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Models to estimate maternally controlled genetic variation in quantitative seed characters

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Summary. Estimating quantitative contributions to specific traits can be accomplished from a variety of genetic models (Mather 1949; Mather and Jinks 1971; Falconer 1981). Residual genetic effects, those beyond main and interaction effects of the embryo genotype, are often pooled under a single classification, termed maternal effects. Maternal contributions to seed-related traits can originate from various maternal sources (e.g., endosperm, testa and cytoplasm). Quantitative contributions of a maternal nature are not predictable from parental performance and effects are largely non-persistent over generations (Jinks et al. 1972). The methods used to determine maternal effects in quantitative traits often do not measure quantitative genetic parameters, while those that do are either complex or partially resolve potential contributions of individual sources of maternal effects. We present simple genetic models for estimating quantitative genetic parameters which take into account maternal effects expressed in the major seed tissues of higher plants.

Key words: Additive genetic variance – Dominance genetic variance – Maternal effects – Generation means analysis

Introduction

Most traits in higher organisms exhibit continuous variation, culminating from the action of multiple genes which individually exert small but significant effects and are further modified by the environment. Genetic improvement of such quantitative traits in plants has relied mainly upon directional changes, in response to selection, of frequencies for genes with independent and additive effects. Numerous mathematical models for the estimation of differential genotypic contributions to quantitative characters have been developed using either generation means (Mather 1949; Hayman 1958; Mather and Jinks 1971) or population variances (Comstock and Robinson 1948; Kempthorne 1957; Hayman 1960; Cockerham 1963; Gardner 1963). The presence of cytoplasmic and maternally controlled genetic effects on important quantitative traits have been documented (Ashri 1964; Bhat and Dhawan 1971; Christiansen and Lewis 1973; Tyson 1973; Rao and Fleming 1978; Millet and Pinthus 1980; Mosjidis and Yermanos 1984; Robertson and Frey 1984; Groot and Karssen 1987), but the magnitude of these genetic effects have not been rigorously estimated and, hence, their value in genetic gains is largely unknown.

Models estimating the components of generation means and variances were developed for populations of diploid organisms in which the genotype of the individual is equated to the sum of the additive and dominance effects of the embryo genotype. Because the models either ignore or fail to fully consider maternal contributions, these estimates can be biased. Even in cases where models consider additional sources of variation, they do not isolate maternal effects arising from different phenomena. For example, cytoplasmic effects may be ignored (Barnes 1968; Mather and Jinks 1971), maternal effects may be considered as a single cytoplasmic component (Tyson 1973; Mosjidis et al. 1989) or as endosperm effects alone (Huidong 1988), or the differences between reciprocal populations may be ascribed to the genotype of the maternal parent (F_1s: Christiansen and Lewis 1973; Cockerham and Weir 1977; Kohel 1980; F_2s: Knowles and Mutwakil 1963; backcrosses: Singh and Hadley 1972; Mosjidis and Yermanos 1984). For seed-re-

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lated traits, contributions can arise either from the triploid endosperm in an otherwise diploid organism (Groot and Karssen 1987; Foolad and Jones 1991) or from the effects of the diploid testa (Foolad and Jones 1991) in addition to, or interacting with those of the embryo (Yermanos and Knowles 1962; Thomas and Kondra 1973) or cytoplasmic effects (Mosjidis et al. 1989). Comparison of reciprocal generations has the advantage of simplicity, but no single comparison can unequivocally identify effects of the testa and/or endosperm with respect to a given trait, or discriminate their effects relative to other sources. A clearly quantitative estimation of gene actions attributable to each source is not possible.

We present a model to isolate the sources of maternal contributions to quantitative characters. The model contributes to current theory because traits of biological and economic significance are expressed in the maternal tissue of the seed. Moreover, it aids in the design of efficient methods to estimate genetic parameters and for their utilization in plant improvement strategies. Procedures are developed to estimate genetic parameters, both means and variances of these characters, and simultaneously determine the relative contributions of main effects from all sources (i.e., embryo, endosperm, testa/perisperm and cytoplasm). The model allows for n loci and can be easily extended to include first order interaction effects.

