Research Article

Dominance, epistasis, heritabilities and expected genetic gains

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Abstract

Although epistasis is common in gene systems that determine quantitative traits, it is usually not possible to estimate the epistatic components of genotypic variance because experiments in breeding programs include only one type of progeny. As the study of this phenomenon is complex, there is a lack of theoretical knowledge on the contribution of the epistatic variances when predicting gains from selection and on the bias in estimating genetic parameters when fitting the additive-dominant model. The objective of this paper is to discuss these aspects. Regarding a non-inbred population, the genetic value due to dominance and the epistatic components of the genotypic value are not indicators of the number of favorable genes present in an individual. Thus, the efficiency of a selection process should be based on the narrow-sense heritability, a function only of additive variance. If there is no epistasis, generally it is satisfactory to assess the selection efficiency and to predict gain based on the broad-sense heritability. Regardless of the selection unit or type of epistasis, the bias in the estimate of the additive variance when assuming the additive-dominant model is considerable. This implies overestimation of the heritabilities at half sib family mean, plant within family and plant levels, and underestimation if the selection units are full sib progenies. The predicted gains will have a bias proportional to that of the heritability.

Introduction

The methodologies generally used to study quantitative trait inheritance, including interaction between non-allelic genes, are generation mean analysis (Mather and Jinks, 1974), that permits estimation of linear components of genotypic means, and triple test cross analysis (Kearsey and Jinks, 1968), which permits testing the existence of epistasis. Rebetzke *et al*. (2003) reported epistatic gene action from crosses between a wheat variety with low leaf conductance and three with high leaf conductance values. Kumar *et al*. (2003) observed that wheat embryo resistance to *Noevossia indica* also depended on epistatic effects. In the presence of the pathogen, they identified complementary epistasis for fresh weight of calli. Interaction between gene combinations of two and three loci was observed by Sharma *et al*. (2003) in a study of spike length inheritance in durum wheat plants. At normal and late sowing, digenic epistatic effects predominated compared to additive and dominance effects. In a study of mungbean resistance to *Erysiphe polygoni*, Gawande and Patil (2003) observed duplicate epistasis for disease incidence and area under disease progress curve. Zhao and Meng (2003) re-

ported gene interaction in rapeseed plant resistance to *Sclerotinia sclerotiorum*. The predominant type of epistasis was additive x additive. With the objective of determining the importance of epistatic effects in maize hybrid production, Hinze and Lamkey (2003) observed significant epistatic interaction in five of the forty cases analyzed. Although with a smaller contribution to soybean plant resistance to *Cercospora sojina*, as compared to the mean effects of genes and allelic interactions, Martins *et al*. (2003) reported epistasis to be responsible for the degree of infection, number of lesions per foliole, mean lesion diameter and disease index. Khattak *et al*. (2003) used a triple test cross to detect that epistasis was responsible for two secondary components of mungbean plant yield, only in the spring/summer planting. Regarding number of branches and biomass, partitioning of total epistasis revealed additive x additive, additive x dominant and dominant x dominant epistatic interactions, with predominance of the first type.

The estimation of epistatic components of genotypic variance is unusual in genetic studies because of limitation of the methodology, as in the case of the triple test cross, the high number of generations to be produced and assessed (Viana, 2000), and mainly because only one type of progeny, half sib, full sib or inbred families, is commonly included in the experiments. Even with the phenotypic values

of plants in the families and in one or more genetically homogeneous populations, for estimation of the environmental variance at plant level, the breeder has two equations, the mean squares between and within families, to estimate at least five genetic variances, assuming a non-inbred population and digenic epistasis (Hallauer and Miranda Filho, 1988). An exception is the study by Braga (1987). Commonly, the additive-dominant model is fitted, assuming epistasis to be negligible or non-existent. As epistasis is common and not occasional, the consequences of this necessary simplification should be known. Furthermore, because of the complexity of theoretical studies on epistasis, there is a lack of information about the contribution of the epistatic components of genotypic variance when predicting gains from selection. The objective of this study was to produce theoretical knowledge on these problems.

