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Analysis of genetic effects of major genes and polygenes on quantitative traits. II. Genetic models for seed traits of crops

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Abstract Genetic models for quantitative seed traits with effects of several major genes and polygenes, as well as their *GE* interaction, were proposed. Mixed linear model approaches were suggested for analyzing the genetic models. Monte Carlo simulations were conducted to evaluate unbiasedness and efficiency for estimating fixed effects and variance components of the embryo and the endosperm models, including effects of a major gene from an unbalanced modified diallel mating design with nine parents, respectively. Simulation results showed that estimates of generalized least squares (GLS) were unbiased and efficient, while those of ordinary least squares (OLS) were almost as good as GLS. Minimum norm quadratic unbiased estimation (MINQUE) could obtain unbiased estimates of the variance components. It was also suggested that precision of MINQUE estimation would be improved with augmentation of experimental size. Data from a modified diallel design in upland cotton (*Gossypium hirsutum* L.) were used as a worked example to illustrate the parameter estimation.

Keywords Major genes · Polygenes · Genetic model for seed traits · *GE* interaction · *Glandless* gene in upland cotton

Introduction

With an increasing requirement for improving the quality of crop products, more and more attention has recently been paid on the inheritance of seed traits. Plant seed is a complex organ, which may be composed of diploid maternal tissue (testa and pericarp), diploid offspring tissue (embryo) and triploid offspring tissue (endosperm). The maternal plant also exerts its impact on seed traits via supplies of nourishment and other physiological activity substances (e.g. longevity mRNA, hormones, etc.). Moreover, cytoplasmic genomes may play a vital role in the development of seed by way of some metabolic control from cytoplasmic genes. Seed traits exhibit miscellaneous genetic behavior. The presence had been widely documented for the nuclear gene effects of embryo and endosperm (e.g. xenia effects), and cytoplasmic as well as maternally controlled genetic effects on seed traits (Garwood et al. 1970; Kondra and Stefansson 1970; Thomas and Kondra 1973; Rao and Fleming 1979; Millet and Pinthus 1980; Kumamaru et al. 1990; Tsai and Tsai 1990; Weiland 1992; Seka and Cross 1995a, b; Shi et al. 1997, 1999; Lou et al. 1998).

Since most seed traits are quantitatively inherited, employing an appropriate mathematical model is essential to draw the right genetic conclusion. Under the multiple factor hypothesis, classic genetic diploid models have been developed to estimate genetic parameters for first-degree parameters or second-degree parameters. The generations used could be descended from a cross between a pair of inbred parents by selfing, sib-mating, random mating and backcrossing, or from progeny families in a mating design with multiple parents. In consideration of the characteristics in the transmission of nuclear and cytoplasmic genes from parents to offspring, and of features in the expression of genes in various genetic systems, genetic models were proposed to analyze the genetic effects of endosperm, embryo, maternal plant and/or cytoplasm by extension of customary models for seed-related traits of generations descended from a cross between two homozygous lines (Bogyo et al. 1988; Mo 1988; Mosjidis et al. 1989; Foolad and Jones 1992; Pooni et al. 1992; Bogyo et al. 1993). Some widely used mating designs with a group of parents randomly drawn from a hypothetical population in linkage equilibrium, e.g. the nested (NC Design I) and factorial (NC Design II) designs (Comstock and Robinson 1952) and the diallel design (Griffing 1956; Kempthorne 1956; Hayman 1960; Cockerham and Weir 1977), were also extended to

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estimate genetic variation. Beavis et al. (1987) suggested a model applicable to random mating populations, and its corresponding methodology for reciprocal mating designs, when reciprocal effects can be ascribed solely to the cytoplasm. Zhu and Weir (1994a, b) and Zhu (1997, 2000) proposed genetic models including effects of embryo, endosperm and the maternal plant, as well as cytoplasm, for quantitative seed traits possessed by progeny in diallel mating designs with a random set of parents.

Some seed traits could be affected both by polygenes and by one or several major genes, which are expressed in the embryo, endosperm, the maternal plant and/or cytoplasm. A typical paradigm of such a situation is provided by protein quality maize breeding (*Zea mays* L.). The discovery concerning the biochemical effects of the *opaque-2* (abbreviated *o2*) gene (Mertz et al. 1964) aroused considerable interest among maize breeders. Some undesirable effects of the *o2* gene, however, which are also controlled by polygenes, such as lower grain weight, poor kernel appearance and susceptibility to insects and diseases, threaten to jeopardize its exploitation. Vasal et al. (1980) demonstrated that it is possible to overcome the adverse effects of the *o2* gene by selecting for favorable polygenes. Another well-known exemple is from some glandless mutants (*gl1*, *gl2*, *gl3*, *gl4*, *gl5*, etc.) of upland cotton (*Gossypium hirsutum* L.). The gossypol content and the number of glands in the seed and plant are largely controlled by alleles at those loci, while genes at other minor loci can modify the phenotypes (Lee 1962; Calhoun 1997). A desired genotype with a high gossypol plant and a low gossypol seed may be developed through screening for mutants with a promising polygenic background. Since utilization of both a major gene(s) and polygenes is usually the principal strategy, deep insight into them will contribute to making an efficient breeding scheme.

In order to investigate that kind of trait, it is necessary to extend genetic models by including effects of major genes. Mo and Xu (1994) proposed methods of identifying major gene genotypes and estimating the effects of a major gene and polygenes for endosperm characters under triploid genetic control in populations derived from a cross between two inbred parental lines. This is only applicable to triploid endosperm traits, and not to motley seed traits. The purpose of the present research is to develop genetic models, and their corresponding analysis methods, for quantitative seed traits with effects of major genes and polygenes.