Models

Components of generation means

Throughout, we consider additive-dominance models with the following assumptions: (1) diploid organism with triploid endosperm present; (2) the contributing genes are unlinked with independent effects on the means and variances; (3) gene and environmental differences contribute to variation in the phenotype independently of one another; (4) different sources of genetic variation (e.g., embryo, endosperm, testa and cytoplasm) contribute independently of one another to the phenotypic variation and (5) the progenies in each generation are considered to represent random members of that generation. In describing the relationship between genotypes and expected phenotypic values we adopt the terminology of Mather and Jinks (1971).

For traits controlled by additive and dominance effects of the diploid embryo genotype, the triploid endosperm genotype (for which two-thirds of the genetic composition is from the maternal parent), the seed coat genotype (diploid tissue entirely of maternal genotype) and non-Mendelian components transmitted only with the maternal cytoplasm, the expected phenotypic value (p_i) of an individual with respect to a given character can be defined as:

$$p_i = m + [d] + [h] + [d_e] + [h_{e1}] + [h_{e2}] + [d_t] + [h_t] + [d_c],$$

where m is the mean effect (determined as the midpoint between the means of the two homozygous parents), and $[d]$ and $[h]$ represent sums of the additive and dominance effects, respectively, of the k contributing genes corresponding to embryo ($[d]$, $[h]$), endosperm ($[d_e]$, $[h_{e1}]$, $[h_{e2}]$), testa ($[d_t]$, $[h_t]$), and the cytoplasm ($[d_c]$). The model can accommodate traits for which no endospermic effect is anticipated (e.g., non-endospermic seeds) by omitting the endosperm components from the model.

Components of generation variances

The components of genetic variance (V_G) in the model for k contributing loci may be defined as follows:

$$V_G = \sum_1^k d^2 + \sum_1^k h^2 + \sum_1^k d_e^2 + \sum_1^k h_{e1}^2 + \sum_1^k h_{e2}^2 + \sum_1^k d_t^2 + \sum_1^k h_t^2$$
$$= D + H + D_e + H_{e1} + H_{e2} + D_t + H_t,$$

where Σd^2 and Σh^2 are sums of the additive and dominance variance components, respectively, assigned to the embryo (d^2, h^2), endosperm (d_e^2, h_{e1}^2, h_{e2}^2), and testa (d_t^2, h_t^2) effects. Within a given segregating generation derived from hybridization between two inbred lines, no variation in the cytoplasmic components should be observed unless both parents contribute cytoplasmic factors. Additionally, all individuals within early segregating generations (i.e., F_2 or BC_1 generation) would have an identical testa constitution and hence, no variance from this source would be expected.

Definition of the genetic parameters

For the case of disomic inheritance (e.g., embryo genotype) we adopt the terminology defined by Mather and Jinks (1971) and extend their model to describe the relationship between genotypes and expected phenotypic values to include additional sources of genetic variation. The coefficients of the genetic parameters for individual tissue effects in the generations derived from a cross between two inbred lines are shown in Table 1. If we consider two alleles, A and a, at a given locus, tissue of a diploid origin (i.e., embryo, testa) can have one of three possible genotypes: *AA*, *Aa* and *aa*. The phenotypic expression of each genotype is determined as the departure from the mid-point (m) between the two homozygous parents. For the diploid embryo, parameter d measures the departure of each homozygote from m, and parameter h measures the departure of heterozygote from m (Fig. 1 A). In the absence of dominance, the phenotypic expression of *AA* would be $m+d$, and that of *Aa* and *aa* would be m and m-d, respectively. Importantly, in the absence of domi-

Table 1. Coefficients of genetic parameters for main effects (m=mid-point, d=additive, h=dominance) attributed to embryo, endosperm, testa and cytoplasmic effects in generations derived from two inbred lines

