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A theoretical model for quantitatively inherited traits influenced by nuclear-cytoplasmic interactions*

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Summary. Cytoplasmic genes of crop species exhibit non-Mendelian inheritance and affect quantitative traits such as biomass and grain yield. Photosynthesis and respiration are physiological processes responsible, in part, for the expression of such quantitative traits and are regulated by enzymes encoded in both the cytoplasm and nucleus. Cytoplasmic genes are located in the chloroplast and mitochondrial genomes. Unlike the nuclear genome, the cytoplasmic genomes consist of single, circular, double-stranded molecules of DNA, and in many crop species, the cytoplasmic genomes are inherited solely through the maternal parent. Maternal inheritance of cytoplasmic genomes and Mendelian inheritance of the nuclear genome were used to model the genotypic value of an individual. The model then was utilized to derive genetic variances and covariances for a random-mating population. Finally, the use of reciprocal mating designs to estimate variance components was investigated.

Key words: Cytoplasmic genome – Extranuclear inheritance – Reciprocal mating designs

Introduction

Caspari (1948) classified the different types of non-Mendelian inheritance as maternal inheritance, dauer-modification, and cytoplasmic inheritance. Willham (1963) developed a quantitative theory for traits influenced by maternal effects in animals. Evidence for cytoplasmic effects in quantitative traits of plants has been adequate since Kihara (1951) developed alloplasmic wheat (*Triticum aestivum* L.) lines. The effects of cytoplasms involve both direct influence and interactions of cytoplasms with nuclear effects (Ashri 1964; Duvick 1958; Robertson and Frey 1984). Indeed, Hermesen (1968) proposed that every nuclear effect has a cytoplasmic component.

Many quantitative traits are end products of metabolic pathways that are controlled by both nuclear and cytoplasmic genes (Beale and Knowles 1978). For example, vegetative biomass is a measure of CO_2 assimilation, which is regulated by photosynthesis and respiration. Both processes are jointly controlled by nuclear and cytoplasmic genes (Iwanaga et al. 1978). Most organelle enzymes are constructed from polypeptides encoded in both the organelle and nuclear genomes (Borst et al. 1983). Ribulose-1,5-bisphosphate carboxylase, an enzyme responsible for carbon fixation in bundle sheath cells of maize (*Zea mays* L.), consists of eight copies of two nonidentical subunits. The large subunit is coded by chloroplast DNA (Bowman et al. 1981), and the smaller one is coded within the nucleus (Highfield and Ellis 1978; Cashmore et al. 1978) by a multigenic family (Dunsmuir et al. 1983).

To date, a theory for quantitative traits influenced by cytoplasmic effects has not been developed. However, given certain assumptions, models that utilize reciprocal effects (Mather and Jinks 1982) can describe cytoplasmic effects. Beavis (1985) used Mather and Jinks' models (1982) to describe the nuclear and cytoplasmic effects on grain yield means of backcross populations from reciprocal matings. The models did not always fit the data, which suggests that the assumptions underlying the application of the models were invalid.

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Most higher plant species exhibit uniparental inheritance of cytoplasmic effects through the maternal parent, although exceptions occur in *Secale*, *Solanum*, and *Oenethera* (Gilham 1978). Developments in biotechnology have allowed molecular geneticists to confirm the inheritance of organellar DNA. Electrophoretic patterns of endonucleic restriction digests of chloroplast and mitochondrial DNA have been used to describe the inheritance of organelle DNA in numerous plant species (Conde et al. 1979; Hatfield et al. 1985; Sisson et al. 1978; Tsunewaki and Ogihara 1983; Vedel et al. 1976). Although there may be numerous mechanisms responsible for uniparental inheritance (Sears 1980), the result is that the cytoplasmic genome from the paternal parent is eliminated from the zygote.

In this paper, we propose a quantitative genetic model for traits influenced by cytoplasmic genes that are maternally inherited, develop variance and covariance components for a random-mating population, and consider the use of a reciprocal mating design for the estimation of variance and covariance components.