Materials and methods

Assuming no interaction between single genes and polygenes, the genetic model for quantitatively inherited seed traits (Zhu and Weir 1994a, b; Zhu 1997, 2000) can be extended when the genetic difference of a quantitative trait is jointly controlled by polygenes and several single genes, which refer to any identifiable locus, not confined to major genes with larger effects. The phenotypic mean of the *k*-th genetic entry, which is derived from maternal line *i* and paternal line *j* in block *l* within environment *h*, can be represented by the following linear model:

$$\begin{aligned} y_{hijkl} &= \mu + E_h + G_{ijk} + GE_{hijk} + B_{l(h)} + e_{hijkl} \\ &= \mu + E_h + Gs_{ijk} + Gp_{ijk} + GsE_{hijk} + GpE_{hijk} \\ &\quad + B_{l(h)} + e_{hijkl}, \end{aligned} \tag{1}$$

where μ is the fixed population; E_h is the effect of macro-environment *h* (e.g. year, location, etc.), fixed or random (determined by context of the data), and is random in most genetic experiments, $E_h \sim (0, \sigma_E^2)$; $B_{l(h)} \sim (0, \sigma_B^2)$ is the random effect of block *l* within environment *h*; $e_{hijkl} \sim (0, \sigma_e^2)$ is the residual effect; G_{ijk} is the genotypic value or genetic main effect, which can be further partitioned into two components with Gs_{ijk} ascribable to single genes of the study and Gp_{ijk} to background polygenes; GE_{hijk} is the total interaction effect between genotype and environment, which can also be broken into two components, GsE_{hijk} relating to the interaction effect of $Gs_{ijk} \times E_h$, and GpE_{hijk} corresponding to the interaction effect of $Gp_{ijk} \times E_h$. Usually, the parents are a sample randomly drawn from a reference population while the genotypes of parents as single gene loci are discernible and the effects themselves are of interest. Then, the single gene effects can be treated as fixed effects and the polygenic effects as random effects.

Further, assuming: (1) a diploid organism with or without a triploid endosperm possessing regular gene segregation; (2) different sources of genetic variation (e.g. embryo, endosperm, maternal tissue or plant, and cytoplasm) independent of one another to the phenotypic variation; (3) cytoplasmic genes transmitted only through the female; (4) no higher-order interaction between alleles of endosperm polygenes; (5) no epistasis among single genes and among polygenes, Gp_{ijk}, Gs_{ijk}, GpE_{hijk} and GsE_{hijk} can be decomposed as follows. The decompositions of Gp_{ijk} and GpE_{hijk} are the same as those suggested by Zhu (1997), but some modification about the notation can be made. Gp_{ijk} is

$$Gp_{ijk} = Gop_{ijk} + Gcp_{ijk} + Gmp_{ijk}, \tag{2}$$

where Gop_{ijk} is the direct effect of nuclear polygenes of embryo or endosperm; Gcp_{ijk} is the effect due to cytoplasmic genes; Gmp_{ijk} is the effect of nuclear polygenes expressed in the maternal plant or tissue. Gop_{ijk} can be further subdivided into direct additive and dominance components, i.e.,

$$\begin{aligned} Gop_{ijk} &= \alpha(k)_i A_i + \alpha(k)_j A_j + \delta(k)_{ii} D_{ii} + \delta(k)_{jj} D_{jj} \\ &\quad + \delta(k)_{ij} D_{ij}, \end{aligned} \tag{3}$$

where A_i or $A_j \sim (0, \sigma_A^2)$ is the cumulative direct additive effect with coefficient $\alpha(k)_i$ or $\alpha(k)_j$; D_{ii}, D_{jj} or $D_{ij} \sim (0, \sigma_D^2)$ is the cumulative direct dominance effect with coefficient $\delta(k)_{ii}$, $\delta(k)_{jj}$ or $\delta(k)_{ij}$. Gcp_{ijk} can be denoted as

$$Gcp_{ijk} = \gamma(k)_i C_i + \gamma(k)_j C_j, \tag{4}$$

where C_i or $C_j \sim (0, \sigma^2_C$ is the cumulative cytoplasmic effect with coefficient $\gamma(k)_i$ or $\gamma(k)_j$. Gmp_{ijk} is also comprised of maternal additive and dominance components,

$$\begin{aligned} Gmp_{ijk} &= \alpha_m(k)_i Am_i + \alpha_m(k)_j Am_j + \delta_m(k)_{ii} Dm_{ii} \\ &\quad + \delta_m(k)_{jj} Dm_{jj} + \delta_m(k)_{ij} Dm_{ij}, \end{aligned} \tag{5}$$

where Am_i or $Am_j \sim (0, \sigma_{Am}^2)$ is the maternal additive effect with coefficient $\alpha_m(k)_i$ or $\alpha_m(k)_j$; Dm_{ii}, Dm_{jj}, or $Dm_{ij} \sim (0, \sigma_{Dm}^2)$ is the maternal dominance effect with coefficient $\delta_m(k)_{ii}$, $\delta_m(k)_{jj}$ or $\delta_m(k)_{ij}$.