Generation	Mating	Embryo			Endosperm			Testa[a]		Cytoplasm
		[m]	[d]	[h]	[d_e]	[h_{e1}]	[h_{e2}]	[d_t]	[h_t]	[d_c]
P_1	$P_1 \times P_1$	1	1	0	1	0	0	1	0	1
P_2	$P_2 \times P_2$	1	−1	0	−1	0	0	−1	0	−1
F_{11}	$(P_1 \times P_2)$	1	0	1	1/3	1	0	1	0	1
F_{12}	$(P_2 \times P_1)$	1	0	1	−1/3	0	1	−1	0	−1
BC_{11}	$(P_1 \times P_2)P_1$	1	1/2	1/2	1/3	0	1/2	0	1	1
BC_{12}	$(P_2 \times P_1)P_1$	1	1/2	1/2	1/3	0	1/2	0	1	−1
BC_{21}	$(P_1 \times P_2)P_2$	1	−1/2	1/2	−1/3	1/2	0	0	1	1
BC_{22}	$(P_2 \times P_1)P_2$	1	−1/2	1/2	−1/3	1/2	0	0	1	−1
RBC_{11}	$P_1(P_1 \times P_2)$	1	1/2	1/2	2/3	1/2	0	1	0	1
RBC_{12}	$P_1(P_2 \times P_1)$	1	1/2	1/2	2/3	1/2	0	1	0	1
RBC_{21}	$P_2(P_1 \times P_2)$	1	−1/2	1/2	−2/3	0	1/2	−1	0	−1
RBC_{22}	$P_2(P_2 \times P_1)$	1	−1/2	1/2	−2/3	0	1/2	−1	0	−1
F_{21}	F_{11} selfed	1	0	1/2	0	1/4	1/4	0	1	1
F_{22}	F_{12} selfed	1	0	1/2	0	1/4	1/4	0	1	−1
F_{31}	F_{21} selfed	1	0	1/4	0	1/8	1/8	0	1/2	1
F_{32}	F_{22} selfed	1	0	1/4	0	1/8	1/8	0	1/2	−1
F_{41}	F_{31} selfed	1	0	1/8	0	1/16	1/16	0	1/4	1
F_{42}	F_{32} selfed	1	0	1/8	0	1/16	1/16	0	1/4	−1

[a] Testa = maternal tissue derived from the integument of the ovule

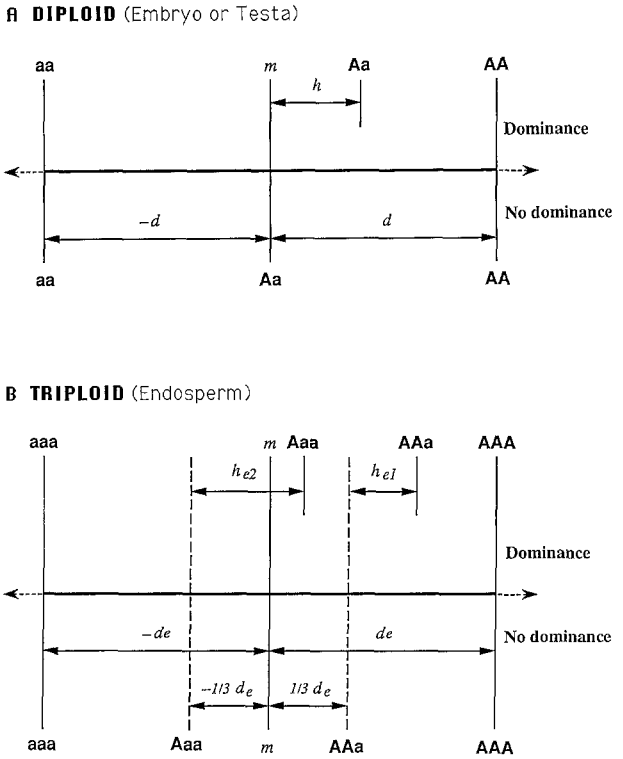

Fig. 1 A, B. Additive (d) and dominance (h) genetic parameters. The possible genotypes of a single locus *A-a* in diploid (**A**) and triploid (**B**) tissue. Deviations are scales relative to the midpoint (m) between the two parents P_1 and P_2. Within each tissue type (**A** or **B**) the deviations shown in the *lower portion* reflect the absence, and the *upper portion* the presence of dominance, respectively

