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A quantitative genetic model for mixed diploid and triploid hybrid progenies in tree breeding and evolution

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Abstract Interspecific hybridization has played a critical role in tree evolution and breeding. The findings of triploidy in forest trees stimulate the development of a quantitative genetic model to estimate the nature of gene action. The model is based on clonally replicated triploid progenies derived from a two-level population and individual-within-population mating design in which offspring have a double dose of alleles from the parent and a single dose of alleles from the other parent. With the same genetic assumptions of a diploid model, except non-Mendelian behavior at meiosis, and the experimental variances estimated from a linear statistical model, total genetic variances in the triploid progenies are separated into additive, dominance, and epistatic components. In addition, by combining the new model with the already existing model based on disomic expression, the partitioning of additive, dominant, and epistatic variances can be obtained for a mixed diploid/triploid F_1 progeny population. This paper provides an alternative technique to study the modes of quantitative inheritance for outcrossing, long-lived forest trees in which inbred lines cannot be easily generated. The accuracy for estimating gene action using this technique is discussed.

Key words Interspecific cross · Two-level mating design Triploidy · Quantitative inheritance · Clone · Forest tree

Introduction

Polyploidy has been found in a wide range of plants, with 30–35% of the angiosperms being polyploid (Stebbin 1971). Levin (1983) demonstrated remarkable differences in cell activity, physiology, development, reproductive system, and ecological tolerances betweem polyploids and their diploid progenitors or siblings. In the genus *Populus*, triploidy occurs both spontaneously (e.g., van Buijtenen et al. 1957) and in crosses among diploid parents (Bradshaw and Stettler 1993). Although triploidy remains unnoticed in many forest trees due to the lack of cytological or molecular analysis, it is suggested that triploidy will have implications for tree breeding and the evolution of natural populations (Einspahr et al. 1964; Bradshaw and Stettler 1993).

The occurrences and potential values of triploidy call for the development of a genetic model to estimate the nature of gene action on quantitative traits. Estimates for the modes of quantitative inheritance are traditionally based on generation means and variances, with inbred lines as initial generations (Mather 1949; Mather and Jinks 1982; Hayman 1958, 1960; Cockerham 1963). The long generation intervals and high genetic loads commonly found in forest trees (Klekowski 1988) make it difficult to use inbred lines to reliably resolve genetic variance into its additive, dominant, and epistatic components (Namkoong and Kang 1990). However, with the advent of more widely successful methods of vegetatively propagating forest trees (Cheliak and Klimaszewska 1991; Park et al. 1993), the clonal replication of genotypes has lent a supplemental tool to variance partitioning. Thus, researchers have made use of clonal replicates of open-pollinated families to estimate additive and non-additive variances via an appropriate mating design (Park and Fowler 1987). Others have estimated dominance and epistatic variances of clonal propagules from control-pollinated progenies (Stonecypher and McCullough 1986; Foster and Shaw 1988; Foster 1990; Mullin and Park 1992; Mullin et al. 1992). However, these conventional genetic models cannot be used to conduct a detailed genetic analysis of gene action in triploid families in which regular diploid Mendelian behavior at meiosis is one of the basic assumptions (Comstock et al. 1958).

The objectives of this paper were (1) to formulate a two-level population and individual-within-population mating design that can provide the necessary genetic structure

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among relatives for estimating genetic variances, (2) to develop a genetic model for the partitioning of genetic variance into additive, dominant, and epistatic components in a triploid progeny with two-thirds contribution from a parent and one-third from the other parent, and (3) to incorporate the traditional genetic model appropriate for diploid progeny and the new model for triploid progeny into a mixed one in accordance with the genetic composition of these hybrid progenies.

Two-level mating design

Definition

For forest trees, the factorial (NC State Design II), as designed by Cockerham (1963), is one of the most important mating designs to provide the necessary genetic structure among relatives for estimating genetic variances (Bradshaw and Foster 1992). Suppose there are f_1 and m_1 random mating populations from two tree species, respectively, used for a cross. Within each population of a species, f_2 individuals are randomly chosen as female parents that are factorially mated with m_2 male parents sampled randomly from each population of the other species. Consequently, the entire mating design includes a total of $f_1m_1 \cdot f_2m_2$ full-sib families that are divided into f_1m_1 factorial population combinations (i.e., "full-sib" families at the population level) within each of which f_2m_2 factorial individual-within-population combinations are nested. Such a design is defined as a two-level (population and individual-within-population) factorial and nested interspecific mating design (Hinkelman 1974).