The coefficients for parents, F_1, F_2, two backcrosses to both parents (BC_1 and BC_2), and two reciprocal backcrosses (RBC_1 and RBC_2) are shown in Table 1. Similarly, the interaction between Gp_{ijk} and E_h consists of three components,

$$GpE_{hijk} = GopE_{hijk} + GcpE_{hijk} + GmpE_{hijk}, \tag{6}$$

where $GopE_{hijk}$ is the interaction between Gop_{ijk} and E_h, $GcpE_{hijk}$ is the interaction between Gcp_{ijk} and E_h, and $GmpE_{hijk}$ is the interaction between Gmp_{ijk} and E_h, respectively. $GopE_{hijk}$ can also be further subdivided as

$$\begin{aligned} GopE_{hijk} &= \alpha(k)_i AE_{hi} + \alpha(k)_j AE_{hj} + \delta(k)_{ii} DE_{hii} \\ &\quad + \delta(k)_{jj} DE_{hjj} + \delta(k)_{ij} DE_{hij}, \end{aligned} \tag{7}$$

Table 1 Coefficients of polygenic effects for commonly used mating types

k[a]	*Gop* for embryo					*Gop* for endosperm					*Gcp*		*Gmp*				
	$\alpha(k)_i$	$\alpha(k)_j$	$\delta(k)_{ii}$	$\delta(k)_{jj}$	$\delta(k)_{ij}$	$\alpha(k)_i$	$\alpha(k)_j$	$\delta(k)_{ii}$	$\delta(k)_{jj}$	$\delta(k)_{ij}$	$\gamma(k)_i$	$\gamma(k)_j$	$\alpha_m(k)_i$	$\alpha_m(k)_j$	$\delta_m(k)_{ii}$	$\delta_m(k)_{jj}$	$\delta_m(k)_{ij}$
0	2	0	1	0	0	3	0	3	0	0	1	0	2	0	1	0	0
1	1	1	0	0	1	2	1	1	0	2	1	0	2	0	1	0	0
2	1	1	0.25	0.25	0.5	1.5	1.5	1	1	1	1	0	1	1	0	0	1
3	1.5	0.5	0.5	0	0.5	2	1	1.5	0.5	1	1	0	1	1	0	0	1
4	0.5	1.5	0	0.5	0.5	1	2	0.5	1.5	1	1	0	1	1	0	0	1
5	1.5	0.5	0.5	0	0.5	2.5	0.5	2	0	1	1	0	2	0	1	0	0
6	0.5	1.5	0	0.5	0.5	0.5	2.5	0	2	1	0	1	0	2	0	1	0

[a] Type of genetic entry, k, designates: 0 = parent (P_i), 1 = F_{1ij} ($P_i \times P_j$), 2 = F_{2ij}, 3 = BC_1 ($F_{1ij} \times P_i$), 4 = BC_2 ($F_{1ij} \times P_j$), 5 = RBC_1 ($P_i \times F_{1ij}$), and 6 = RBC_2 ($P_j \times F_{1ij}$), respectively

Table 2 Coefficients of single-gene genotype effects for commonly used mating types

k[a]	*Gos*				*Gms*		
	$\tau(k)_{ii}$	$\tau(k)_{ij}$	$\tau(k)_{ji}$	$\tau(k)_{jj}$	$\tau_m(k)_{ii}$	$\tau_m(k)_{jj}$	$\tau_m(k)_{ij}$
0	1	0	0	0	1	0	0
1	0	1	0	0	1	0	0
2	0.25	0.25	0.25	0.25	0	0	1
3	0.5	0	0.5	0	0	0	1
4	0	0.5	0	0.5	0	0	1
5	0.5	0.5	0	0	1	0	0
6	0	0	0.5	0.5	0	1	0

[a] Type of genetic entry, k, is the same as in Table 1

where AE_{hi} or $AE_{hj} \sim (0, \sigma_{AE}^2)$ is the direct additive by environment interaction effect; DE_{hii}, DE_{hjj} or $DE_{hij} \sim (0, \sigma_{DE}^2)$ is the direct dominance by environment interaction effect. $GcpE_{hijk}$ can be written as

$$GcpE_{hijk} = \gamma(k)_i CE_{hi} + \gamma(k)_j CE_{hj}, \tag{8}$$

where CE_{hi} or $CE_{hj} \sim (0, \sigma_{CE}^2)$ is the interaction of $C_i \times E_h$ or $C_j \times E_h$. $GmpE_{hijk}$ can be also decomposed as

$$\begin{aligned} GmpE_{hijk} = {} & \alpha_m(k)_i AmE_{hi} + \alpha_m(k)_j AmE_{hj} \\ & + \delta_m(k)_{ii} DmE_{hii} + \delta_m(k)_{jj} DmE_{hjj} \\ & + \delta_m(k)_{ij} DmE_{hij}, \end{aligned} \tag{9}$$

where AmE_{hi} or $AmE_{hj} \sim (0, \sigma_{AmE}^2)$ is the interaction of $Am_i \times E_h$ or $A_{mj} \times E_h$; DmE_{hii}, DmE_{hjj} or $DmE_{hij} \sim (0, \sigma_{DmE}^2)$ is the interaction of $D_{mii} \times E_h$, $D_{mjj} \times E_h$ or $D_{mij} \times E_h$.

The genotypic effect of single genes Gs_{ijk} can be partitioned as

$$Gs_{ijk} = Gos_{ijk} + Gms_{ijk}, \tag{10}$$

where Gos_{ijk} is the genetic main effect of the embryo or endosperm genotype attributable to single nuclear loci and Gms_{ijk} is the maternal genetic effect, respectively. Gos_{ijk} with

$$Gos_{ijk} = \sum_s \left[\tau(k)_{ii} S_{sii} + \tau(k)_{jj} S_{sjj} + \tau(k)_{ij} S_{sij} + \tau(k)_{ji} S_{sji} \right], \tag{11}$$

where S_{sii}, S_{sjj}, S_{sij} or S_{sji} is the genotypic effect of embryo or endosperm at locus s, with coefficient $\tau(k)_{ii}$, $\tau(k)_{jj}$, $\tau(k)_{ij}$ or $\tau(k)_{ji}$. $S_{sij} = S_{sji}$ for the embryo. Gms_{ijk} can be expressed as

$$Gms_{ijk} = \sum_s \left[\tau_m(k)_{ii} Sm_{sii} + \tau_m(k)_{jj} Sm_{sjj} + \tau_m(k)_{ij} Sm_{sij} \right], \tag{12}$$

where Sm_{sii}, Sm_{sjj} or Sm_{sij} is the effect of maternal genotype at locus s with coefficient $\tau_m(k)_{ii}$, $\tau_m(k)_{jj}$ or $\tau_m(k)_{ij}$.