nance and genotype × environment interactions, the heterozygote's expression of the character is midway between the expression of the two homozygotes (Fig. 1 A, bottom). With dominance present, the phenotypic expression of the heterozygote is $m+h$ (Fig. 1 A, top). The magnitude of h will change depending upon the degree and direction of dominance. If dominance is complete, h will be equal to d. When more than one locus is involved, the expected phenotypic expression of each genotype is determined as the sum of the effects at the individual loci (Mather and Jinks 1977). Common to disomic inheritance, three parameters will also describe the phenotypic expression of the testa genotypes: genotype *AA* departs from the midpoint (m) by d_t (testa additive effects) and *aa* departs from m by $-d_t$. The heterozygote, *Aa*, departs from m by h_t (testa dominance effects) if dominance is present. It is important to point out that the values of d_t and h_t are not necessarily equal to those of d and h, respectively.

Considering the same two alleles, *A* and *a*, the triploid endosperm can have one of four possible genotypes at this locus: *AAA, AAa, Aaa* and *aaa*. The phenotypic expression of each genotpye is determined by four parameters: m, the mid-point between the two homozygote parents (*AAA* and *aaa*); d_e endosperm additive effects, which measures the additive departure of each genotype from m, and; h_{e1} and; h_{e2} endosperm dominance effects, which measure the deviation of the phenotype of each heterozygous individual (*AAa* and *Aaa*, respectively) from their expected additive phenotypic values without dominance. The genetic effects are scaled as shown in

Table 2. Components of variation in the parents and F_1, F_2, F_3, F_4 and reciprocal backcross generations derived from a cross between two inbred lines

Statistic	Embryo		Endosperm			Testa		Environmental		Sampling variance[a]
	D	H	D_e	H_{e1}	H_{e2}	D_t	H_t	E_w	E_b	
V_P	0	0	0	0	0	0	0	1	0	0
V_{F1}	0	0	0	0	0	0	0	1	0	0
V_{BC1}	1/4	1/4	4/9	0	1/4	0	0	1	0	0
V_{BC2}	1/4	1/4	4/9	1/4	0	0	0	1	0	0
V_{RBC1}	1/4	1/4	1/9	1/4	0	0	0	1	0	0
V_{RBC2}	1/4	1/4	1/9	0	1/4	0	0	1	0	0
V_{F2}	1/2	1/4	5/9	3/16	3/16	0	0	1	0	0
V_{F3}	3/4	3/16	7/9	7/64	7/64	1/2	1/4	1	0	0
V_{bF3}	1/2	1/16	1/2	1/64	1/64	1/2	1/4	0	1	1/n V_{wF3}
V_{wF3}	1/4	1/8	5/18	3/32	3/32	0	0	1	0	0
V_{F4}	7/8	7/64	8/9	15/256	15/256	3/4	3/16	1	0	0
V_{bF4}	3/4	3/64	3/4	3/256	3/256	3/4	3/16	0	1	1/n V_{wF4}
V_{1F4}	1/2	1/64	1/2	1/256	1/256	1/2	1/16	0	0	1/n' V_{2F4}
V_{2F4}	1/4	1/32	1/4	1/128	1/128	1/4	1/8	0	1	1/n V_{wF4}
V_{wF4}	1/8	1/16	5/36	3/64	3/64	0	0	1	0	0

V_{bF3}, V_{wF3}, V_{bF4} and V_{wF4} are the between and within family variances in the F_3 and F_4 generations, respectively. V_{1F4} is the variance between F_4 groups; each group traces back to one F_2 individual. V_{2F4} is the variance between families within F_4 groups