Methods, Results and Discussion

Genotypic variance in non-inbred populations

The genotypic value of an individual in relation to two loci (A and B) is (Kempthorne, 1955):

$$G_{ijmn} = M + \left(\alpha_i^A + \alpha_j^A + \alpha_m^B + \alpha_n^B\right) + \left(\delta_{ij}^A + \delta_{mn}^B\right) + \left(\alpha_i^A \alpha_m^B + \alpha_i^A \alpha_n^B + \alpha_j^A \alpha_m^B + \alpha_j^A \alpha_n^B\right) + \left(\alpha_i^A \delta_{mn}^B + \alpha_j^A \delta_{mn}^B\right) + \left(\delta_{ij}^A \alpha_m^B + \delta_{ij}^A \alpha_n^B\right) + \delta_{ij}^A \delta_{mn}^B = M + A + D + AA + AD + DA + DD$$

where α represents the effect of a gene, δ represents the effect of interaction between alleles (due to the presence of a pair of alleles), $\alpha\alpha$ represents the additive x additive epistatic effect (due to the presence of a pair of non-allelic genes), $\alpha\delta$ represents the additive x dominant epistatic effect (due to the presence of a gene of one locus and a pair of alleles from another locus), and $\delta\delta$ represents the dominant x dominant epistatic effect (due to the presence of two pairs of non-allelic genes). Under the restrictions

$$E\left(\alpha_i^A\right) = \sum_i p_i \alpha_i^A = E\left(\alpha_m^B\right) = \sum_m p_m \alpha_m^B = \sum_j p_j \delta_{ij}^A$$

$$\sum_n p_n \delta_{mn}^B = \sum_i p_i \left(\alpha_i^A \alpha_m^B\right) = \sum_m p_m \left(\alpha_i^A \alpha_m^B\right) =$$

$$\sum_n p_n \left(\alpha_i^A \delta_{mn}^B\right) = \sum_i p_i \left(\alpha_i^A \delta_{mn}^B\right) = \sum_j p_j \left(\delta_{ij}^A \alpha_m^B\right) =$$

$$\sum_m p_m \left(\delta_{ij}^A \alpha_m^B\right) = \sum_i p_i \left(\delta_{ij}^A \delta_{mn}^B\right) = \sum_m p_m \left(\delta_{ij}^A \delta_{mn}^B\right) = 0$$

where p represents the probability of a gene, M is the population mean with

$$E\left(G_{ijmn}\right) = \sum_i \sum_j \sum_m \sum_n p_i p_j p_m p_n G_{ijmn} = \overline{G}_{....} = M$$

and the expectations of the additive (A), due to dominance (D), additive x additive (AA), additive x dominant (AD),

dominant x additive (DA) and dominant x dominant (DD) genetic values are nil.

The mean of the genotypes containing gene A_i is:

$$\overline{G}_{i...} = \sum_j \sum_m \sum_n p_j p_m p_n G_{ijmn} = M + \alpha_i^A$$

The effect of gene A_i is, thus, $\alpha_i^A = \overline{G}_{i...} - \overline{G}_{....}$. Assuming two allelic forms, $\alpha_i^A = q_a \alpha_a$ if the gene increases the trait expression, or $\alpha_i^A = -p_a \alpha_a$ if the gene decreases it, where p_a is the frequency of the gene that increases the expression of the characteristic and $\alpha_a = a_a + (q_a - p_a)d_a$ is the average effect of a gene substitution. Parameter a_a is the deviation between the genotypic value of the homozygote of greatest expression and the mean of the homozygotes, and d_a is the deviation due to dominance (Falconer and Mackay, 1996).

The mean of the genotypes containing the A_i and A_j alleles is:

$$\overline{G}_{ij..} = \sum_m \sum_n p_m p_n G_{ijmn} = M + \alpha_i^A + \alpha_j^A + \delta_{ij}^A$$

The genetic value due to dominance of the genotype A_iA_j is, therefore, $\delta_{ij}^A = \overline{G}_{ij..} - \overline{G}_{i...} - \overline{G}_{.j..} + \overline{G}_{....}$. Taking two alleles, $\delta_{ij}^A = -2q_a^2 d_a$ if the individual is homozygous for the gene that increases the trait expression, $\delta_{ij}^A = 2p_a q_a d_a$ if it is heterozygous, or $\delta_{ij}^A = -2p_a^2 d_a$ if it is homozygous for the gene that decreases the expression of the characteristic (Falconer and Mackay, 1996).