Assumptions

Cytoplasmic genome

The cytoplasmic genome consists primarily of DNA in the chloroplasts (cpDNA) and mitochondria (mtDNA) (Beale and Knowles 1978). The number of chloroplasts and mitochondria per cell is variable and depends upon the differentiated state of the cell (Butterfass 1979). Organelles in both biparentally and maternally inherited cytoplasms tend toward a homogeneous state within the cell (Michael 1978; Sears 1980), and, unlike nuclear DNA, replication and transmission of orgenellar DNA are independent of nuclear control (Birky 1983). Michaelis (1958) proposed that the partitioning of organelles during mitosis is stochastic; i.e., there is a positive probability that daughter cells will not receive equal quantities of organelles. Birky (1983) also indicated that maternal inheritance perpetuates the homoplasmic condition within maternal lines of descent. Thus, it can be assumed that a plant population that exhibits maternal inheritance of the cytoplasm consists of individuals with genetically homogeneous chloroplasts and mitochondria.

The structure of the mitochondrial genome is not well established. Kolodner and Tewari (1972) found that the mitochondrial genome from pea (*Pisum sativum* L.) consists of circular, double-stranded molecules of DNA. However, according to Leaver and Gray (1982) and authors cited therein, maize (*Zea maize* L.) mitochondria contain circular and linear DNA of various sizes. Leaver and Gray (1982) suggested that heterogeneous pieces of mtDNA may be a manifestation of a genome organized into different-sized "chromosomes". An alternative hypothesis supported by most investigators (Leaver et al. 1983; Levings 1983; Lonsdale et al. 1983) is that the genome usually exists as a single, large, circular, double-stranded molecule of DNA from which smaller molecules arise. The smaller molecules of DNA are formed by intramolecular recombination events between short repeated sequences. The structure of the chloroplast genome also is that of single, circular, double-stranded DNA molecules (Gillham 1978; Palmer 1985). Until recently, the occurrence of intramolecular recombination was not known in the chloroplast genome of higher plants, but Brears et al. (1986) have reported that an inverted repeat of both sugar beet (*Beta vulgaris* L.) and maize can recombine to give two isomeric forms of the chloroplast genome. The evidence for intramolecular recombination suggests that inter-molecular recombination also occurs, although it has been difficult to detect in organellar genomes because the genomes are homozygous and are maternally inherited in most plant species. However, work on cytoplasm fusion among Nicotiana spp. (Maliga et al. 1982; Cséplő and Maliga 1984) indicated that inter-molecular recombination events can occur. Recombination events of the cytoplasmic genomes are of interest to the study of gene expression, but they have no effect on the concept of linkage for maternally inherited cytoplasms; i.e., all cytoplasmic loci will appear to be completely linked. Thus, the structure of the cytoplasmic genomes can be viewed as two populations of polyploid organelles with a basic chromosome number of one.

Population model

Consider a metric trait possessed by diploid individuals in an infinite random-mating population. Assume that diverse cytoplasms exist within the population, but not within maternal lines of descent. Also assume that there is no environmental influence. Let two genetic loci, one located in the nuclear genome and the other in the cytoplasmic genome, procude a genotypic value Y. Because the cytoplasmic genome of plant species consists of two distinct genomes, arbitrarily assign the cytoplasmic locus to either the chloroplast or mitochondrial genome. At each locus, it is possible to have an arbitrary number of alleles. Denote the nuclear alleles, $A_1, A_2, A_3, \ldots, A_k$ and the cytoplasmic alleles $C_1, C_2, C_3, \ldots, C_m$. Each of the alleles have arbitrary allelic frequencies in the population of $p_1, p_2, \ldots, p_k$ such that $\sum_i p_i = 1$ for the nuclear locus and $q_1, q_2, \ldots, q_m$ such that $\sum_k q_k = 1$ for the cytoplasmic locus.

The genotype of an individual in the population may be denoted $A_i A_j C_t$. Given Mendelian inheritance of the nuclear genes and strict maternal inheritance of the cytoplasmic genes, the probability of selecting $A_i A_j C_t$ is $p_i p_j q_t$. Following the algebra of Kempthorne (1957), the genotypic value of this individual may be denoted Y_{ijt} and can be modelled as

$$Y_{ijt} = \mu + \alpha_i^n + \alpha_j^n + \gamma_t^c + \delta_{ij}^n + (\alpha^n \gamma^c)_{it} + (\alpha^n \gamma^c)_{jt} + (\delta^n \gamma^c)_{ijt}. \quad (1)$$