The coefficients of the various generations are presented in Table 2. In view of flexibility and generality (e.g. multiple allelism), genetic effects of single genes are expressed as genotypic effects here since they provide the ultimate information and the first hand interpretation of genetic meaning. If necessary, additive and dominance effects or other parameters at a given locus can readily be parameterized following the traditional line. Correspondingly, GsE_{hijk} can be also decomposed as

$$GsE_{hijk} = GosE_{hijk} + GmsE_{hijk}, \tag{13}$$

where $GosE_{hijk}$ is the interaction effect between Gos_{ijk} and E_h, with

$$\begin{aligned} GosE_{hijk} = \sum_s \big[& \tau(k)_{ii} SE_{hsii} + \tau(k)_{jj} SE_{hsjj} + \tau(k)_{ij} SE_{hsij} \\ & + \tau(k)_{ji} SE_{hsji} \big], \end{aligned} \tag{14}$$

where SE_{hsii}, SE_{hsjj}, SE_{hsij}, or $SE_{hsji} \sim (0, \sigma_{SE}^2)$ is the interaction of $S_{sii} \times E_h$, $S_{sjj} \times E_h$, $S_{sij} \times E_h$ or $S_{sji} \times E_h$; $GmsE_{hijk}$ is the interaction effect between Gms_{ijk} and E_h, with

$$\begin{aligned} GmsE_{hijk} = \sum_s \big[& \tau_m(k)_{ii} SmE_{hsii} + \tau_m(k)_{jj} SmE_{hsjj} \\ & + \tau_m(k)_{ij} SmE_{hsij} \big], \end{aligned} \tag{15}$$

where SmE_{hsii}, SmE_{hsjj} or $SmE_{hsij} \sim (0, \sigma_{SmE}^2)$ is the interaction of $Sm_{sii} \times E_h$, $Sm_{sjj} \times E_h$ or $Sm_{sij} \times E_h$.

Considering a locus with two alleles, single gene effects can be expressed as direct additive (a), direct dominance (d), maternal additive (a_m) and maternal dominance (d_m), in line with the conventional usage. For embryo traits, the genotypic value and GE interaction effect of parent P_i ($k = 0$) are

$$G_{ii0} = x_{ii} a + x_{ii} a_m + 2A_i + D_{ii} + 2Am_i + Dm_{ii} + C_i$$

$$\begin{aligned} GE_{hii0} = {} & x_{ii} aE_h + x_{ii} a_m E_h + 2AE_{hi} + DE_{hii} + 2AmE_{hi} \\ & + DmE_{hii} + CE_{hi}, \end{aligned}$$

those of F_{1ij} ($k = 1$) from maternal parent $i \times$ paternal parent j are

$$\begin{aligned} G_{ij1} = {} & x_{ij} a + z_{ij} d + x_{ii} a_m + A_i + A_j + D_{ij} + 2Am_i \\ & + Dm_{ii} + C_i \end{aligned}$$

$$GE_{hij1} = x_{ij}aE_h + z_{ij}dE_h + x_{ii}a_mE_h + AE_{hi} + AE_{hj} + DE_{hij} + 2AmE_{hi} + DmE_{hii} + CE_{hi},$$

those of F_{2ij} (k = 2) selfed from F_{1ij} are

$$G_{ij2} = x_{ij}a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + A_i + A_j + 0.25D_{ii} + 0.25D_{jj} + 0.5D_{ij} + Am_i + Am_j + Dm_{ij} + C_i$$

$$GE_{hij2} = x_{ij}aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + AE_{hi} + AE_{hj} + 0.25DE_{hii} + 0.25DE_{hjj} + 0.5DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi},$$

those of BC_{1ij}(k = 3) ($F_{1ij} \times P_i$) are

$$G_{ij3} = 0.5(x_{ii} + x_{ij})a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + 1.5A_i + 0.5A_j + 0.5D_{ii} + 0.5D_{ij} + Am_i + Am_j + Dm_{ij} + C_i$$

$$GE_{hij3} = 0.5(x_{ii} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + 1.5AE_{hi} + 0.5AE_{hj} + 0.5DE_{hii} + 0.5DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi},$$

those of BC_{2ij} (k = 4) ($F_{1ij} \times P_j$) are

$$G_{ij4} = 0.5(x_{jj} + x_{ij})a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + 0.5A_i + 1.5A_j + 0.5D_{jj} + 0.5D_{ij} + Am_i + Am_j + Dm_{ij} + C_i,$$

$$GE_{hij4} = 0.5(x_{jj} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + 0.5AE_{hi} + 1.5AE_{hj} + 0.5DE_{hjj} + 0.5DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi},$$

those of RBC_{1ij} (k=5) ($P_i \times F_{1ij}$) are

$$G_{ij5} = 0.5(x_{ii} + x_{ij})a + 0.5z_{ij}d + x_{ii}a_m + 1.5A_i + 0.5A_j + 0.5D_{ii} + 0.5D_{ij} + 2Am_i + Dm_{ii} + C_i$$

$$GE_{hij5} = 0.5(x_{ii} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ii}a_mE_h + 1.5AE_{hi} + 0.5AE_{hj} + 0.5DE_{hii} + 0.5DE_{hij} + 2AmE_{hi} + DmE_{hii} + CE_{hi},$$