The mean of the genotypes containing both the A_i and B_m genes is:

$$\overline{G}_{i.m.} = \sum_j \sum_n p_j p_n G_{ijmn} = M + \alpha_i^A + \alpha_m^B + \left(\alpha_i^A \alpha_m^B\right)$$

Thus, $\left(\alpha_i^A \alpha_m^B\right) = \overline{G}_{i.m.} - \overline{G}_{i...} - \overline{G}_{..m.} + \overline{G}_{....}$. The mean of the genotypes containing genes A_i, B_m and B_n is:

$$\overline{G}_{i.mn} = \sum_j p_j G_{ijmn} = M + \alpha_i^A + \alpha_m^B + \alpha_n^B + \delta_{mn}^B + \left(\alpha_i^A \alpha_m^B\right) + \left(\alpha_i^A \alpha_n^B\right) + \left(\alpha_i^A \delta_{mn}^B\right)$$

Thus,

$$\left(\alpha_i^A \delta_{mn}^B\right) = \overline{G}_{i.mn} + \overline{G}_{i...} + \overline{G}_{..m.} + \overline{G}_{...n} - \overline{G}_{i.m.} - \overline{G}_{i..n} - \overline{G}_{..mn} - \overline{G}_{....}.$$

Finally,

$$\delta_{ij}^A \delta_{mn}^B = G_{ijmn} - M - \alpha_i^A - \alpha_j^A - \alpha_m^B - \alpha_n^B - \delta_{ij}^A - \delta_{mn}^B - \left(\alpha_i^A \alpha_m^B\right) - \left(\alpha_i^A \alpha_n^B\right) - \left(\alpha_j^A \alpha_m^B\right) - \left(\alpha_j^A \alpha_n^B\right) - \left(\alpha_i^A \delta_{mn}^B\right) - \left(\alpha_j^A \delta_{mn}^B\right) - \left(\delta_{ij}^A \alpha_m^B\right) - \left(\delta_{ij}^A \alpha_n^B\right)$$

Assuming that the population is in linkage equilibrium, all the genetic effects and values are un-correlated variables because

$$\text{Cov}\left(\alpha_i^A, \alpha_j^A\right) = \sum_i \sum_j p_i p_j \left(\alpha_i^A\right)\left(\alpha_j^A\right) =$$

$$\left[\sum_i p_i\left(\alpha_i^A\right)\right]\left[\sum_j p_j\left(\alpha_j^A\right)\right] = \dots = \text{Cov}\left(\delta_{ij}^A \alpha_m^B, \delta_{ij}^A \delta_{mn}^B\right) =$$

$$\sum_i \sum_j \sum_m \sum_n p_i p_j p_m p_n \left(\delta_{ij}^A \alpha_m^B\right)\left(\delta_{ij}^A \delta_{mn}^B\right) =$$

$$\sum_i \sum_j p_i p_j \left[\sum_m p_m\left(\delta_{ij}^A \alpha_m^B\right)\right]\left[\sum_n p_n\left(\delta_{ij}^A \delta_{mn}^B\right)\right] = 0$$

and, therefore,

$$V(G_{ijmn}) = \sigma_G^2 = E\left[\left(G_{ijmn}\right)^2\right] - M^2 = 2\left[E(\alpha_i^A)^2\right] +$$

$$2\left[E(\alpha_m^B)^2\right] + \left[E(\delta_{ij}^A)^2\right] + \left[E(\delta_{mn}^B)^2\right] + 4\left[E(\alpha_i^A \alpha_m^B)^2\right] +$$

$$2\left[E(\alpha_i^A \delta_{mn}^B)^2\right] + 2\left[E(\delta_{ij}^A \alpha_m^B)^2\right] + \left[E(\delta_{ij}^A \delta_{mn}^B)^2\right] =$$

$$\sigma_{A_a}^2 + \sigma_{A_b}^2 + \sigma_{D_a}^2 + \sigma_{D_b}^2 + \sigma_{AA}^2 + \sigma_{AD}^2 + \sigma_{DA}^2 + \sigma_{DD}^2 =$$

$$\sigma_A^2 + \sigma_D^2 + \sigma_{AA}^2 + \sigma_{AD}^2 + \sigma_{DA}^2 + \sigma_{DD}^2$$

where:

$$\sigma_{A_a}^2 = 2\sum_i p_i\left(\alpha_i^A\right)^2$$

$$\sigma_{A_b}^2 = 2\sum_m p_m\left(\alpha_m^B\right)^2$$

$$\sigma_{D_a}^2 = \sum_i \sum_j p_i p_j\left(\delta_{ij}^A\right)^2$$

$$\sigma_{D_b}^2 = \sum_m \sum_n p_m p_n\left(\delta_{mn}^B\right)^2$$

$$\sigma_{AA}^2 = 4\sum_i \sum_m p_i p_m\left(\alpha_i^A \alpha_m^B\right)^2$$

$$\sigma_{AD}^2 = 2\sum_i \sum_m \sum_n p_i p_m p_n\left(\alpha_i^A \delta_{mn}^B\right)^2$$

$$\sigma_{DA}^2 = 2\sum_i \sum_j \sum_m p_i p_j p_m\left(\delta_{ij}^A \alpha_m^B\right)^2$$

$$\sigma_{DD}^2 = \sum_i \sum_j \sum_m \sum_n p_i p_j p_m p_n\left(\delta_{ij}^A \delta_{mn}^B\right)^2$$

When each gene has two allelic forms, these expressions are equivalent to those obtained by Cockerham (1954).

Correlation between number of genes that increase the trait expression and components of the genotypic value

When considering two genes with independent assortment, both with two alleles, and a population in Hardy-Weinberg and linkage equilibrium, the expectation and the variance of the number of genes that increase the trait expression (N) are:

$$E(N) = p_a^2 p_b^2 (4) + \dots + q_a^2 q_b^2 (0) = 2p_a + 2p_b$$

$$\sigma_N^2 = p_a^2 p_b^2 (4)^2 + \dots + q_a^2 q_b^2 (0)^2 - (2p_a + 2p_b)^2 =$$

$$2p_a q_a + 2p_b q_b$$

The covariances between the number of genes that increase the trait expression and the components of the genotypic value are:

$$\text{Cov}(N, A) = p_a^2 \cdot p_b^2 (4)(A_{22}) + p_a^2 \cdot 2p_b q_b (3)(A_{21}) +$$

$$p_a^2 \cdot q_b^2 (2)(A_{20}) + 2p_a q_a \cdot p_b^2 (3)(A_{12}) + 2p_a q_a \cdot$$

$$2p_b q_b (2)(A_{11}) + 2p_a q_a \cdot q_b^2 (1)(A_{10}) + q_a^2 \cdot p_b^2 (2)(A_{02}) +$$

$$q_a^2 \cdot 2p_b q_b (1)(A_{01}) + q_a^2 \cdot q_b^2 (0)(A_{00}) - E(N).0 =$$

$$2p_a q_a \alpha_a + 2p_b q_b \alpha_b$$

$$\text{Cov}(N, D) = p_a^2 \cdot p_b^2 (4)(D_{22}) + \dots + q_a^2 q_b^2 (0)(D_{00}) -$$

$$E(N).0 = 0$$

where A_{rs} and D_{rs} are the additive and due to dominance genetic values of the genotype containing r and s copies, respectively, of the genes of loci A and B that increase the trait expression,

$$\text{Cov}(N, AA) = p_a^2 \cdot p_b^2 (4)\left[4(\alpha_A \alpha_B)\right] + p_a^2 \cdot 2p_b q_b (3).$$

$$\left[2(\alpha_A \alpha_B) + 2(\alpha_A \alpha_b)\right] + p_a^2 \cdot q_b^2 (2)\left[4(\alpha_A \alpha_b)\right] +$$

$$2p_a q_a \cdot p_b^2 (3)\left[2(\alpha_A \alpha_B) + 2(\alpha_a \alpha_B)\right] + 2p_a q_a \cdot 2p_b q_b (2)$$

$$\left[(\alpha_A \alpha_B) + (\alpha_A \alpha_b) + (\alpha_a \alpha_B) + (\alpha_a \alpha_b)\right] + 2p_a q_a \cdot q_b^2 (1)$$

$$\left[2(\alpha_A \alpha_b) + 2(\alpha_a \alpha_b)\right] + q_a^2 \cdot p_b^2 (2)\left[4(\alpha_a \alpha_B)\right] +$$

$$q_a^2 \cdot 2p_b q_b (1)\left[2(\alpha_a \alpha_B) + 2(\alpha_a \alpha_b)\right] +$$

$$q_a^2 \cdot q_b^2 (0)\left[4(\alpha_a \alpha_b)\right] - E(N).0 = 0$$

$$\text{Cov}(N, AD) = p_a^2 \cdot p_b^2 (4)\left[2(\alpha_A \delta_{BB})\right] + \dots +$$