In (1), μ is the population mean and is calculated as the sum of the product of gene frequencies by genotypic values; i.e.,

$$\mu = \sum_i \sum_j \sum_t p_i p_j q_t Y_{ijt}.$$

α_i^n and α_j^n are the additive effects of alleles i and j at the nuclear locus and are calculated as average deviations from the population mean; i.e.,

$$\alpha_i^n = \sum_j \sum_t p_j q_t Y_{ijt} - \mu, \quad \text{and}$$

$$\alpha_j^n = \sum_i \sum_t p_i q_t Y_{ijt} - \mu.$$

γ_t^c is the additive effect of allele t at the cytoplasmic locus; i.e.,

$$\gamma_t^c = \sum_i \sum_j p_i p_j Y_{ijt} - \mu.$$

δ_{ij}^n is the dominance deviation attributed to the nuclear locus and is calculated as an average deviation from the population mean after it is adjusted for additive effects from the nuclear

locus; i.e.,

$$\delta^n_{ij} = \sum_t q_t Y_{ijt} - (\mu + \alpha^n_i + \alpha^n_j).$$

Because of the assumption of a homozygous organelle, no dominance deviation needs to be defined for the cytoplasmic locus. The three nuclear-cytoplasmic interaction effects can be defined in a manner similar to the nuclear dominance effects; i.e.,

$$(\alpha^n \gamma^c)_{it} = \sum_j p_j Y_{ijt} - (\mu + \alpha^n_i + \gamma^c_t),$$

$$(\alpha^n \gamma^c)_{jt} = \sum_i p_i Y_{ijt} - (\mu + \alpha^n_j - \gamma^c_t), \quad \text{and}$$

$$(\delta^n \gamma^c)_{ijt} = Y_{ijt} - (\mu + \alpha^n_i + \alpha^n_j + \gamma^c_t + \delta^n_{ij} + (\alpha^n \gamma^c)_{it} + (\alpha^n \gamma^c)_{jt}).$$

Important algebraic properties resulting from these definitions are:

1. $\sum_i p_i \alpha^n_i = \sum_t q_t \gamma^c_t = 0,$
2. $\sum_i p_i \delta^n_{ij} = \sum_j p_j \delta^n_{ij} = 0,$
3. $\sum_i \sum_t p_i q_t (\alpha^n \gamma^c)_{it} = 0,$
4. $\sum_i p_i (\delta^n \gamma^c)_{it} = \sum_t q_t (\alpha^n \gamma^c)_{it} = 0.$

Results

Variances

The genotypic variance for a random-mating population can be partitioned into components:

$$\begin{aligned}\sigma^2_g &= \sum_i \sum_j \sum_t p_i p_j q_t (Y_{ijt} - Y\ldots)^2 \\ &= 2\sum_i p_i (\alpha^n_i)^2 + \sum_t q_t (\gamma^c_t)^2 + \sum_i \sum_j p_i p_j (\delta^n_{ij})^2 \\ &\quad + 2\sum_i \sum_t p_i q_t (\alpha^n \gamma^c)^2_{it} + \sum_i \sum_j \sum_t p_i p_j q_t (\delta^n \gamma^c)^2_{ijt}.\end{aligned} \tag{2}$$

Notice that two terms describe variability from the additive effects at the nuclear locus and one term describes the variability due to additive effects at the cytoplasmic locus. One term describes variability derived from effects of dominance deviations at the nuclear locus, but analogous term is associated with the cytoplasmic locus. The epistatic components consist of three terms, two associated with additive cytoplasmic by additive nuclear variability and one associated with cytoplasmic additive by nuclear dominance effects. The result is similar to the more general two-locus model with arbitrary epistasis and no linkage (Kempthorne 1957) and may be written as:

$$\sigma^2_g = \sigma^2_{A_n} + \sigma^2_{A_c} + \sigma^2_{D_n} + \sigma^2_{(AA)_{nc}} + \sigma^2_{(DA)_{nc}}. \tag{3}$$

