib family (*Table 2*).

Although there was little power in partitioning the genetic effects of yellow cypress clones from Population 2, we were able to estimate total genetic variance of clones using the reconstructed pedigree (*Table 5*). Based on the magnitude of the standard errors, the total genetic variance for height and form was statistically significant and similar across seven trials for both populations (*Table 5*). However, there was a general trend with higher $\hat{\sigma}^2_g$ for Population 2 when the reconstructed pedigree was incorporated compared with Population 1 with no pedigree.

There have been several studies using reconstructed pedigrees in natural populations to estimate quantitative genetic parameters (Thomas et al., 2002; Wilson et al., 2003; Herbinger et al., 2006; Blonk et al., 2010). Low-input breeding strategies ("breeding without breeding", BwB) have also been proposed in forest trees using reconstructed pedigrees (El-Kassaby and Lstiburek, 2009). Wang et al. (2010) demonstrated that identification of both parents of trees in commercial plantations originating from a seed orchard is technically and logistically possible and concluded that marker-based pedigree reconstruction could facilitate the use of low input BwB strategies for forest trees. However, when Wang et al. (2010) compared the applicability of BwB to the Swedish breeding programs they concluded that the methods would yield lower genetic gains in their well-structured advanced generation Scots pine (*Pinus sylvestris*) breeding program and advocated its use in their infant Norway spruce (*Picea abies*) program. Pedigree reconstruction was effectively used to convert the incomplete pedigree of polycross (Lambeth et al., 2001; Doerksen and Herbinger, 2010), as well as in open-pollinated families (Grattapaglia et al., 2004; Gaspar et al., 2009; Doerksen and Herbinger, 2010; Hansen and McKinney, 2010) to complete pedigree, thus removing all the inaccuracies associated with both methods' assumption of complete half-sib analysis. Additionally, pedigree reconstruction has been used as a supplemental tool for tree breeders for verifying the authenticity of controlled crosses and identifying pedigree errors (Adams et al., 1988; Ericcson, 1999; Doerksen and Herbinger, 2008) and determining differential male and/or female mating success in several seed orchard populations (Doerksen and Herbinger, 2008; El-Kassaby et al., 2010; Lai et al., 2010; Funda et al., 2011).

Both height and form were under low to moderate genetic control as indicated by clonal repeatability, and estimates were relatively similar between the two populations (*Table 5*). However, $\hat{H}^2$ was usually higher for

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Table 5. – Comparison of genetic variance ($\hat{\sigma}^2_g$), clonal repeatability ($\hat{H}^2$), repeatability of clonal means ($\hat{H}^2_C$) and coefficient of genetic variation (CGV) between Population 1 with no pedigree and Population 2 using the reconstructed pedigree for seven yellow cypress clonal trials for height and form. Genetic variance is partitioned into additive ($\hat{\sigma}^2_a$), specific combining ability ($\hat{\sigma}^2_s$) and clone within family ($\hat{\sigma}^2_c$) for Population 2. Standard errors are given in parentheses.

a. Height

	Population 1 – No Pedigree				*Population 2 – Reconstructed Pedigree*						
Trial	$\hat{\sigma}^2_g$	$\hat{H}^2$	$\hat{H}^2_C$	**CGV (%)**	$\hat{\sigma}^2_g$	$\hat{\sigma}^2_a$	$\hat{\sigma}^2_s$	$\hat{\sigma}^2_c$	$\hat{H}^2$	$\hat{H}^2_C$	**CGV (%)**
1	2016.5 (370.5)	0.23 (0.04)	0.69 (0.05)	18.2%	1932.7 (293.2)	466.8 (454.6)	0	1466.0 (472.8)	0.26 (0.03)	0.72 (0.04)	17.3%
2	1085.6 (317.4)	0.19 (0.05)	0.64 (0.07)	14.9%	1069.4 (317.4)	941.2 (637.8)	0	128.2 (559.1)	0.18 (0.05)	0.58 (0.08)	15.1%
3	805.6 (215.8)	0.26 (0.06)	0.63 (0.07)	15.6%	1020.3 (235.8)	293.1 (875.8)	136.8 (496.6)	590.5 (524.4)	0.27 (0.05)	0.67 (0.05)	16.8%
4	1135.2 (203.1)	0.26 (0.04)	0.72 (0.04)	16.4%	1472.6 (205.0)	0	110.3 (177.3)	1362.2 (247.8)	0.30 (0.03)	0.77 (0.03)	17.3%
5	1228.6 (340.4)	0.19 (0.05)	0.61 (0.08)	11.6%	2418.2 (445.2)	1721.7 (1838.1)	187.4 (856.1)	509.1 (1024.1)	0.34 (0.04)	0.78 (0.04)	15.3%
6	1982.3 (455.5)	0.27 (0.05)	0.73 (0.05)	10.3%	3301.2 (539.2)	0	1292.7 (562.3)	2008.4 (512.3)	0.45 (0.04)	0.81 (0.03)	12.8%
7	1734.8 (249.3)	0.35 (0.04)	0.80 (0.02)	15.4%	2027.0 (241.5)	140.7 (671.3)	241.9 (356.6)	1644.5 (432.8)	0.38 (0.03)	0.82 (0.02)	15.9%