$$q_a^2 \cdot q_b^2 (0)\left[2(\alpha_a \delta_{bb})\right] - E(N).0 = 0$$

$$\text{Cov}(N, DA) = p_a^2 \cdot p_b^2 (4)\left[2(\delta_{AA} \alpha_B)\right] + \dots +$$

$$q_a^2 \cdot q_b^2 (0)\left[2(\delta_{aa} \alpha_b)\right] - E(N).0 = 0$$

$$\text{Cov}(N, DD) = p_a^2 \cdot p_b^2 (4)(\delta_{AA} \delta_{BB}) + \dots +$$

$$q_a^2 \cdot q_b^2 (0)(\delta_{aa} \delta_{bb}) - E(N).0 = 0$$

under the restrictions previously established.

Considering k genes, the correlations between the number of genes that increases the trait expression and the components of the genotypic value are:

$$\rho_{N,A} = \frac{2\sum_{i=1}^{k} p_i q_i \alpha_i}{\sqrt{\left(2\sum_{i=1}^{k} p_i q_i\right)\left(2\sum_{i=1}^{k} p_i q_i \alpha_i^2\right)}}$$

$$\rho_{N,D} = \rho_{N,AA} = \rho_{N,AD} = \rho_{N,DA} = \rho_{N,DD} = 0$$

and further:

$$\rho_{N,G} = \frac{2\sum\limits_{i=1}^{k} p_i q_i \alpha_i}{\sqrt{\left(2\sum\limits_{i=1}^{k} p_i q_i\right)\left(\sigma_A^2 + \sigma_D^2 + \sigma_{AA}^2 + \sigma_{AD}^2 + \sigma_{DA}^2 + \sigma_{DD}^2\right)}}$$

Thus, of the components of the genotypic value, only the additive genetic value correlates with the number of favorable genes that an individual possesses (the correlation between the number of genes that decreases the trait expression and A has the same magnitude and opposite sign to $\rho_{N,A}$). In the presence of dominance and epistasis or only dominance, the correlation between the number of favorable genes and the genotypic value is less than the correlation between the number of favorable genes and A. Thus, the additive genetic value is generally the best indicator of the number of favorable genes present in an individual. When considering one gene, $\rho_{N,A}$ is equal to 1, with complete dominance, partial dominance or in the absence of dominance, or equal to 1 or -1, with overdominance, depending on the gene frequencies, since the average effect of a gene substitution may be negative (Falconer and Mackay, 1996). Therefore, regardless of the gene frequencies and the degree of dominance, in non-inbred populations the genetic values due to dominance, additive x additive, additive x dominant, dominant x additive and dominant x dominant, among other more complex epistatic values, cannot be used to indicate the number of favorable genes present in an individual.

Based on the values of the correlation between N and A considering three genes (Table 1), assuming $a_i = a$, for all i, it can be inferred that, regardless of the number of genes and of the gene frequencies, the lower the degree of dominance, the greater the linear association between the number of favorable genes and the additive genetic value. The correlation is of low magnitude or negative only in the case

of overdominance and when the frequencies of the dominant genes are high. The correlation values should be high in the most complex polygenic systems, if there is no overdominance. For example, regardless of the number of genes, assuming that for 15%, 25%, and 60% of the loci the frequency of the dominant genes is low (0.1), intermediate (0.5) or high (0.9), respectively, and where in 2%, 70%, 20% and 8% of these loci overdominance ($|d/a| = 2$), complete dominance ($|d/a| = 1$), partial dominance ($|d/a| = 1/2$) and absence of dominance applies, the correlation value is 0.86. Assuming that the frequencies of the dominant genes are low (0.1), intermediate (0.5) and high (0.9), the correlation values are, respectively, 0.94, 1 and 0.45. The value of 0.45 is a consequence of the 2% of the genes with overdominance because, by altering the degree of dominance in these loci to complete dominance, the correlation value increases to 0.83.