Statistical model

The experimental design provides the method to partition components of genetic and environmental variance (Cockerham 1963). Assume that each full-sib family produces c siblings, all of which are vegetatively propagated into clones each with rs copies (ramets). A total of $f_1m_1f_2m_2c$ clones are planted in a randomized complete block design with r replicates in each of s environments. An analysis of variance model explaining genotype, environment, and genotype × environment interaction effect can be expressed as

$$Y_{ijk} = \mu + G_i + E_j + (G \times E)_{ij} + \varepsilon_{ijk} \quad (1)$$
$$\text{for } i = 1, 2, \ldots, f_1m_1f_2m_2c;\ j = 1, 2, \ldots, s;\ k = 1, 2, \ldots, r$$

where Y_{ijk} the observed value for the kth ramet of the ith genotype (clone) in jth environment; μ is the overall mean; G_i is the effect due to the ith genotype; E_j is the effect due to the jth environment; $(G \times E)_{ij}$ is the effect due to the interaction between the ith genotype and the jth environment; ε_{ijk} is the error term.

The ith genotype effect can be partitioned into full-sib family and clone within full-sib family components, that is,

$$G_i = FS_p + C/(FS)_{q/p} \quad (2)$$
$$\text{for } p = 1, 2, \ldots, f_1m_1f_2m_2;\ q = 1, 2, \ldots, c$$

where FS_p is the effect due to the pth full-sib family; $C/(FS)_{q/p}$ is the effect due to the qth clone within the pth full-sib family.

The pth full-sib family effect above is further partitioned into half-sib family components based on the female and male parents and their interaction effect component, that is,

$$FS_p = HSF_v + HSM_{v'} + (F \times M)_{vv'} \quad (3)$$
$$\text{for } v = 1, 2, \ldots, f_1f_2;\ v' = 1, 2, \ldots, m_1m_2$$

where HSF_v and $HSM_{v'}$ is the vth female and v'th male general combining ability (GCA) effect, respectively; $(FM)_{vv'}$ the specific combining ability (SCA) effect of the vth female and the v'th male parent. The female and male GCAs are further divided into female (T) and male parental population effects (D) and their corresponding individual-within-population effects [F/T or M/D], respectively, i.e.,

$$HSF_v = T_\xi + F/T_{\eta/\xi} \quad \text{for } \xi = 1, 2, \ldots, f_1;\ \eta = 1, 2, \ldots, f_2 \quad (4a)$$

$$HSM_{v'} = D_{\xi'} + M/D_{\eta'/\xi'} \quad (4b)$$
$$\text{for } \xi' = 1, 2, \ldots, m_1;\ \eta' = 1, 2, \ldots, m_2$$

The SCA may be partitioned into four interaction effects, i.e., population $T \times$ population D, population $T \times$ individual-within-population D, individual-within-population $T \times$ population D and individual-within-population $T \times$ individual-within-population D:

$$(F \times M)_{vv'} = T_\xi \times D_{\xi'} + T_\xi \times M/D_{\eta'/\xi'} + F/T_{\eta/\xi} \times D_{\xi'} + F/T_{\eta/\xi} \times M/D_{\eta'/\xi'} \quad (4c)$$
$$\text{for } \xi = 1, 2, \ldots, f_1;\ \eta = 1, 2, \ldots, f_2;\ \xi' = 1, 2, \ldots, m_1;\ \eta' = 1, 2, \ldots, m_2$$

The group of Eqs. 4a–c forms the full ANOVA model that considers all information at both the population and individual-within-population levels. The G×E interaction effect can also be partitioned into the corresponding hierarchical components, as shown by Eqs. 2–4c. If the data are balanced and all of the model effects are considered to be random, the analysis of variance is carried out with degrees of freedom and expected mean squares as given in Table 1. The mean squares are equated to the expected mean squares and solved to derive estimates of the variance components for each term in the linear models above (Kempthorne 1957).

Genetic model

Triploid progeny

Let us now first assume that the cross between the two species produces a pure triploid progeny to which two parents contribute different doses of alleles. Without loss of generality, the female parent is assumed to contribute two al-

Table 1 Analysis of variance and expected mean squares (EMS) for diploid or triploid hybrid progenies from a two-level population and individual-within-population factorial and nested mating design