b. Form

	Population 1 – No Pedigree				*Population 2 – Reconstructed Pedigree*						
Trial	$\hat{\sigma}^2_g$	$\hat{H}^2$	$\hat{H}^2_C$	**CGV (%)**	$\hat{\sigma}^2_g$	$\hat{\sigma}^2_a$	$\hat{\sigma}^2_s$	$\hat{\sigma}^2_c$	$\hat{H}^2$	$\hat{H}^2_C$	**CGV (%)**
1	0.037 (0.009)	0.13 (0.03)	0.53 (0.07)	13.4%	0.056 (0.01)	0.044 (0.018)	0	0.013 (0.015)	0.19 (0.03)	0.66 (0.05)	16.8%
2	0.038 (0.016)	0.09 (0.04)	0.44 (0.11)	13.1%	0.027 (0.016)	0.027 (0.016)	0	0	0.06 (0.04)	0.27 (0.13)	11.1%
3	0.095 (0.028)	0.21 (0.05)	0.58 (0.08)	20.7%	0.113 (0.026)	0.06 (0.058)	0	0.054 (0.053)	0.25 (0.05)	0.67 (0.05)	22.4%
4	0.059 (0.01)	0.27 (0.04)	0.67 (0.04)	16.9%	0.073 (0.01)	0.013 (0.015)	0	0.06 (0.016)	0.32 (0.03)	0.72 (0.03)	19.2%
5	0.04 (0.01)	0.19 (0.04)	0.62 (0.06)	13.8%	0.051 (0.012)	0	0.036 (0.012)	0.014 (0.009)	0.20 (0.04)	0.62 (0.06)	15.9%
6	0.109 (0.025)	0.23 (0.04)	0.70 (0.05)	22.0%	0.153 (0.027)	0	0	0.153 (0.027)	0.27 (0.04)	0.67 (0.04)	26.1%
7	0.039 (0.008)	0.16 (0.03)	0.58 (0.05)	13.4%	0.09 (0.012)	0.013 (0.041)	0.021 (0.022)	0.056 (0.023)	0.28 (0.03)	0.74 (0.03)	20.5%

Population 2 (Reconstructed Pedigree) than for Population 1 (No Pedigree). For example, the average $\hat{H}^2$ in Population 2 was 0.31 for height (range: 0.18–0.45) and 0.22 for form (range: 0.06–0.32) across the seven trials. While average $\hat{H}^2$ in Population 1 was 0.25 for height (range: 0.19–0.35) and 0.18 for form (range: 0.09–0.27) across the seven trials. Nevertheless, these values are generally typical to what has been reported in other species. For example, BALTUNIS and BRAWNER (2010) reported $\hat{H}^2$ ranging from 0.09-0.31 for height at age five