[a] n, no. of individuals per family; n', no. of families per group

Fig. 1 B. In contrast to the diploid condition, the additive phenotypic values of the heterozygous triploid genotypes are not equal to the midparental value when dominance is absent (Fig. 1 B, bottom). In the absence of dominance, the phenotypic value of the *AAA* genotype would be $m+d_e$, and the values of *AAa*, *Aaa*, and *aaa* would be $m+(1/3)d_e$, $m-(1/3)d_e$, and $m-d_e$, respectively. Two dominance parameters should be considered in the model where the heterozygotes *AAa* and *Aaa* deviate from their respective expected additive phenotypic values under the assumption of no dominance by increments (or decrements) of h_{e1} and h_{e2} (Fig. 1 B, top). The realized values for *AAa* and *Aaa* would then be defined as $m+(1/3)d_e+h_{e1}$ and $m-(1/3)d_e+h_{e2}$, respectively. When h_{e1} and h_{e2} are equal, the phenotypic difference between the two heterozygotes will be determined by the additive effect of allele *A*. In the case of full dominance, where *AAA*, *AAa* and *Aaa* exhibit similar phenotypes, the estimated value for h_{e2} will be larger than h_{e1}. Huidong (1988) formulated a model to study endosperm effects on major storage products elaborated in the endosperm. In deriving the coefficients for additive endosperm effects, he selected a different base line which generated ratios different from those presented here (Table 1). Theoretically both derivations are correct when adjusted for the differing base lines.

Cytoplasmic components are assumed to have a constant additive effect (d_c) that shifts the genotypic value of an individual according to the cytoplasm associated with the genotype. The cytoplasm associated with the nuclear genotype *AA* is assumed to have a positive effect (d_c) and that associated with genotype *aa*, a negative effect ($-d_c$). In describing this model we assume that the cytoplasmic contributions are due solely to maternal cytoplasm, which is the most common case in angiosperms (Tilney-Bassett 1978).

Estimating genetic parameters

The procedure for estimating the genetic parameters in the model can be the joint scaling test (Cavalli 1952), using the generation means available, by a matrix approach as described by Mather and Jinks (1971). Because the observed generation means do not have the same error variances, the observed and the expected means are, therefore, weighted by the inverse of the variance of the generation means. The adequacy of the genetic model is then tested by comparing the expected and the observed mean values using a X^2 test. The degree of freedom for the X^2 test would be the number of generation means used in the analysis minus the number of parameters estimated. The significance of each parameter can be tested by dividing the estimates by their standard errors,

derived from the variance-covariance matrix, and comparing the result with the t values for appropriate degree of freedom and probability levels.

A faster estimation procedure is by weighted least square (WLS) multiple regression analysis. In this method, which can be performed on any computer with regression analysis capabilities, the generation means are used as the dependent variable and the coefficients of the genetic parameters as the independent variables. Outputs include the least square estimates of the genetic parameters, their standard deviations and t values and the predicted generation means for the model tested. The residual sums of squares in this analysis is equivalent to the weighted X^2 and, therefore, can be used to test the adequacy of the model. The proportion of the total variation among the generation means explained by the genetic model as well as the percentage contribution of each parameter to the genotype sums of squares may also be obtained from the regression output. The speed of the procedure allows one to quickly test various genetic models and identify the most adequate model.

Estimating components of genetic and environmental variances

The components of the genetic and the environmental variances for several generations derived from hybridization between two inbred lines are shown in Table 2. In deriving the coefficients for the components of genetic variances due to endosperm and testa sources, we have followed Mather and Jinks' approach (1971, 1977) for the diploid condition. Simultaneous estimation and testing of the components of genetic variances attributed to the embryo, endosperm and the seed coat as well as the environmental contributions (total of nine components) requires testing of at least ten different generations. Pedigrees maintained for inbred populations derived from individual F_2 plants by selfing provides greater observational resolution of components of variance. The genetic and environmental variances in the F_3 and F_4 generations can be partitioned into the between (i.e., V_{bF3}, V_{bF4}) and within (V_{wF3}, V_{wF4}) family variances. The between family variances in the F_4 generation are further partitioned into those attributed to between F_4 groups (V_{1F4}) – each group consists of F_4 families originated from one F_3 family that trace back to one F_2 individual – and those attributed to between families within groups (V_{2F4}) (Mather and Jinks 1971). This partitioning reduces the number of generations that must be grown in order to estimate and test the various components of genetic variances. This should be evident since we can generate two equations for the F_3 generation and three equations for the F_4 generation. In a similar manner one could calculate four types of variance in the F_5 generation.