Source	*df*	EMS[a]
Environment (E)	***s*-1**	$\boldsymbol{\sigma^2_\varepsilon + r\sigma^2_{GE} + rf_1m_1f_2m_2c\sigma^2_E}$
Genotype (G)	$\boldsymbol{f_1m_1f_2m_2c\text{-}1}$	$\boldsymbol{\sigma^2_\varepsilon + r\sigma^2_{GE} + rs\sigma^2_G}$
Full-sib (FS)	$f_1m_1f_2m_2\text{-}1$	$\sigma^2_W + r\sigma\ c^2_{(FS)E} + rs\sigma^2_{C/(FS)} + rcs\sigma^2_{(FS)}$
Half-sib on female (HSF)	$f_1f_2\text{-}1$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME} + rcm_1m_2\sigma^2_{(HSF)E} + rcs\sigma^2_{FM} + rcm_1m_2s\sigma^2_{(HSF)}$
Female population (T)	$f_1\text{-}1$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE} + rcm_1m_2\sigma^2_{(F/T)E} + rcm_1m_2f_2\sigma^2_{TE} + rcs\sigma^2_{(F/T)(M/D)} + rcm_2s\sigma^2_{(F/T)D} + rcf_2s\sigma^2_{T(M/D)}) + rcf_2m_2s\sigma^2_{TD} + rcm_1m_2s\sigma^2_{(F/T)} + rcm_1m_2f_2s\sigma^2_T$
Female individual (F/T)	$f_1(f_2\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcm_1m_2\sigma^2_{(F/T)E} + rcs\sigma^2_{(F/T)(M/D)} + rcm_2s\sigma^2_{(F/T)D} + rcm_1m_2s\sigma^2_{(F/T)}$
Half-sib on male (HSM)	$m_1m_2\text{-}1$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME} + rcf_1f_2\sigma^2_{(HSM)E} + rcs\sigma^2_{FM} + rcf_1f_2s\sigma^2_{(HSM)}$
Male population(D)	$m_1\text{-}1$	$\sigma^2_w + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE} + rcf_1f_2\sigma^2_{(M/D)E} + rcf_1f_2m_2\sigma^2_{DE} + rcs\sigma^2_{(F/T)(M/D)} + rcm_2s\sigma^2_{(F/T)D} + rcf_2s\sigma^2_{T(M/D)} + rcf_2m_2s\sigma^2_{TD} + rcf_1f_2s\sigma^2_{MD} + rcf_1f_2m_2s\sigma^2_D$
Male individual (M/D)	$m_1(m_2\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcf_2\sigma^2_{T(M/D)E} + rcf_1f_2\sigma^2_{(M/D)E} + rcs\sigma^2_{(F/T)(M/D)} + rcf_2s\sigma^2_{T(M/D)} + rcf_1f_2s\sigma^2_{M/D}$
Female × Male (F × M)	$(f_1f_2\text{-}1)(m_1m_2\text{-}1)$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME} + rcs\sigma^2_{FM}$
T × D	$(f_1\text{-}1)(m_1\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE} + rcs\sigma^2_{(F/T)(M/D)} + rcsm_2\sigma^2_{(F/T)D} + rcf_2s\sigma^2_{T(M/D)} + rcm_2f_2s\sigma^2_{TD}$
T × (M/D)	$m_1(f_1\text{-}1)(m_2\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcs\sigma^2_{(F/T)(M/D)} + rcm_2s\sigma^2_{(F/T)D}$
(F/T) × D	$f_1(f_2\text{-}1)(m_1\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcf_2\sigma^2_{T(M/D)E} + rcs\sigma^2_{(F/T)(M/D)} + rcf_2s\sigma^2_{T(M/D)}$
(F/T) × (M/D)	$f_1m_1(f_2\text{-}1)(m_2\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcs\sigma^2_{(F/T)(M/D)}$
Clone (C)/FS	$f_1m_1f_2m_2(c\text{-}1)$	$\sigma^2_W + rs\sigma^2_{C(FS)}$
G × E	$\boldsymbol{(f_1m_1f_2m_2c\text{-}1)(s\text{-}1)}$	$\boldsymbol{\sigma^2_\varepsilon + r\sigma^2_{GE}}$
FS × E	$(f_1m_1f_2m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(FS)E}$
HSF × E	$(f_1f_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME} + rcm_1m_2\sigma^2_{(HSF)E}$
T × E	$(f_1\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE} + rcm_1m_2f_2\sigma^2_{TE}$
(F/T) × E	$f_1(f_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcm_1m_2\sigma^2_{(F/T)E}$
HSM × E	$(m_1m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME} + rcf_1f_2\sigma^2_{(HSM)E}$
D × E	$(m_1\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE} + rcf_1f_2m_2\sigma^2_{DE}$
(M/D) × E	$m_1(m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcf_2\sigma^2_{T(M/D)E} + rcf_1f_2\sigma^2_{(M/D)E}$
F × M × E	$(f_1f_2\text{-}1)(m_1m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rcf_1m_1\sigma^2_{FME}$
T × D × E	$(f_1\text{-}1)(m_1\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE} + rcf_2\sigma^2_{T(M/D)E} + rcm_2f_2\sigma^2_{TDE}$
T × (M/D) × E	$m_1(f_1\text{-}1)(m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcf_2\sigma^2_{T(M/D)E}$
(F/T) × D × E	$f_1(f_2\text{-}1)(m_1\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E} + rcm_2\sigma^2_{(F/T)DE}$
(F/T) × (M/D) × E	$f_1m_1(f_2\text{-}1)(m_2\text{-}1)(s\text{-}1)$	$\sigma^2_W + rc\sigma^2_{(F/T)(M/D)E}$
C/FS × E	$f_1m_1f_2m_2(c\text{-}1)(s\text{-}1)$	σ^2_W
Residual (R)	$\boldsymbol{f_1m_1f_2m_2cs(r\text{-}1)}$	$\boldsymbol{\sigma^2_\varepsilon}$