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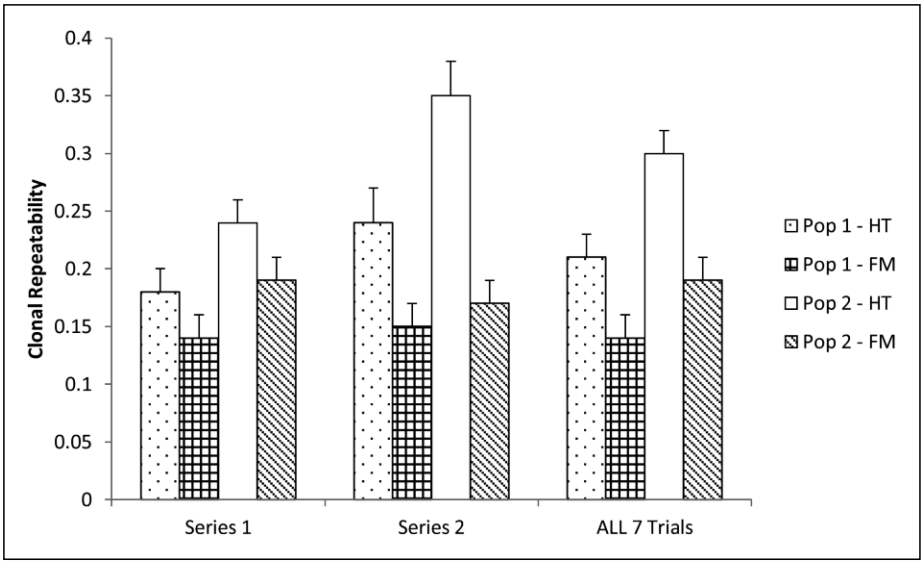

Figure 1. – Clonal repeatability ($\hat{H}^2$) from the across-trial analyses of Series 1 (Trials 1–4), Series 2 (Trials 5–7), and from analysis of ALL trials (Trials 1–7). Standard error bar is included in all estimates of $\hat{H}^2$.

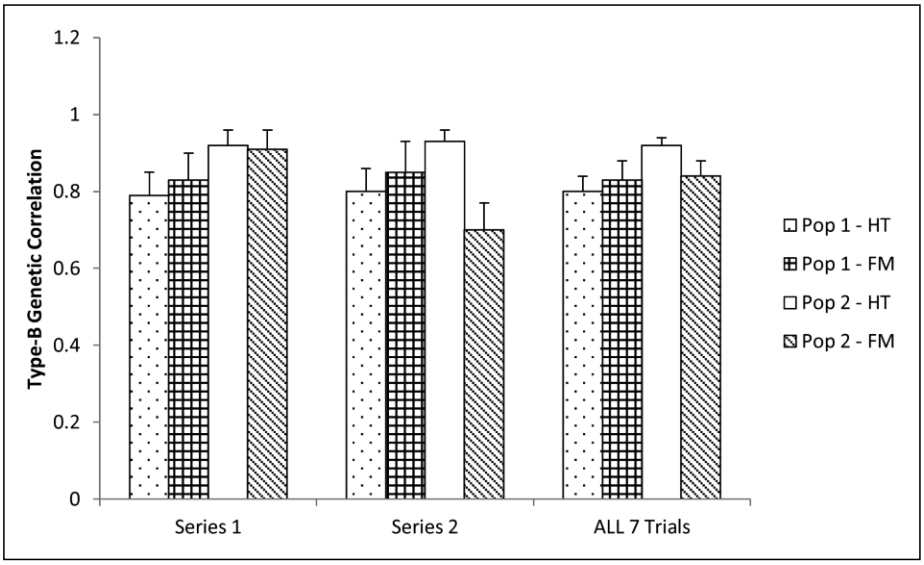

Figure 2. – Type-B genetic correlations ($\hat{r}_B$) from the across-trial analyses of Series 1 (Trials 1–4), Series 2 (Trials 5–7), and from the analysis of ALL trials (Trials 1–7). Standard error bar is included in all estimates of $\hat{r}_B$.

for clones of *Pinus radiata*. In addition, clonal repeatability was estimated from across-trial analyses in order to calculate unbiased estimates of clonal repeatability for height and form. For example, $\hat{H}^2$ for height from the analysis of all seven trials (ALL) was 0.21 ± 0.02 and 0.30 ± 0.02 for Populations 1 and 2, respectively. Similarly for form, $\hat{H}^2$ was 0.14 ± 0.02 and 0.19 ± 0.02 for Populations 1 and 2, respectively, from the ALL analysis (*Figure 1*). Our assumption that clones from Population 1 were unrelated may have been false based on the findings of MASSAH et al. (2010) for Population 2 clones (Reconstructed Pedigree). This may have led to biased estimates of genetic variance and clonal repeatability for Population 1 (NAMKOONG, 1966; SQUILLACE, 1974; ASKEW and EL-KASSABY, 1994). Nevertheless, $\hat{H}^2$ was very similar across populations possibly indicating that there may be little bias in estimates from Population 1 assuming unrelated genotypes compared to Population 2 clones. This may be attributable to the general lack of structure in these populations, eg. ~ three clones per half-sib family on average (*Table 2*). Additionally, when Population 2 clones were assumed to be unrelated (eg., no pedigree information), total genetic variance and clonal repeatability were very similar to results obtained using the reconstructed pedigree (data not shown).