Consider a case of two unliked nuclear loci with arbitrary epistasis and one cytoplasmic locus. Again, the algebra of Kempthorne (1957) is utilized to determine the genotypic variance for a random-mating population:

$$\sigma^2_g = \sum_i \sum_j \sum_k \sum_l \sum_t p^1_i p^1_j p^2_k p^2_l q_t (Y_{ij,kl,t} - Y\ldots)^2,$$

where the superscript above p is used to designate the nuclear locus with $\sum_k p^2_k = 1$. The algebraic properties that apply to the first nuclear locus also apply to the second, and the decomposed genetic variance may be written as:

$$\begin{aligned}\sigma^2_g &= \sigma^2_{A_{n_1}} + \sigma^2_{A_{n_2}} + \sigma^2_{D_{n_1}} + \sigma^2_{D_{n_2}} + \sigma^2_{(AA)_{n_1 n_2}} + \sigma^2_{(AD)_{n_1 n_2}} \\ &\quad + \sigma^2_{(DD)_{n_1 n_2}} + \sigma^2_{A_c} + \sigma^2_{(AA)_{n_1 c}} + \sigma^2_{(AA)_{n_2 c}} + \sigma^2_{(AD)_{c n_1}} \\ &\quad + \sigma^2_{(AD)_{c n_2}} + \sigma^2_{(AAA)_{n_1 n_2 c}} + \sigma^2_{(ADA)_{n_1 n_2 c}} + \sigma^2_{(DDA)_{n_1 n_2 c}}.\end{aligned} \tag{4}$$

Note that the decomposition consists of the same terms derived by Kempthorne (1957) for a two-locus model with arbitrary epistasis, where the subscripts describe the source and the locus (loci) responsible for the effects. For example, A_{n_1} refers to the additive effects from the first nuclear locus and $AD_{n_1 n_2}$ refers to the additive by dominance epistatic effects from the nuclear loci. The decomposition also includes a term that describes variability at the cytoplasmic locus, $\sigma^2_{A_n}$, and seven terms that describe variability due to nuclear by cytoplasmic interactions. For example, $\sigma^2_{(AAA)_{n_1 n_2 c}}$ describes the variability due to additive by additive nuclear epistasis interacting with additive cytoplasmic effects.

The model obtained by increasing the number of cytoplasmic loci that affect a trait can be obtained by reconsidering the structure, replication, and inheritance of the cytoplasmic genomes. Recall that we assumed that each copy of organellar DNA is an exact replica of its sisters and that the organellar loci are completely linked. Because at least one copy of the chloroplast and mitochondrial genomes will be passed on to all daughter cells, the integrity of the cytoplasmic genome will remain constant in a maternally inherited cytoplasm. Therefore, the results given in (4) apply for two nuclear loci and any number of cytoplasmic loci. The extension to any number of nuclear and cytoplasmic loci is immediate.

Covariances

Consider two individuals, X and Y, drawn from an infinite random-mating population with genotypes denoted $X = A_i A_j C_t$ and $Y = A_k A_l C_u$. Setting the population mean equal to zero, the genotypic values for X and Y may be modelled as:

$$X_{ijt} = \alpha^n_i + \alpha^n_j + \gamma^c_t + \delta^n_{ij} + (\alpha^n \gamma^c)_{it} + (\alpha^n \gamma^c)_{jt} + (\delta^n \gamma^c)_{ijt}$$

and

$$Y_{klu} = \alpha_k^n + \alpha_l^n + \gamma_u^c + \delta_{kl}^n + (\alpha^n \gamma^c)_{ku} + (\alpha^n \gamma^c)_{lu} + (\delta^n \gamma^c)_{klu}.$$

The Cov (X, Y) is $E(X_{ijt}, Y_{klu})$, which equals

$$E[(\alpha_i^n + \alpha_j^n + \gamma_t^c + \delta_{ij}^n)(\alpha_k^n + \alpha_l^n + \gamma_u^c + \delta_{kl}^n)]$$
$$+ E[((\alpha\gamma)_{it} + (\alpha\gamma)_{jt})((\alpha^n \gamma^c)_{ku} + (\alpha\gamma)_{lu})]$$
$$+ E[(\delta^n \gamma^c)_{ijt}(\delta^n \gamma^c)_{klu}].$$