[a] σ^2_ε is the variance component due to residual error. $\sigma^2_W = \sigma^2_\varepsilon + r\sigma^2_{C(FS)E}$ where $\sigma^2_{C(FS)E}$ is the variance component due to clone-within-families × environment. The sources of variance expressed in Eq. 1 are in boldface

leles and the male parent to contribute one at each locus. If a locus has two alleles, say A/A', with equal allele frequency in random breeding parental populations, then the genotypes in the populations are AA, AA', and $A'A'$ whose genotypic frequencies are 1/4, 1/2, and 1/4, respectively. Clearly, the gametic array formed in meiosis is $(1/4)AA + (1/2)AA' + (1/4)A'A'$ for the female populations generating nondisjunction during female gametogenesis (e.g., Bradshaw and Stettler 1993) and $(1/2)A + (1/2)A'$ for the male populations with a normal meiosis. If the female and male parents used for a cross are randomly sampled from such populations, respectively, the F_1 triploid hybrids within a female parental population appear in four unique genotypes whose frequencies and values can be defined as follows (see Bogyo et al. 1988; Pooni et al. 1992):

Genotype	Frequency	Genotypic value
AAA	1/8	$\mu_{AAA} = u + (3/2)a$
AAA'	3/8	$\mu_{AAA'} = u + (1/2)a + d_1$
$AA'A'$	3/8	$\mu_{AA'A'} = u - (1/2)a + d_2$
$A'A'A'$	1/8	$\mu_{A'A'A'} = u - (3/2)a$

where u is the midpoint of the two homozygotes AAA and $A'A'A'$; a is the average effect of substituting allele A for A', i.e., the additive effect; d_1 is the dominance effect of AA

to A' when AA is dominant or that of A' to AA when A' is dominant; d_2 is the dominance effect of A to $A'A'$ when A is dominant or $A'A'$ to A when $A'A'$ is dominant.

If the derivation from u is taken, the population mean of four triploid genotypes at the locus is expressed as

$$\mu = 1/8(3/2a)+3/8(1/2a+d_1)+3/8(-1/2a+d_2)+1/8(-3/2a) = 3/8d_1+3/8d_2 \quad (5)$$

and the genetic variance is

$$\sigma^2 = 1/8(3/2a)^2+3/8(1/2a+d_1)^2+3/8(-1/2a+d_2)^2 + 1/8(-3/2a)^2-(3/8d_1+3/8d_2)^2 \quad (6a)$$
$$= 3/4a^2+15/64d_1^2+15/64d_2^2+3/8ad_1-3/8ad_2 - 9/32d_1d_2$$

Let $d_1=d_2=d$ where d is the dominance effect of A to A' when A is dominant or A' to A when A' is dominant. Thus, Eq. 6a is simplified as

$$\sigma^2 = 3/4a^2+3/16d^2 \quad (6b)$$

It can be seen that the genetic variance of triploid hybrids, within female parental populations, at a locus is composed of additive variance (a^2) and dominance variance (d^2), which is regarded as the "full-sib" variance of triploid hybrids at the female parental population level. For all m independent loci governing a quantitative trait, the triploid "full-sib" variance of the F_1 generation at the female parental population level can be written as

$$\mathrm{COV}(\text{"FS"})_{\mathrm{Triploid}} = 3/4V_A+3/16V_D+9/16V_{AA}+9/64V_{AD}+9/256V_{DD}+\ldots \quad (6c)$$

where $V_A=\Sigma_{l=1}^{m} a_l^2$, total additive variance; $V_D=\Sigma_{l=1}^{m} d_l^2$, total dominance variance; $V_{AA'}$ $V_{AD'}$ and $V_{DD'}$ epistatic variances due to additive × additive, additive × dominance, and dominance × dominance interaction effects, respectively, with similar notations for higher-order interactions; $V_I=V_{AA}+V_{AD}+V_{DD}+\ldots$, total epistatic variance.