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Repeatability of clonal means was also estimated for height and form and was moderate to high across the seven clonal trials (*Table 5*). For example, in Population 2, $\hat{H}^2_{\bar{C}}$ ranged from 0.67–0.82 and 0.27–0.74 for height and form, respectively. Repeatability of clonal means is a function of the total genetic variance and number of ramets of the tested clones, thus more ramets of a clone is required for testing for traits under low as compared to those under high genetic control (RUSSELL and LIBBY, 1986). Genetic gains will be maximized as repeatability of clonal means approaches unity. Moderately high $\hat{H}^2_{\bar{C}}$ for growth traits has been reported for clones in other conifers (MULLIN et al., 1992; O'NEILL et al., 2005; ISIK et al., 2005; BALTUNIS and BRAWNER, 2010). In addition, the coefficient of genetic variation for height and form ranged from 10.3–18.2% and 11.1–26.1% for height and form, respectively (*Table 5*). These are similar to other reported values from clonal trials in other conifer species (BALTUNIS and BRAWNER, 2010) and are within acceptable variability levels.

The growth and form responses of the yellow cypress clones in both populations across the test environments were relatively stable. Type-B genetic correlations were all moderately high between pairs of trials in Series 1

Table 6. – Type-B genetic correlations ($\hat{r}_B$) for height (above diagonal) and form (below diagonal) between pairs of yellow cypress clonal trials within Series 1 (Trials 1–4) and Series 2 (Trials 5–7). Standard errors are given in parentheses.

a. Population 1 – No pedigree

	PML1 (1)	MRH1 (2)	JRH1 (3)	JRL1 (4)	PML3 (5)	JRL3 (6)	JRH3 (7)
PML1 (1)		0.74 (0.14)	0.67 (0.14)	0.84 (0.07)			
MRH1 (2)	0.90 (0.19)		0.80 (0.14)	0.68 (0.14)			
JRH1 (3)	0.66 (0.18)	0.69 (0.20)		0.80 (0.11)			
JRL1 (4)	0.98 (0.09)	0.78 (0.17)	0.79 (0.13)				
PML3 (5)						0.88 (0.10)	0.81 (0.10)
JRL3 (6)					0.90 (0.09)		0.79 (0.08)
JRH3 (7)					0.90 (0.11)	0.82 (0.11)	

b. Population 2 – Reconstructed Pedigree

	PML1 (1)	MRH1 (2)	JRH1 (3)	JRL1 (4)	PML3 (5)	JRL3 (6)	JRH3 (7)
PML1 (1)		0.97 (0.10)	0.95 (0.07)	0.93 (0.05)			
MRH1 (2)	1		0.90 (0.11)	0.95 (0.09)			
JRH1 (3)	0.90 (0.09)	0.93 (0.17)		0.89 (0.08)			
JRL1 (4)	0.94 (0.05)	0.87 (0.18)	0.77 (0.09)				
PML3 (5)						0.94 (0.04)	0.95 (0.04)
JRL3 (6)					0.64 (0.11)		0.96 (0.04)
JRH3 (7)					0.82 (0.09)	0.65 (0.09)	

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Table 7. – Genetic (r_G) and phenotypic (r_P) correlations between height and form for two populations of yellow cypress clones at seven trials. Standard errors are given in parentheses.

	Population 1 – No Pedigree		*Population 2 – Reconstructed Pedigree*	
Trial	r_G	r_P	r_G	r_P
1	-0.72 (0.09)	-0.44 (0.03)	-0.57 (0.08)	-0.38 (0.03)
2	-0.62 (0.17)	-0.37 (0.04)	-0.88 (0.19)	-0.29 (0.04)
3	-0.75 (0.13)	-0.24 (0.06)	-0.67 (0.10)	-0.30 (0.05)
4	-0.72 (0.08)	-0.35 (0.04)	-0.68 (0.06)	-0.32 (0.03)
5	-0.40 (0.17)	-0.13 (0.05)	-0.51 (0.12)	-0.16 (0.04)
6	-0.06 (0.16)	-0.08 (0.05)	-0.03 (0.11)	-0.004 (0.04)
7	-0.48 (0.10)	-0.24 (0.03)	-0.58 (0.07)	-0.19 (0.03)
Average	*-0.54*	*-0.26*	*-0.56*	*-0.23*