and those of RBC_{2ij} (k = 6) ($P_j \times F_{1ij}$) are

$$G_{ij6} = 0.5(x_{jj} + x_{ij})a + 0.5z_{ij}d + x_{jj}a_m + 0.5A_i + 1.5A_j + 0.5D_{jj} + 0.5D_{ij} + 2Am_j + Dm_{jj} + C_j$$

$$GE_{hij6} = 0.5(x_{jj} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{jj}a_mE_h + 0.5AE_{hi} + 1.5AE_{hj} + 0.5DE_{hjj} + 0.5DE_{hij} + 2AmE_{hj} + DmE_{hjj} + CE_{hj}$$

where $aE_h \sim (0, \sigma^2_{aE})$, $dE_h \sim (0, \sigma^2_{dE})$, $a_mE_h \sim (0, \sigma^2_{a_mE})$ and $d_mE_h \sim (0, \sigma^2_{d_mE})$; x_{ij} and z_{ij} are indicator variables with

$$x_{ij} = \begin{cases} 1 & \textit{when both parent i and j carry the given allele} \\ -1 & \textit{when both parent i and j carry the other allele} \\ 0 & \textit{otherwise}, \end{cases}$$

and

$$z_{ij} = \begin{cases} 1 & \textit{when parent i and j carry different alleles} \\ 0 & \textit{otherwise}. \end{cases}$$

For endosperm traits,

$$G_{ii0} = 1.5x_{ii}a + x_{ii}a_m + 3A_i + 3D_{ii} + 2Am_i + Dm_{ii} + C_i$$

$$GE_{hii0} = 1.5x_{ii}aE_h + x_{ii}a_mE_h + 3AE_{hi} + 3DE_{hii} + 2AmE_{hi} + DmE_{hii} + CE_{hi}$$

$$G_{ij1} = (0.5x_{ii} + x_{ij})a + z_{ij}d + x_{ii}a_m + 2A_i + A_j + D_{ii} + 2D_{ij} + 2Am_i + Dm_{ii} + C_i$$

$$GE_{hij1} = (0.5x_{ii} + x_{ij})aE_h + z_{ij}dE_h + x_{ii}a_mE_h + 2AE_{hi} + AE_{hj} + DE_{hii} + 2DE_{hij} + 2AmE_{hi} + DmE_{hii} + CE_{hi}$$

$$G_{ij2} = 1.5x_{ij}a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + 1.5A_i + 1.5A_j + D_{ii} + D_{jj} + D_{ij} + Am_i + Am_j + Dm_{ij} + C_i$$

$$GE_{hij2} = 1.5x_{ij}aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + 1.5AE_{hi} + 1.5AE_{hj} + DE_{hii} + DE_{hjj} + DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi}$$

$$G_{ij3} = (0.5x_{ii} + x_{ij})a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + 2A_i + A_j + 1.5D_{ii} + 0.5D_{jj} + D_{ij} + Am_i + Am_j + Dm_{ij} + C_i$$

$$GE_{hij3} = (0.5x_{ii} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + 2AE_{hi} + AE_{hj} + 1.5DE_{hii} + 0.5DE_{hjj} + DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi}$$

$$G_{ij4} = (0.5x_{jj} + x_{ij})a + 0.5z_{ij}d + x_{ij}a_m + z_{ij}d_m + A_i + 2A_j + 0.5D_{ii} + 1.5D_{jj} + D_{ij} + Am_i + Am_j + Dm_{ij} + C_i$$

$$GE_{hij4} = (0.5x_{jj} + x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ij}a_mE_h + z_{ij}d_mE_h + AE_{hi} + 2AE_{hj} + 0.5DE_{hii} + 1.5DE_{hjj} + DE_{hij} + AmE_{hi} + AmE_{hj} + DmE_{hij} + CE_{hi}$$

$$G_{ij5} = (x_{ii} + 0.5x_{ij})a + 0.5z_{ij}d + x_{ii}a_m + 2.5A_i + 0.5A_j + 2D_{ii} + D_{ij} + 2Am_i + Dm_{ii} + C_i$$

$$GE_{hij5} = (x_{ii} + 0.5x_{ij})aE_h + 0.5z_{ij}dE_h + x_{ii}a_mE_h + 2.5AE_{hi} + 0.5AE_{hj} + 2DE_{hii} + DE_{hij} + 2AmE_{hi} + DmE_{hii} + CE_{hi}$$

$$G_{ij6} = (x_{jj} + 0.5x_{ij})a + 0.5z_{ij}d + x_{jj}a_m + 0.5A_i + 2.5A_j + 2D_{jj} + D_{ij} + 2Am_j + Dm_{jj} + C_j$$

$$GE_{hij6} = (x_{jj} + 0.5x_{ij})aE_h + 0.5z_{ij}dE_h + x_{jj}a_mE_h + 0.5AE_{hi} + 2.5AE_{hj} + 2DE_{hjj} + DE_{hij} + 2AmE_{hj} + DmE_{hjj} + CE_{hj}$$

The additive effect (a_e) measuring the average effect of allele substitution, and dominance effects (d_{e1} and d_{e2}) reflecting the deviations of two heterozygotes from their expected values without dominance, can also be constructed in the triploid form for reduced endosperm models without maternal effects of single gene(s).

Mixed linear model approaches can be used to analyze the above model. The statistical analysis is similar to that of Lou and Zhu (2001). Variance components can be estimated by methods of minimum norm quadratic unbiased estimation (MINQUE, Rao 1971), restricted maximum-likelihood estimation (REML, Paterson and Thomson 1971), or maximum-likelihood estimation (ML, Hartley and Rao 1967). Fixed effects can be estimated via the ordinary least squares (OLS or LS) method or the generalized least squares (GLS) method. By methods of the best linear unbiased pre-

diction (BLUP) (Henderson 1963), linear unbiased prediction (LUP) (Zhu and Weir 1994b) and adjusted unbiased prediction (AUP) (Zhu 1993; Zhu and Weir 1996), predicted values of random effects can be obtained. Some traditional statistical test methods (e.g. *z*-test, chi-square test) can be used for significance tests. Alternatively, the jackknife numerical re-sampling procedure (Miller 1974; Efron 1982) can be employed for estimating the sampling variance of estimated or predicted values, and then Student's *t*-test can be applied for detecting the significance of parameters.