Owing to different contributions of the two parents to their F_1 triploid progeny, the triploid "half-sib" variance at the female parental population level is independently estimated from the triploid half-sib variance based on the male parental individuals. For the triploid progeny with two alleles from the female and one from the male, the mating types and frequencies of the parents and the corresponding progeny genotypes and frequencies are presented in Table 2. The mean genotypic value of triploid progeny with the common female parental populations is $1/16(3/2)a+1/8(a+1/2d_1)+1/16(1/2a+d_1)=1/4a+1/8d_1$ for AA, $1/8(1/2a+d_1)+2/8(1/2d_1+1/2d_2)+1/8(-1/2a+d_2)=1/4d_1$ $1/4d_2$ for AA', and $1/16(-1/2a+d_2)+1/8(-a+1/2d_2)+1/16(-3/2a)=-1/4a+1/8d_2$ for $A'A'$. Therefore, the covariance of triploid "half-sibs" at the female parental population level can be calculated as

$$\mathrm{COV}(\text{"HS"F})_{\mathrm{Triploid}} = 1/4(1/4a+1/8d_1)^2+1/2(1/4d_1+1/4d_2)^2 + 1/4(-1/4a+1/8d_2)^2-(3/8d_1+3/8d_2)^2 \quad (7a)$$
$$= 1/32a^2-27/256d_1^2-27/256d_2^2 - 7/32d_1d_2+1/64ad_1-1/64ad_2$$

Assuming $d_1=d_2=d$ and considering all m independent loci (see above), Eq. 7a is changed as

$$\mathrm{COV}(\text{"HS"F})_{\mathrm{Triploid}} = 1/32V_A-55/128V_D+1/1024V_{AA} \quad (7b)$$
$$-55/4096V_{AD}+3025/16384V_{DD}+\ldots \approx 1/32V_A-55/128V_D$$

where the epistatic terms are ignored because of their minor contribution to the equation. Similarly, the covariance of half-sib families with the common male parental individuals will be derived as

$$\mathrm{COV}(\mathrm{HSM})_{\mathrm{Triploid}} = 1/128V_A-243/512V_D+1/16384V_{AA} \quad (8)$$
$$-243/65536V_{AD}+59049/262144V_{DD}+\ldots \approx 1/128V_A-243/512V_D$$

Mixed progeny

For a normal diploid progeny population, the covariances of "full-sibs" and "half-sibs" at the female parental population level and of half-sibs based on the male parental individuals are, respectively, expressed as (Falconer 1989; Becker 1984)

Table 2 The genotypic types and values of triploid progeny with two-third maternal and one-third paternal contribution derived from a normal diploid mating. [a] Female×male indicates the mating between the female parental population and the male parental individuals

Parental genotype		Triploid progeny				
Female × male[a]	Frequency	AAA $3/2a$	AAA' $1/2a+d_1$	$AA'A'$ $-1/2a+d_2$	$A'A'A'$ $-3/2a$	Genotypic value
$AA\times AA$	1/16	1	0	0	0	$3/2a$
$AA\times AA'$	1/8	1/2	1/2	0	0	$a+1/2d_1$
$AA\times A'A'$	1/16	0	1	0	0	$1/2a+d_1$
$AA'\times AA$	1/8	0	1	0	0	$1/2a+d_1$
$AA'\times AA'$	2/8	0	1/2	1/2	0	$1/2d_1+1/2d_2$
$AA'\times A'A'$	1/8	0	0	1	0	$-1/2a+d_2$
$A'A'\times AA$	1/16	0	0	1	0	$-1/2a+d_2$
$A'A'\times AA'$	1/8	0	0	1/2	1/2	$-a+1/2d_2$
$A'A'\times A'A'$	1/16	0	0	0	1	$-3/2a$

$$\mathrm{COV}(\text{"FS"})_{\mathrm{Diploid}} = 1/2V_A + 1/4V_D + 1/4V_{AA} + 1/8V_{AD} + 1/16V_{DD} + \ldots \quad (9)$$

$$\mathrm{COV}(\text{"HS"F})_{\mathrm{Diploid}} = \mathrm{COV}(\mathrm{HSM})_{\mathrm{Diploid}} = 1/4V_A + 1/16V_{AA} + 1/64V_{AAA} + \ldots \approx 1/4V_A \quad (10)$$

According to Eqs. 6c and 9, the "full-sib" covariance of the mixed diploid and triploid hybrids at the female parental population level can be written as

$$\begin{aligned}\mathrm{COV}(\text{"FS"})_{\mathrm{Mixed}} &= (1-\gamma)^2\mathrm{COV}(\text{"FS"})_{\mathrm{Diploid}} + \gamma^2\mathrm{COV}(\text{"FS"})_{\mathrm{Triploid}} \\ &= 1/4(5\gamma^2-4\gamma+2)V_A + 1/16(7\gamma^2-8\gamma+4)V_D \\ &\quad + 1/16(13\gamma^2-8\gamma+4)V_{AA} + 1/64(17\gamma^2-16\gamma+8)V_{AD} \\ &\quad + 1/256(25\gamma^2-32\gamma+16)V_{DD} + \ldots\end{aligned} \quad (11)$$