and Series 2 (*Table 6*). For height, type-B genetic correlations ranged from 0.67–0.88 and 0.89–0.97 in Populations 1 and 2, respectively, indicating little biologically important GxE. For form, type-B genetic correlations ranged from 0.64 to unity across both populations with more observed GxE generally within Series 2 trials (trials 5–7) for Population 2. When all seven trials were collectively analyzed, the overall type-B genetic correlations for Population 1 clones were 0.80 ± 0.04 and 0.83 ± 0.05 for height and form, respectively (*Figure 2*). Likewise, the overall type-B genetic correlations for Population 2 clones were 0.92 ± 0.02 and 0.84 ± 0.04 for height and form, respectively (*Figure 2*). It is important to note that the low and high elevation sites within the two series of trials differed by more than 600 m and were within completely different ecological classification zones (MEIDINGER and POJAR, 1991). The lack of GxE in this population is encouraging because it implies that clones selected for production populations should respond favorably across the seed planning zone for yellow cypress in coastal BC.

Genotype by environment interaction at the clonal level has been studied in other taxa, and there have been examples of significant GxE and clonal stability. For example, YU and PULKKINEN (2003) reported significant clone x site interaction for early growth of 24 hybrid aspen (*Populus tremula* x *Populus tremuloides*) clones in Finland. Similarly, significant GxE was detected for volume in *Populus davidiana* clones in Korea, but stable genotypes were identified which behaved in a predictable manner across sites (KOO et al., 2007). BALTUNIS et al. (2008) reported evidence of GxE for height in 3-year old clones of *Pinus taeda* tested in the southeastern United States which they hypothesized it was due to clones changing ranks on high-versus low-fertility sites, but little evidence of GxE for stable carbon isotope discrimination. KANZLER et al. (2003) identified rainfall as the main driver affecting changes in rank across five sites in South Africa for *Pinus patula*. In contrast, GxE interaction for growth appeared to be of insignificant importance in clones of *Picea abies* (ISIK and KLEINSCHMIT, 2003) and *Eucalyptus globulus* (COSTA E SILVA et al., 2004). In addition, little GxE was observed for DBH, and stable clones were identified for *Pinus radiata* within New Zealand (BALTUNIS and BRAWNER, 2010); however, more extensive GxE was observed for form traits (stem straightness and branch quality).

There was a moderate to high, favorable genetic correlation between height and form (*Table 7*). The average genetic correlation across all seven trials was –0.54 and –0.56 in Populations 1 and 2, respectively. In Population 1 r_g ranged from –0.06 ± 0.16 to –0.75 ± 0.13. The average phenotypic correlation for Population 1 was –0.26 (*Table 7*). r_G ranged from –0.03 ± 0.11 to –0.88 ± 0.19 in Population 2, while r_P averaged –0.23. This favorable genetic correlation implies that selection for height will also increase genetic merit of form in the selected population of clones.

Conclusion

The deployment of elite adaptable clones across the landscape is a sound gene resource management strategy for maximizing realized genetic gains. For this to be possible, a large number of genetically-diverse clones that are tested across multiple sites should have significant total genetic variation and minimal GxE. This has been demonstrated with the clonal study presented here. However, because there was initially no pedigree information, optimization of clonal selection for deployment between genetic gain and diversity through estimating effective population size, was not possible. As well, although short-term genetic gains may be maximized through deployment of well-tested clones, long-

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term gains need to involve both clonal selection and recurrent selection for additive genetic variation through repeated selection and breeding. The inclusion of a post hoc pedigree reconstruction for Population 2 in the current study was not effective in partitioning genetic variance components due to the small sample sizes discussed above. Therefore, clones could not be selected based on breeding values and incorporated into a long-term breeding program. One alternative is to use pedigree reconstruction to guide selection of clones from bulk seedlots in order to create a structured pedigree to test in field trials which may be more effective. However, if a genetic improvement program is economically and biologically justified for both clonal deployment and a recurrent selection for additive genetic variation, then a balanced mating design with cloned individuals may be more appropriate.

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