Results

Monte Carlo Simulation

Monte Carlo simulations were conducted to evaluate the efficiency of parameter estimation based on a set of genetic entries from a modified diallel design including parents, F_1s and F_2s. Nine pure lines differing at a single locus (two allelic variants, 5 vs 4) and supposed by being randomly drawn from a reference population were used as parents. Unbalanced progeny generations of 56 combinations as well as all parents were produced following a diallel mating design with irregular missing observations. Phenotypic values in six environments each containing two randomized complete blocks were built based on the genetic models including single-locus effects for a diploid embryo trait (Model I) and for triploid endosperm traits (Model II), respectively.

The parameter setting is shown in Tables 3 and 4. Pseudo-random normal deviates with a zero mean and a unit variance were generated by the method of Kinderman and Monahan (1977). MINQUE (0/1) (Zhu and Weir 1994a) was applied to estimate variance components, while GLS and OLS were employed to estimate fixed effects. For each case, 500 simulations were run to obtain sample means of estimates, bias and Mean Squared Error (MSE).

The simulation results of bias and MSE are summarized in Table 3 for fixed effects and in Table 4 for variance components. All the absolute values of bias in Table 3 were less than 5% of the parameter values, which is the conventional criterion of unbiased estimation. As indicated, fixed effects can be estimated without bias through both GLS and OLS. MSEs of GLS appeared to be consistently smaller than those counterparts of OLS, but differences were not obvious. It could be concluded that OLS is almost equally efficient in estimating fixed effects.

All the bias of estimated variances approached zero by using MINQUE (0/1) in Model I, while there was an absolute value of bias larger than 5% of the true values in Model II, but less than 10%, which can be regarded as being well-estimated. As suggested, estimates of MINQUE (0/1) are unbiased. That a few of the MSEs were large implied that some estimates were unstable or varied largely between replications under this situation. Checking those large MSEs, they were σ_E^2, σ_{aE}^2 and $\sigma_{a_mE}^2$ in Model I, and σ_E^2, σ_{aE}^2, σ_{dE}^2 and $\sigma_{a_mE}^2$ in Model II. This may result from a small sample (only six environments). From the comparison between simulations

Table 3 Bias and MSE of fixed effects estimated by OLS and GLS for Model I and Model II

Model	Parameter[a]	True value	OLS		GLS	
			Bias	MSE	Bias	MSE
I	μ	100	–0.236	40.83	–0.263	36.83
	a	10	0.026	17.10	0.008	15.26
	d	5	–0.082	7.46	–0.087	6.04
	a_m	10	–0.138	22.31	–0.046	15.29
	d_m	5	0.163	8.11	0.105	6.33
II	μ	100	0.316	68.85	0.234	63.59
	a	10	–0.008	20.28	0.064	18.92
	d	5	–0.104	16.42	–0.170	14.03
	a_m	10	–0.009	24.60	–0.120	15.31
	d_m	5	–0.117	9.92	–0.095	6.18

[a] μ, a, d, a_m, and d_m are population mean, direct additive, direct dominance, maternal additive, and maternal dominance, respectively

Table 4 Bias and MSE of variance and covariance components estimated by MINQUE(0/1) for Model I and Model II

Parameter[a]	True value	Model I		Model II	
		Bias	MSE	Bias	MSE
σ_E^2	20	–0.437	734.87	–0.095	1,434.51
σ_A^2	20	–0.016	193.19	–0.080	316.39
σ_D^2	20	0.045	42.02	0.245	38.54
σ_{Am}^2	20	0.599	206.32	–0.387	222.93
σ_{Dm}^2	20	0.136	47.34	–0.057	38.34
σ_C^2	20	0.617	157.60	–0.526	147.34
σ_{aE}^2	20	0.003	360.70	0.373	422.07
σ_{dE}^2	20	–0.976	166.30	1.130	393.13
$\sigma_{a_mE}^2$	20	–0.158	441.25	–0.718	386.60
$\sigma_{d_mE}^2$	20	0.106	195.12	0.586	231.90
σ_{AE}^2	20	0.092	32.32	–0.451	54.57
σ_{DE}^2	20	–0.138	7.64	0.021	6.27
σ_{AmE}^2	20	–0.047	33.32	0.058	38.91
σ_{DmE}^2	20	0.059	7.19	–0.080	6.57
σ_{CE}^2	20	0.205	28.64	–0.190	25.06
σ_B^2	20	0.150	142.92	–0.204	138.26
$\sigma_{A\cdot Am}$	10	–0.125	113.67	–0.578	145.69
$\sigma_{D\cdot Dm}$	–10	–0.345	26.20	–0.367	23.15
$\sigma_{AE\cdot AmE}$	–10	0.287	18.15	–0.070	25.40
$\sigma_{DE\cdot DmE}$	10	–0.043	3.95	–0.103	3.71
σ_e^2	10	0.003	0.20	0.050	0.22