where γ is the fraction of triploid to total (triploid and diploid) number within each "full-sib" family at the female parental population level and where diploidy and triploidy are assumed to be independent of each other. From Eqs. 7b and 10 as well as Eqs. 8 and 10 the "half-sibs" covariance based on the female parental population, $\mathrm{COV}(\text{"HS"F})_{\mathrm{Mixed}}$, and the half-sib covariance $[\mathrm{COV}(\mathrm{HSM})_{\mathrm{Mixed}}]$ on the male parental individuals for the mixed progeny are expressed as

$$\begin{aligned}\mathrm{COV}(\text{"HS"F})_{\mathrm{Mixed}} &= (1-\gamma)^2\mathrm{COV}(\text{"HS"F})_{\mathrm{Diploid}} + \gamma^2\mathrm{COV}(\text{"HS"F})_{\mathrm{Triploid}} \\ &\approx 1/32(9\gamma^2-16\gamma+8)V_A - 55/128\gamma^2V_D\end{aligned} \quad (12)$$

$$\begin{aligned}\mathrm{COV}(\mathrm{HSM})_{\mathrm{Mixed}} &= (1-\gamma)^2\mathrm{COV}(\mathrm{HSM})_{\mathrm{Diploid}} + \gamma^2\mathrm{COV}(\mathrm{HSM})_{\mathrm{Triploid}} \\ &\approx 1/128(33\gamma^2-64\gamma+32)V_A - 243/512\gamma^2V_D\end{aligned} \quad (13)$$

Derivation of genetic variance components

The "full-sib" family variance ($\sigma^2_{(\text{"FS"})}$) at the female population level, the female parental population (σ^2_T) and male parent-based general combining ability variance ($\sigma^2_{(HSM)}$), the specific combining ability variance between female parental populations and male parents ($\sigma^2_{TD}+\sigma^2_{T(M/D)}$), and residual genetic variance ($\sigma_Q{}^2=\sigma^2_{F/T}+\sigma^2_{(F/T)D}+\sigma^2_{(F/T)(M/D)}+\sigma^2_{C/(FS)}$) in diploid or triploid or mixed progenies derived from the linear stastistical model (see Table 1) may be expressed in terms of expected covariances among relatives (e.g., Becker 1984; Foster and Shaw 1988; Mullin and Park 1992):

$$\sigma^2_{(\text{"FS"})} = \mathrm{COV}(\text{"FS"})$$
$$\sigma^2_T = \mathrm{COV}(\text{"HS"F})$$
$$\sigma^2_{(HSM)} = \mathrm{COV}(\mathrm{HSM})$$
$$\sigma^2_{TD}+\sigma^2_{T(M/D)} = \mathrm{COV}(\text{"FS"}) - \mathrm{COV}(\text{"HS"F}) - \mathrm{COV}(\mathrm{HSM})$$
$$\sigma^2_Q = V_G - \mathrm{COV}(\text{"FS"})$$

where V_G is the total genetic variance, $V_G=V_A+V_D+V_I$. Thus, from Eqs. 6c, 7b, and 8, Eqs. 9a and 10, and Eqs. 11–13, three regular equations can be expressed as follows:

$$\sigma^2_T+\sigma^2_{(HSM)} \approx 5/128V_A - 463/512V_D \quad (14a)$$

$$\sigma^2_{TD}+\sigma^2_{T(M/D)} \approx 91/128V_A + 559/512V_D \quad (14b)$$

$$\begin{aligned}\sigma^2_Q &= 1/4V_A + 13/16V_D + 7/16V_{AA} + 55/64V_{AD} \\ &\quad + 247/256V_{DD} + \ldots \\ &= 0.3344(\sigma^2_{TD}+\sigma^2_{T(M/D)}) - 0.4445(\sigma^2_T+\sigma^2_{(HSM)}) \\ &\quad + (0.3939V_{AA} + 0.7924V_{AD} + 1.2764V_{DD} + \ldots) \\ &= 0.3344(\sigma^2_{TD}+\sigma^2_{T(M/D)}) - 0.4445(\sigma^2_T+\sigma^2_{(HSM)}) + \rho V_I\end{aligned} \quad (14c)$$

for triploid progeny,

$$\sigma^2_T+\sigma^2_{(HSM)} = 1/2V_A + 1/8V_{AA} + 1/32V_{AAA} + \ldots \approx 1/2V_A \quad (15a)$$

$$\begin{aligned}&\sigma^2_{TD}+\sigma^2_{T(M/D)} \\ &\quad = 1/4V_D + 1/8V_{AA} + 1/8V_{AD} + 1/16V_{DD} + \ldots \approx 1/4_D\end{aligned} \quad (15b)$$