The algebraic properties given previously and an additional property, $E(\alpha_{i_x} \gamma_{u_y}) = 0$, which can be derived from property 1, make Cov (X, Y) equal to

$$(P_{ik} + P_{il} + P_{jk} + P_{jl}) \tfrac{1}{2} \sigma^2_{A_n} + P_{tu} \sigma^2_{A_c}$$
$$+ (P_{ik,jl} + P_{il,jk}) \sigma^2_{D_n}$$
$$+ P_{tu} \cdot (P_{ik} + P_{il} + P_{jk} + P_{jl}) \tfrac{1}{2} \sigma^2_{(AA)_{nc}}$$
$$+ P_{tu} \cdot (P_{ik,jl} + P_{il,jk}) \sigma^2 (DA)_{nc},$$

where P_{ik} is the probability that A_i is identical by descent to A_k, and $P_{ik,jl}$ is the joint probability that A_i is identical by descent to A_k and A_j is identical by descent to A_l. Notice that P_{tu} is the probability that X and Y have the same cytoplasmic genes by descent. In plant species that exhibit maternal inheritance of cytoplasmic genes,

$$P(C_t = C_u | X \text{ and } Y \text{ are full sibs}) = P(C_t = C_u | X \text{ and } Y \text{ are maternal half sibs}) = 1, \quad \text{and}$$

$$P(C_t = C_u | X \text{ and } Y \text{ are reciprocal full sibs}) = P(C_t = C_u | X \text{ and } Y \text{ are paternal half sibs}) = 0.$$

By utilizing Malecot's coefficient of parentage (r_{xy}), Kempthorne's U_{xy} to denote ($P_{ik,jl} + P_{il,jk}$), and C_{xy} to denote P_{tu},

$$\text{Cov}(X, Y) = 2 r_{xy} \sigma^2_{A_n} + C_{xy} \sigma^2_{A_c} + U_{xy} \sigma^2_{D_n} + 2 r_{xy} C_{xy} \sigma^2_{(AA)_{nc}} + U_{xy} C_{xy} \sigma^2_{(DA)_{nc}}. \quad (5)$$

The model may be extended to any number of nuclear and cytoplasmic loci. Again, by making use of Kempthorne's results and assuming that all cytoplasmic loci are completely linked, Cov (X, Y) is given by

$$\sum_{n_1} \sum_{n_2} (2 r_{xy})^{n_1} (U_{xy})^{n_2} \sigma^2_{(AD)_{n_1 n_2}} + C_{xy} \sigma^2_{A_c} + C_{xy} \sum_{n_1} \sum_{n_2} (2 r_{xy})^{n_1} (U_{xy})^{n_2} \sigma^2_{(AD)_{n_1 n_2}} A_c$$

where n_1 is the total number of nuclear loci involved in the interaction of additive effects and n_2 is the total number of nuclear loci involved in the interaction of dominance effects.

Estimation of parameters

Model (1) can provide biological interpretations for parameters generated from statistical analyses of reciprocal crosses. Yates (1947) was the first to analyze data from a complete set of reciprocal crosses. Griffing (1956) considered such an analysis as one of four possible diallel methods. Cockerham (1963) related variance components of reciprocal crosses generated by the complete diallel and North Carolina Design II with reciprocals of Comstock and Robinson (1948) but presented no biological interpretation for reciprocal or maternal effects. Cockerham and Weir (1977) considered the relationships among three models used to describe reciprocal effects generated by quadratic analyses. One of the models, a "bio model", considers nuclear and extranuclear effects from both parents.

Our model (1) seems to be a special case of the "bio model", which, in the notation of Cockerham and Weir (1977) is

$$G_{ij} = n_i + n_j + t_{ij} + m_i + p_j + k_{ij}. \quad (c)$$

G_{ij} is the coded genotypic mean of effects attributable to maternal parent i and paternal parent j, where n and t refer to nuclear effects, and m, p, and k refer to

Table 1. Values of Malecot's coefficient of parentage r_{xy}, Kempthornes' U_{xy}, and the cytoplasmic coefficient C_{xy} for relationships obtained from reciprocal matings designs

Relationship	Cockerham-Weir[a] notation	$2r_{xy}$	U_{xy}	C_{xy}
Full sibs	$\mathscr{C}_f$	$\frac{1}{2}$	$\frac{1}{4}$	1
Reciprocal full sibs	$\mathscr{C}_{rf}$	$\frac{1}{2}$	$\frac{1}{4}$	0
Maternal half sibs	$\mathscr{C}_m$	$\frac{1}{4}$	0	1
Paternal half sibs	$\mathscr{C}_p$	$\frac{1}{4}$	0	0
Reciprocal half sibs	$\mathscr{C}_r$	$\frac{1}{4}$	0	0