[a] Parameter represents the variance or covariance component where σ_E^2 = environment, σ_A^2 = embryo additive, σ_D^2 = embryo dominance, σ_{Am}^2 = maternal additive, σ_{Dm}^2 = maternal dominance, σ_C^2 = cytoplasm, σ_{aE}^2 = *GE* interaction of embryo additive at locus, σ_{dE}^2 = *GE* interaction of embryo dominance at locus, $\sigma_{a_mE}^2$ = *GE* interaction of maternal additive at locus, $\sigma_{d_mE}^2$ = *GE* interaction of maternal dominance at locus, σ_{AE}^2 = embryo additive *GE* interaction of polygenes, σ_{DE}^2 = embryo dominance *GE* interaction of polygenes, σ_{AmE}^2 = maternal additive *GE* interaction of polygenes, σ_{DmE}^2 = maternal dominance *GE* interaction of polygenes, σ_{CE}^2 = cytoplasm *GE* interaction, σ_B^2 = block, $\sigma_{A\cdot Am}$ = covariance between embryo additive and maternal additive, $\sigma_{D\cdot Dm}$ = covariance between embryo dominance and maternal dominance, $\sigma_{AE\cdot AmE}$ = covariance between embryo additive interaction and maternal additive interaction, $\sigma_{DE\cdot DmE}$ = covariance between embryo dominance interaction and maternal dominance interaction, and σ_e^2 = error, respectively

with different sample sizes (data not shown), all the counterparts of MSE were greatly decreased in the larger sample case. As suggested, the efficiency of MINQUE would largely enhance with increasing experimental size, and a large enough size is required to obtain a reliable estimation of variance.

Most of the MSEs of both fixed effects and variance components in Model I were a little smaller than the counterparts in Model II. That may arise because there is larger phenotypic variation in the Model II case when parameters of the variance components remain the same. Additionally, a tendency was revealed by simulations with different sample sizes towards the decrement of MSE with augmentation of sample size, and improvement on the precision of estimation through enlarging the sample will be partially offset in Model II (data not shown). To compensate for the precision of estimation, more parents and environments are needed in the genetic analysis for endosperm traits.

Worked example

The seed quality of upland cotton (*Gossypium hirsutum* L.) is heavily affected by gossypol content. Some glandless mutations can substantially reduce gossypol content and upgrade seed quality. An experiment was conducted to investigate the genetic effects of a glandless gene and background polygenes on some seed quality traits. Crosses and reciprocal crosses were made using eight varieties (six glanded vs two glandless cultivars) of upland cotton as parents in an 8 × 8 diallel design with some missing entries. The seeds of all parents, F_1s, RF_1s, F_2s, and RF_2s were obtained at Zhejiang Agricultural University Experimental Farmer (Hangzhou) in both 1994 and 1995, each with two randomized complete blocks. All the above seeds were produced by hand-pollination and collected at maturity. The number of glands, the gossypol content, the oil content and the protein content were measured. Data from this experiment were used as an example for estimating genetic parameters. As the *glandless* gene can be expressed in both plant and seed, the model for a quantitative seed trait with embryo and maternal effects at a single locus was employed to analyze the experimental data. MINQUE (0/1) (Zhu and Weir 1994a) was applied for estimating the variance components and GLS for the fixed effects. A jackknife procedure using the genotype as a resampling unit was used to test for the significance of the parameters.

The estimated results are summarized in Table 5 for the genetic main effects of the *glandless* gene, and in Table 6 for variances and covariances of the random effects. Significantly, large embryo additive and dominance effects of the *glandless* gene were detected and the *glandless* allele is almost entirely recessive to the normal type for the number of glands. Zero interaction variance implied that its influence is quite stable across different environments. Mutative genes considerably decreased the gossypol content via the embryo, and a little through the maternal plant. Both embryo and maternal effects displayed a pattern of nearly completely additive inheritance. As revealed, the influence of the *glandless* gene on gossypol content is not only exerted through direct control over metabolic pathways of the seed but also resulted from its expression in the maternal plant. There were small but significant maternal additive effects on oil content. It was suggested that gene substitution at this locus affects formation of the gland as well as gossypol and oil syntheses, but exhibits different action patterns. This was evidence that a gene will produce multiple phenotypic consequences by controlling a series of metabolic pathways. The influence of the *glandless* gene on protein content was very weak. It can be concluded that the impact of the *glandless* gene is almost in excess of protein metabolism.

It can also be concluded that the polygenic difference is abundant in the population studied for all traits evaluated from the estimated variance and covariance components (Table 6). Variation affected by polygenes was controlled both by genetic main effects and by *GE* inter-

Table 5 Estimates of population mean and genetic effects of the *glandless* gene in cotton

Parameter[a]	Number of glands[b]	Gossypol content (%)	Oil content (%)	Protein content (%)
μ	23.45**	0.455**	31.48**	29.02**
a	22.77**	0.369**	–3.44	1.82
d	15.73**	0.039	0.66	–0.76
a_m	0.74	0.075**	4.00**	–1.38
d_m	–1.23	–0.021	0.33	0.93

[a] Parameter is the same as in Table 3
[b] * and ** are significant at 0.05 and 0.01 levels, respectively

Table 6 Estimates of variance and covariance components for the number of glands, gossypol content, oil content and protein content in cotton

Parameter[a]	Number of glands[b]	Gossypol content (%) × 10^{-2}	Oil content (%)	Protein content (%)
σ_E^2	5.26**	0.791**	3.59**	9.16**
σ_A^2	0.00	0.394**	6.49**	2.60**
σ_D^2	14.84**	0.661**	2.45**	1.64**
σ_{Am}^2	24.92**	0.000	5.56**	2.25**
σ_{Dm}^2	0.00	1.261**	5.21**	0.00
σ_C^2	51.60**	0.000	0.00	4.35**
σ_{aE}^2	0.00	0.000	0.00	3.19**
σ_{dE}^2	0.00	2.481**	12.76**	0.00
σ_{amE}^2	0.00	0.000	0.00	0.00
σ_{dmE}^2	0.00	0.000	0.00	9.31**
σ_{AE}^2	18.42**	0.000	4.07**	0.00
σ_{DE}^2	11.92**	0.000	2.37**	2.71**
σ_{AmE}^2	0.00	0.744**	6.40**	0.00
σ_{DmE}^2	84.60**	1.705**	5.01*	7.02**
σ_{CE}^2	0.00	0.000	0.00	0.00
$\sigma_{A\cdot Am}$	0.00	0.000	–2.18	–1.23
$\sigma_{D\cdot Dm}$	0.00	–0.068	1.80**	0.00
$\sigma_{AE\cdot AmE}$	0.00	0.000	–3.57*	0.00
$\sigma_{DE\cdot DmE}$	1.19	0.000	–1.51*	–2.31**
σ_e^2	28.67**	1.613**	7.18	3.09**