$$\begin{aligned}\sigma^2_Q &= 1/2V_A + 3/4V_D + 3/4V_{AA} + 7/8V_{AD} + 15/16V_{DD} + \ldots \\ &= \sigma^2_T + \sigma^2_{(HSM)} + 3(\sigma^2_{TD}+\sigma^2_{T(M/D)}) \\ &\quad + (1/4V_{AA} + 1/2V_{AD} + 3/4V_{DD} + \ldots) \\ &= \sigma^2_T + \sigma^2_{(HSM)} + 3(\sigma^2_{TD}+\sigma^2_{T(M/D)}) + \rho V_I\end{aligned} \quad (15c)$$

for diploid progeny, and

$$\begin{aligned}&\sigma^2_T+\sigma^2_{(HSM)} \\ &\quad \approx 1/128(69\gamma^2-128\gamma+64)V_A - 463/512\gamma^2V_D\end{aligned} \quad (16a)$$

$$\begin{aligned}&\sigma^2_{TD}+\sigma^2_{T(M/D)} \\ &\quad \approx 91/128\gamma^2V_A + 1/512(687\gamma^2-256\gamma+128)V_D\end{aligned} \quad (16b)$$

$$\begin{aligned}\sigma^2_Q &= 1/4(2+4\gamma-5\gamma^2)V_A + 1/16(12+8\gamma-7\gamma^2)V_D \\ &\quad + [1/16(12+8\gamma-13\gamma^2)V_{AA} + 1/64(56+16\gamma-17\gamma^2)V_{AD} \\ &\quad + 1/256(240+32\gamma-25\gamma^2)V_{DD} + \ldots] \\ &= 1/4(2+4\gamma-5\gamma^2)V_A + 1/16(12+8\gamma-7\gamma^2)V_D + \rho V_I\end{aligned} \quad (16c)$$

for mixed diploid and triploid progeny. The solutions for the three groups of equations above can be easily determined: V_A and V_D are approximate estimates of additive and dominance variances in the three progenies, respectively; and ρV_I is a fraction of total epistatic variance in the corresponding progenies.

Discussion

An appropriate mating design offers an excellent opportunity to study population structure differentiation (Widén and Andersson 1993; Li and Margolies 1993; Kasule 1992) and to develop an efficient breeding strategy (Namkoong and Roberds 1974; Libby 1992; Park et al. 1993). Unlike most annual plants, populations of many forest tree species still exist in untapped conditions, and their genetic structure reflects a cause-effect evolutionary relationship to their environments and natural history (Critchfield 1984). Recently, an understanding of tree population structure from a quantitative or molecular genetic respective has received serious attention (Rogers et al. 1989; Namkoong and Kang 1990; Ledig and Conkle 1983; Dancik and Yeh 1983). A two-level population and individual-within-pop-

ulation mating design has two significant implications for evolutionary genetic studies and the domestication of forest trees. First, the population general and specific combining abilities of parental species, derived from Eq. 3, can provide fundamental information on the evolutionary force of population differentiation, and the type of gene action, estimated from them, at the population level influences the technique of population selection (e.g., Namkoong and Kang 1990). Population general combining ability (PGCA) of a quantitative trait may or may not parallel the tendencies of variation in the trait itself among populations (Rogers et al. 1989). If it is a property independent of the trait in evolution, PGCA will provide more powerful insights into phenomena such as clinal or ecotypic variation in tree morphology, physiology, and life-history traits than the trait itself because its expression is less affected by various temporal "developmental noises" (see Namkoong and Kang 1990). Second, by dividing general combining ability variances into population and individual-within-population components (Eqs. 4a–c), the allocation pattern of the total variation among and within populations can be estimated, as shown by morphological (Rogers et al. 1989) and allozymic traits (Dancik and Yeh 1983).

Namkoong and Kang (1990) argued that the effective loci underlying tree biological traits might be widely distributed through the genomes, although some genomic fragments contain more loci than others. Also, there is no evidence that trees have fewer quantitative trait loci (QTLs) than any other species. Thus, it is essential for tree species to keep breeding population size large enough to contain and maintain favorable alleles (e.g., Lande and Barrowclough 1987). While the effective population size seems to be sufficient for many tree species in current programs, there is no distribution adequacy of the parentages to their different geographic or ecological sources (Namkoong and Kang 1990). Population level mating would be a potent means of broadening and conserving genetic variation in trees.