[a] Cockerham and Weir (1977)

Table 2. Covariances of related individuals obtained from a reciprocal mating design

Relationship[a]	Covariance	
	Model (1)	Model (c)[a]
$\mathscr{C}_f$	$\frac{1}{2}\sigma^2_{A_n} + \frac{1}{4}\sigma^2_{D_n} + \sigma^2_{A_c} + \frac{1}{2}\sigma^2_{AA_{nc}} + \frac{1}{4}\sigma^2_{DA_{nc}}$	$\sigma^2_M + \sigma^2_P + \sigma^2_{MP}$
$\mathscr{C}_{rf}$	$\frac{1}{2}\sigma^2_{A_n} + \frac{1}{4}\sigma^2_{D_n}$	$2\sigma^2_n + \sigma^2_t$
$\mathscr{C}_m$	$\frac{1}{4}\sigma^2_{A_n} + \sigma^2_{A_c} + \frac{1}{4}\sigma^2_{AA_{nc}}$	σ^2_M
$\mathscr{C}_p$	$\frac{1}{4}\sigma^2_{A_n}$	σ^2_P
$\mathscr{C}_r$	$\frac{1}{4}\sigma^2_{A_n}$	σ^2_n

[a] Notation of Cockerham and Weir (1977)

simple and epistatic extranuclear effects. The relationship between our model (1) and model (c) of Cockerham and Weir (1977) can be shown as follows. If a maternal parent, $A_i A_j C_t$, and a paternal parent, $A_v A_w C_u$, are mated, the array of full sib offspring from the mating is

$$\tfrac{1}{4}(A_i A_v C_t + A_i A_w C_t + A_j A_v C_t + A_j A_w C_t). \quad (7)$$

Thus, if Cockerham and Weir's (i) is replaced by (i j t) and (j) is replaced by (v w u), the coded genotypic mean, G_{ij}, becomes

$$\begin{aligned} G_{(ijt),(vwu)} &= n_{ij} + n_{vw} + t_{ij,vw} + m_{ijt} + p_{vwu} + k_{(ijt)(vwu)} \\ &= (\tfrac{1}{2}(\alpha_i^n + \alpha_j^n)) + (\tfrac{1}{2}(\alpha_v^n + \alpha_w^n)) \\ &\quad + \tfrac{1}{4}(\delta_{iv}^n + \delta_{iw}^n + \delta_{jv}^n + \delta_{jw}^n) \\ &\quad + (\gamma_t^c + \tfrac{1}{2}(\alpha^n \gamma^c)_{it} + \tfrac{1}{2}(\alpha^n \gamma^c)_{jt}) \\ &\quad + (\tfrac{1}{2}(\alpha^n \gamma^c)_{vt} + \tfrac{1}{2}(\alpha^n \gamma^c)_{wt} + \tfrac{1}{4}(\delta^n \gamma^c)_{ivt} \\ &\quad + \tfrac{1}{4}(\delta^n \gamma^c)_{iwt} + \tfrac{1}{4}(\delta^n \gamma^c)_{jvt} + \tfrac{1}{4}(\delta^n \gamma^c)_{jwt}). \end{aligned} \quad (8)$$

By (7), $A_i A_v C_u$, $A_i A_w C_u$, $A_j A_v C_u$, and $A_j A_w C_u$ do not exist. Therefore, the components of variance are related as

$$\sigma_n^2 = \tfrac{1}{4}\sum_i \sum_j p_i p_j (\alpha_i + \alpha_j)^2 = \tfrac{1}{4}\sigma_{A_n}^2,$$

$$\sigma_t^2 = \tfrac{1}{4}\sum_i \sum_j p_i p_j (\delta_{ij}^n)^2 = \tfrac{1}{4}\sigma_{D_n}^2,$$

$$\begin{aligned} \sigma_m^2 &= \sum_t q_t (\gamma_t^c)^2 + \tfrac{1}{4}\sum_i \sum_t p_i q_t (\alpha^n \gamma^c)_{it}^2 \\ &\quad + \tfrac{1}{4}\sum_j \sum_t p_i q_t (\alpha^n \gamma^c)_{jt}^2 \\ &= \sigma_{A_c}^2 + \tfrac{1}{4}\sigma_{(AA)_{nc}}^2, \end{aligned}$$