[a] Parameter is the same as in Table 4
[b] * and** are significant at 0.05 and 0.01 levels, respectively

action effects. This indicated that expression of those polygenes depends to a great extent on specific environments. It also appeared that those traits are susceptible to influences of the environment, and residual effects with significant environmental and residual variances. The polygenes responsible for the number of glands and gossypol content seem to be expressed more in the maternal plant than in the seed. Maternal genotypes with an elite polygenic source should be prior to seed breeding. There were significantly larger direct and maternal additive main effects for oil content and protein content, and maternal additive main effects for the number of glands. The two portions of the phenotypic variation capable of being steadily transmitted from parents to their offspring will lead a high degree of resemblance between relative individuals across different environments, and can also be easily fixed in early generations. Early generation selection and sib indirect selection based on the performance in one environment will be fruitful to genetic improvement, not only in similar environments but also in other environments. The occurrence of embryo and maternal additive interactions for oil content, and embryo additive interaction for the number of glands, suggested that there were also some components of heritability and resemblance between relative individuals relied on in a particular environment. Genetic progress arising from them will localize only in similar environments to a certain extent. Negative covariance between embryo additive and maternal additive interactions suggested that specific nicking of them deserves considering the utilization of polygenes for oil content. Direct and maternal dominance main effects were highly significant for gossypol content and oil content. Maternal dominance *GE* interaction was significant for all traits studied, while direct dominance *GE* interaction was significant only except for gossypol content. As implied, heterosis might have varyied from one environment to another. Cytoplasmic effects occupied a small share of the total variation for the number of glands and the protein content. The portion of variation can also be steadily transmitted from the maternal plant to its offspring and resulted in a degree of resemblance between them.

Discussion

Manifestation of a character is the consequence of joint action by multiple factors, e.g. numerous genes and many environmental factors. It is well revealed that there are some traits whose variation may be affected by major genes with relative larger effects and by polygenes with relative smaller effects, as well as by environments. The genes responsible for the genetic difference of a trait may be expressed in different parts of the organism, tissues and cell components, and act in a distinct manner. As a result of morphological and physiological complexity, the inheritance of quantitative seed traits with some major gene effects behaves in a complicated manner. As shown in this worked example, both the *glandless* gene and polygenes are involved in the traits investigated. The *glandless* gene not only bears a pleiotropic impact on seed traits through metabolic controls from different genetic systems, such as gossypol amassment, which is tightly tied up with the emergence of glands and lipoidal metabolism, which may share some common biochemical reactions with gossypols, but also exhibits varied genetic mechanisms. Thus, genetic analysis for this kind of seed trait needs an appropriate methodology. The present genetic model for seed traits enumerates some possible effects of polygenes and any identifiable single genes. Since gene expression possesses spatial and/or temporal specificity, it is not universal that a gene can be expressed in all organs and tissues. So, the full genetic model is only a theoretical or tentative one and may not always be true. However, it is of consequence because it can provide a jumping-off point for genetic analysis as well as some promising candidates involving reduced models or submodels for specific traits. It is also vital to select a proper submodel for a given trait based on actual situations to estimate efficient genetic parameters. Although the statistical analyses are made on the assumption of independence between random effects in this worked example and simulation, data with correlated random effects can be handled by the proposed methodology,

Simulation in the present studies indicated that sample size bears a notable impact on estimating the precision and power of tests for detecting significance but with minor influence on unbiasedness. A sufficiently large sample is therefore necessary to obtain a reliable parameter estimation. A sample size of eight or more parents is reasonable when parents are regarded as a random sample from a reference population. Since a mating design usually includes multiple generations such as F_1s, BCs and F_2s for analyses of such complex seed models, the experiment size is several times larger than the general diallel analysis with the same number of parents. For many cereal crops artificial emasculation and pollination are very labor-intensive. Plant breeders usually cannot afford very large experimental sizes in conducting genetic research since seeds of F_1s and BCs should be produced by manual hybridization. Moreover, manipulation errors could become unwieldy for too-large experiments. Simulations also suggest that unbiased estimation could be obtained if five parents are selected at random for mating. If more parents are involved in mating, the estimation precision and power of tests could be increased. A rational coverage of combinations is also helpful to improve estimation under an equal experiment size. Partial diallel design and NC Design II usually hold greater efficiency than a full diallel design by increasing sampling size but not experimental size.

The genetic model in the present research is developed mostly for the case of multiple parents, especially of those randomly sampled from the population studied, and their genotypes concerning, major genes can be inferred from pedigree data or scored by morphological phenotype or biochemical methods. Genetic parameters of a reference population can be estimated by the joint analysis of several generations derived from a mating design with multiple parents. It may also easily be extend-

ed to other genetic entries. When only using two parents, with some changes about the definition of parameters, the genetic model will reduce to a traditional one for generation means, which resemble those of Pooni et al. (1992) and Foolad and Jones (1992). To estimate genetic variances, individual phenotypic values of parents and their offspring should be contained in a statistical model and the information about genetic architecture should also be added. A mixed-model approach can also be employed for statistical analysis.

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