This paper develops a quantitative genetic model to estimate the nature of gene action in a mixed diploid and triploid progeny. The derivation of genetic variance components is considerably more complicated when based on polyploidy rather than on diploidy (Mather and Jinks 1982). Analogous to the diploid model, the translation of the experimental components of variance given in Table 1 to causal components requires some assumptions. They include the following: (1) parents are randomly sampled from a random breeding population; (2) there are no cytoplasmic or maternal effects; (3) linkage equilibrium exists such that loci affecting a trait segregate independently, or where linkage does exist, the distribution of genotypes is as expected in the absence of linkage (Comstock et al. 1958), and (4) C-effects are assumed to be absent (Libby and Jund 1962). The first assumption, i.e., equal allele frequency at each QTL in natural populations (Bogyo et al. 1988), may lead to a biased estimate for additive variance and an overestimate for dominance variance, expressed as a function of the product of the allele frequencies at each QTL (Falconer 1989). The presence of cytoplasmic and maternally controlled genetic effects on quantitative traits clearly influences the estimates of genetic variance components (Jinks et al. 1972; Millet and Pinthus 1980; Robertson and Frey 1984). Recently, a genetic model, which takes into account maternal effects expressed in the triploid tissues of plants, has been developed by Foolad and Jones (1992). However, unequal allele frequency and linkage disequilibria, assumption (3), cannot be relaxed until a reasonably dense genetic linkage map is availabe on which the QTLs governing tree traits can be identified (Bonierbale et al. 1993; Bradshaw et al. 1994).

For mixed progeny, the fraction (γ) of triploid progenies to total progenies has been assumed to be identical within all full-sib families. However, previous breeding results show that γ is not constant but seems to follow a binomial distribution (R.F. Stettler, unpublished data). Clearly, the value of γ has to be determined for any set of families so as to modify Eqs. 11–13.

It should be noted that V_A, V_D, and V_I obtained from Eqs. 14a–c, 15a–c, and 16a–c are approximate estimates of additive, dominance, and epistatic variances in triploid, diploid, and their mixed progenies, respectively. Under the assumption of regular diploid behavior at meiosis, the first two estimators are contaminated by a portion of the epistasis caused primarily by groups of two or three interacting loci; and the last is only a fraction of epistasis contained by a clone variance component (see Mullin and Park 1992 for detailed discussion of this). Mullin and Park (1992) further demonstrated that the estimates of genetic variance components would be reasonable if the greatest portion of the total epistatic variance is due to interactions involving groups of more than two or three loci. For a complex polygenic trait such as growth, it is possible that a large portion of the epistatic variance results from the many possible interactions among large numbers of gene loci. However, if epistasis is derived only from the lowest order locus interaction, estimates of additive and dominant variance will be seriously contaminated by interloci interaction variance and ρV_I will estimate as little as one-quarter of the total epistasis. In the case of a triploid, the estimates of additive variance can be expected to be less contaminated by epistasis because of the smaller fraction involved in the half-sib covariances based on either the female or male parents (see Eqs. 7b and 8 vs. 10). However, more epistasis in the full-sib triploid covariance will lead to a more biased estimate for dominance variance. The estimate for epistatic variance is more close to its actual value in triploid progenies than in diploid progenies (see Eqs. 14c vs. 15c). If only digenic interactions are considered, 40% of additive×additive and 80% of additive×dominance epistatic variances will be included in ρV_I for triploid progenies though the coefficient of dominance×dominance epistatic component is greater than 1.0 (Eq. 14c). The corresponding percentages are much lower in diploid progenies. It should be noted that the estimate of dominance variance is based on the assumption of $d_1 = d_2$ (see Eqs. 6a–b and 7a–b). If $d_1 \neq d_2$, the estimate of genetic variance components in triploid progeny will require more generations, as shown in Pooni et al. (1992, 1993a).

An assumed mechanism of 2n female gamete formation in this model is complete nondisjunction during female gametogenesis, as found by Bradshaw and Stettler (1993) in the cross of poplars. However, they also noted that some partial nondisjunction led to the production of aneuploids. Obviously, after the specific quantitative trait loci that show nondisjunction are identified by a marker-aided QTL analysis, this model can be extended to an aneuploid case. In general, an understanding of the frequency and mechanisms of diploid gamete formation, as obtained by Stelly and Peloquin (1986), will help better apply this model.

There have been some genetic models available for the investigation of triploid-expressed quantitative characteristics, such as endosperm traits, in annual plants (Gale 1976; Mo 1987; Bogyo et al. 1988; Foolad and Jones 1992). Both Pooni et al. (1992) and Bogyo et al. (1993) extended these triploid models to a more realistic case in which crosses are segregating at a number of loci and, thus, probably display dispersion of the 'increasing' and 'decreasing' alleles in the parental lines. These new models have been applied to investigate the genetic control of triploid quantitative traits in rice (Pooni et al. 1993a, b) and maize (Bogyo et al. 1993). However, they cannot be easily applied to forest trees because they are based on a number of segregating advanced generations, which are unavailable, difficult, or even impossible to generate with long-lived forest trees. This is especially true for triploids which have low fertility and yield few if any offspring. Furthermore, such techniques are usually initiated with inbred lines to control gene frequencies (see above; Mather and Jinks 1982). Thus, their application may violate some assumptions for predominantly outcrossing tree species.

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