$$\sigma_p^2 = 0,$$

$$\begin{aligned} \sigma_k^2 &= \tfrac{1}{4}\sum_i \sum_t p_i q_t (\alpha^n \gamma^c)_{it}^2 + \tfrac{1}{4}\sum_j \sum_t p_j q_t (\alpha^n \gamma^c)_{jt}^2 \\ &\quad + \tfrac{1}{4}\sum_i \sum_j \sum_t p_i p_j q_t (\delta^n \gamma^c)_{ijt}^2 = \tfrac{1}{4}(\sigma_{(AA)_{nc}}^2 + \sigma_{(DA)_{nc}}^2). \end{aligned}$$

Covariances for specific relationships investigated by Cockerham and Weir (1977) are determined for model (1) by using the general formula for covariances (5) and the coefficients of Table 1. Results of covariances for Cockerham and Weir's model (c) and our model (1) are summarized in Table 2. The analysis of variance table for a reciprocal mating design (Table 3 from Cockerham and Weir) can be recast in our notation (Table 3). It is obvious that model (1) will only produce estimates of genetic variance components for

$$\sigma_{A_n}^2,\ \sigma_{D_n}^2,\ \sigma_{A_c}^2 + \tfrac{1}{4}\sigma_{(AA)_{nc}}^2, \quad \text{and} \quad \sigma_{(AA)_{nc}}^2 + \sigma_{(DA)_{nc}}^2$$

from a reciprocal mating design.

Discussion

On the basis of biological features of the cytoplasmic genome, a quantitative genetic model was developed for traits influenced by cytoplasmic genes that are strictly inherited from the maternal parent. Application of the model will depend, in part, upon the biological validity of the assumptions. For cytoplasms that are maternally inherited, we assumed that each organellar genome is homoplasmic, that each organelle was polyploid with a basic chromosome number of one (x = 1), and that each organelle was homozygous at all loci.

There is evidence that both the chloroplasts and mitochondria of a cell tend toward a homoplastic state; i.e., that a population of chloroplasts or mitochondria within each cell tends to be homogeneous (Birky 1983). This tendency is best shown in species that exhibit biparental cytoplasmic inheritance but also has been shown in studies on protoplast fusion (Izhar 1980) and cytoplasm-protoplast fusion (Maliga et al. 1982). Work on cytoplast fusion among *Nicotiana* spp. (Maliga et al. 1982; Cséplő and Maliga 1984) showed that heterogenous populations could be maintained through plant regeneration and one generation of seed production. Nevertheless, the tendency toward a homoplasmic condition was evident. Indeed, taxonomic studies on cytoplasmic genomes, which utilize electrophoretic patterns of endonuclease restricted cpDNA and/or mtDNA, rely on the assumption that the organellar genomes are homogeneous within maternal lines of descent. Thus, it is not likely that a heteroplasmic condition needs to be considered in a genetic model with cytoplasmic effects that are maternally inherited.

As noted in the introduction, the structure of the genome in both the chloroplast and mitochondrion is hypothesized to consist of double-stranded, circular molecules of DNA. We assumed that each chloroplast and mitochondrion is a polyploid (x = 1) homozygous organelle. Of course, the impact of inter-organellar recombination and segregation on the model is negligible, given that the cytoplasm consists of homogeneous organelles that are homozygous. In a system where recombination events occur between heterogeneous organelles, it is unlikely that the integrity of the cytoplasm will be maintained under the relaxed controls of replication and segregation described by Birky (1983), and the assumptions presented herein will need to be revised.

To our knowledge, the heterozygosity of organelles has not been tested, and the technology needed to investigate intra-organellar heterozygosity may not exist. A heterozygous, homoplasmic, cellular condition of the mitochondrial genome might explain observed heterogeneous restriction endonuclease digests of

Table 3. Expected mean squares for indicated sources of variability in an analysis of variance of offspring from a reciprocal mating design