If the number of components to be estimated is equal to the number of equations available, one can estimate the components by a perfect fit solution. Unfortunately, neither the standard deviations of the estimate can be calculated nor can the adequacy of the model be tested in such cases. The approximate significance of the variance components can be tested, however, by employing the square root of the theoretical variance of the variance. The theoretical variance of a variance V may be calculated as $2V^2/N$, where N is the number of degrees of freedom from which V is estimated (Mather and Jinks 1977).

The variance components can be more precisely estimated by weighted least square regression analysis (Hayman 1960). This procedure requires that the number of equations (generation variances) be greater than the number of variance components being estimated. The estimation procedure is essentially that used for the estimation of the genetic parameters using generation means. The only notable difference would be in estimating the weights for different generation variances. Procedure for calculating the weights and the components of generation variances by the WLS regression technique are available (Mather and Jinks 1977).

Discussion

For seed-related traits the phenotype of the seed may be determined by the genotype of its embryo, maternal tissues and cytoplasm. Maternal contributions arising from the triploid endosperm, especially in large endospermic seeds (e.g., cereals and certain legumes), may be substantial. Identification of the contributing sources of genetic variation and the type of gene actions involved will assist in the selection of the most appropriate breeding strategy. The genetic model presented here enables one not only to precisely discriminate effects exerted by the zygotic genotype of the individuals from those attributable to the individual sources of maternal contributions, but also to determine the nature of the gene actions involved. We have considered the 18 populations described above: P_1, P_2, RF_1's and the eight BC's and the reciprocal F_2's, F_3's and F_4's. Coefficients of the genetic parameters describing these generations are listed in Table 1. Simultaneous estimation of all nine parameters of the main model is possible and requires only ten generations. The model considers applied situations which arise in pedigree breeding programs using self-fertilization. The generations indicated are consistent with self-pollinated crops where repeated artificial crossing is costly and therefore should be minimized, yet are sufficient to provide accuracy in the parameter estimations. In contrast, the endosperm model of Huidong (1988) includes 27 generations, many of which are difficult to generate in self-pollinated crops and their inclusion would not signif-

icantly improve the reliability of the estimation process. In the present model, even with fewer number of generations, analysis of all components can be accommodated by pairwise inclusion of the tissue effects, and the model reiterated in all possible combinations until the best fit is achieved.

The coefficients in Table 1 can also be used to guide the selection of the most appropriate generations to obtain information about specific sources. For example, information concerning the role of endosperm can be most efficiently obtained through comparison of the reciprocal F_1's and BC's, whereas persistence of the cytoplasmic effects over generations can best be tested by comparing the reciprocal differences in F_2's, F_3's and F_4's. To eliminate possible scalar effects, the mean differences between reciprocal crosses should be standardized by the generation means. Application of this model prohibits any confounding of the various effects and variances originating from the different sources. If simple models including main effects alone do not provide adequate fit to the data as measured by the X^2 test, the model can be extended to include first order non-allelic (epistatic) interaction components. Derivation of the epistatic coefficients is accomplished by simply multiplying the respective coefficients of the main effects scaled from the midpoint (Mather and Jinks 1971). Simultaneous estimation of the main and epistatic effects would require the development of many generations. It is advisable, before proceeding to the evaluation of more complex models, to determine if the adequacy of the simple model (i.e., main effects alone) can be satisfactorily improved by suitable transformation of the data (Mather and Jinks 1977).

Because we are considering discrete contributions from different sources of genotypes (i.e., embryo, endosperm and/or testa) among populations, it is important to consider that these sources may also interact with each other. Several reports have indicated between-source interactive contributions to seed characters (Beale and Knowles 1978; Kondra and Stefanson 1970; Robertson and Frey 1984). In such situations, appropriate interaction coefficients can easily be derived and included in the model for analysis. Further, if biparental transmission of the cytoplasmic organelles has been reported for the species under study, appropriate cytoplasmic parameters can be included in the model (Jinks et al. 1972; Mosjidis et al. 1989).

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