Source	df	Expected mean squares	
		Model (1)	Model (c)[a]
General	$2(N-1)$	$\sigma^2+\frac{U}{4}(\sigma^2_{(AA)Nc})+\sigma^2_{(DA)nc})+\frac{U}{2}\sigma^2_{D_n}+\frac{UN}{2}(\sigma^2_{A_c}+\frac{1}{4}\sigma^2_{(AA)Nc})+\frac{UN}{2}\sigma^2_{A_n}$	$\sigma^2+U\sigma^2_k+2U\sigma^2_t+\frac{UN}{2}(\sigma^2_m+\sigma^2_p)+2UN\sigma^2_n$
Specific	$(N-1)^2$	$\sigma^2+\frac{U}{4}(\sigma^2_{(AA)nc}+\sigma^2_{(DA)nc})+\frac{U}{2}\sigma^2_{D_n}$	$\sigma^2+U\sigma^2_k+2U\sigma^2_t$
Reciprocal general	$2(N-1)$	$\sigma^2+\frac{U}{4}(\sigma^2_{(AA)nc}+\sigma^2_{(DA)nc})+\frac{UN}{2}(\sigma^2_{A_c}+\frac{1}{4}\sigma^2_{(AA)nc})$	$\sigma^2+U\sigma^2_k+\frac{UN}{2}(\sigma^2_m+\sigma^2_p)$
Reciprocal specific	$(N-1)^2$	$\sigma^2+\frac{U}{4}(\sigma^2_{(AA)nc}+\sigma^2_{(DA)nc})$	$\sigma^2+U\sigma^2_k$
Error	$2N^2(U-1)$	σ^2	σ^2
Total	$2N^2U-2$		

[a] In the notation of Cockerham and Weir (1977)

mtDNA (Leaver and Gray 1982). If the organelles are heterozygous, then the cytoplasmic effect (γ^1_c) may be a composite of additive, dominance, and interactions of additive and dominance, genetic effects. Nuclear by cytoplasmic interaction effects in (1) also would be more complex. However, if the integrity of the cytoplasm is maintained (i.e., a homoplasmic condition is maintained from one generation to the next), then the individual component effects of a heterozygous cytoplasm could not be detected from a reciprocal mating design and the model would still apply.

The population assumptions are the usual ones for a diploid species except for the assumption that multiple cytoplasmic alleles exist in a random-mating population. Theoretical models describing the origination, evolution, and equilibrium conditions of cytoplasmic gynodieocy (male sterility) have been proposed (Charlesworth and Ganders 1979; Clark 1984; Gregorius and Ross 1984; Ross and Gregorius 1985). All these studies were developed on simple models with two cytoplasm types (e.g., male sterile and male fertile) and could be generalized to cover multiple cytoplasmic types. Under the condition of complete linkage, multiple alleles are equivalent to multiple cytoplasms. In addition, cytoplasmic variability in the form of cytoplasmic gynodioecy has been observed in natural random-mating populations (Ganders 1978) and is well utilized in plant species that are economically important (Duvick 1965; Quinby 1970). Therefore, the assumption of multiple cytoplasmic alleles is valid, and the resulting model should be applicable to some random-mating populations.

To apply model (1) to populations derived from reciprocal matings, it must be assumed that the only source of extranuclear effects are cytoplasmic. For most plant breeding experiments where careful field husbandry is practiced, maternal effects not attributed to cytoplasms will be minimized. However, for those species where a triploid endosperm affects growth and development of the seedling, reciprocal effects cannot be ascribed solely to the cytoplasm. Our results indicate that, even in the absence of endosperm effects, the use of a reciprocal mating design will not provide unique estimates of the cytoplasmic variance components for species that experience maternal inheritance of cytoplasms. Also, the results show that the estimate of general reciprocal effects will be an amalgam of cytoplasmic variability and additive nuclear by cytoplasmic variability. The reason is revealed in equation (8). Cockerham and Weir's (1977) extranuclear maternal effects include cytoplasmic effects and additive nuclear by cytoplasmic interaction effects from the maternal parent. From a biological perspective, it is not possible to separate cytoplasmic effects from additive nuclear by cytoplasmic interactions because both are inherited as a unit in species that exhibit maternal inheritance of the cytoplasm. Also, metabolic processes that are known to affect quantitative traits are regulated by enzymes that are coded at both nuclear and cytoplasmic loci. Indeed, the possibility of a pure cytoplasmic effect is remote. Finally, from a plant breeding perspective, confounding $\sigma^2_{A_c}$ with $\sigma^2_{(AA)_{nc}}$ will have little impact on estimates of heritability or genetic gain.

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