The consequence of the absence of correlation between the additive x additive epistatic genetic value and the number of favorable genes is that, also in the presence of epistasis, the efficiency of mass selection is proportional to the correlation between the phenotypic value of the individual, the value used in the selection, and its additive genetic value. This correlation is equal to the square root of the narrow-sense heritability at the individual level (Viana, 2002). If the selection units are half sib families, the efficiency of among-family selection is proportional to the correlation between the mean phenotypic value of the progeny and the additive genetic value of the common parent, that is equal to the square root of the narrow-sense heritability at the family level (Viana, 2002), because the correlation between the number of genes that increase the trait expression in the common parent (N) and the mean frequency of these genes in the family ($\bar{p}$) is:

Table 1 - Correlations between the number of genes that increase the trait expression (N) and the additive genetic value (A) and the genotypic value (G; assuming absence of epistasis), considering three genes, different populations (p is the frequency of the dominant gene) and four degrees of dominance.

p_a	p_b	p_c	$|d/a| = 2$		$|d/a| = 1$		$|d/a| = 1/2$		$|d/a| = 0$	
			$\rho_{N,A}$	$\rho_{N,G}$	$\rho_{N,A}$	$\rho_{N,G}$	$\rho_{N,A}$	$\rho_{N,G}$	$\rho_{N,A}$	$\rho_{N,G}$
0.1	0.1	0.1	1.0000	0.9507	1.0000	0.9733	1.0000	0.9887	1.0000	1.0000
0.1	0.1	0.5	0.9041	0.7562	0.9590	0.8795	0.9860	0.9553	1.0000	1.0000
0.1	0.1	0.9	0.7129	0.6631	0.8592	0.8257	0.9488	0.9342	1.0000	1.0000
0.1	0.5	0.5	0.9077	0.6482	0.9687	0.8380	0.9909	0.9455	1.0000	1.0000
0.1	0.5	0.9	0.6948	0.5318	0.8881	0.7823	0.9681	0.9291	1.0000	1.0000
0.1	0.9	0.9	0.2955	0.2603	0.6971	0.6465	0.9169	0.8947	1.0000	1.0000
0.5	0.5	0.5	1.0000	0.5774	1.0000	0.8165	1.0000	0.9428	1.0000	1.0000
0.5	0.5	0.9	0.7958	0.4594	0.9503	0.7686	0.9885	0.9319	1.0000	1.0000
0.5	0.9	0.9	0.3860	0.2228	0.8600	0.6774	0.9730	0.9174	1.0000	1.0000
0.9	0.9	0.9	-1.0000	-0.5774	1.0000	0.4264	1.0000	0.9428	1.0000	1.0000

$$\rho_{N,\bar{p}} = \frac{\dfrac{1}{2k}\displaystyle\sum_{i=1}^{k} p_i q_i}{\sqrt{\left(2\displaystyle\sum_{i=1}^{k} p_i q_i\right)\left(\dfrac{1}{8k^2}\displaystyle\sum_{i=1}^{k} p_i q_i\right)}} = 1$$

(The correlation considering the number of genes that decreases the expression of the trait is -1).

When the selection units are full sib families, the efficiency of among-family selection is proportional to the correlation between the mean phenotypic value of the progeny and the mean of the additive genetic values of the parents, that is equal to the square root of the narrow-sense heritability at the family level (Viana, 2002), because the correlation between the mean number of genes that increases the trait expression ($\overline{N}$) and the mean frequency of these genes in the family ($\bar{p}$) is:

$$\rho_{\overline{N},\bar{p}} = \frac{\dfrac{1}{2k}\displaystyle\sum_{i=1}^{k} p_i q_i}{\sqrt{\left(\displaystyle\sum_{i=1}^{k} p_i q_i\right)\left(\dfrac{1}{4k^2}\displaystyle\sum_{i=1}^{k} p_i q_i\right)}} = 1$$

(The correlation considering the number of genes that decrease the trait expression is -1).

In the case of selection within non-inbred families, the efficiency is proportional to the mean of the coefficients of correlation between the phenotypic and the additive genetic values of the individual, as calculated for each family. This is equal to the square root of the narrow-sense heritability at the plant within family level. Therefore, when considering a non-inbred population, the numerator for all narrow-sense heritabilities must be a function of the additive genetic variance, since the genetic values due to dominance and epistasis do not correlate with the number of favorable genes that an individual possesses.

In many situations, depending on the selection unit and on the experimental data, it is only possible to assess the efficiency of a selective process from the broad-sense heritability, which is equal to the square of the correlation between the phenotypic and genotypic values of the selection unit (Viana, 2002). In these cases, as this heritability is greater than the corresponding narrow-sense heritability, the efficiency of selection is in general overestimated because of the lower value of the correlation between the genotypic value of the individual and the number of favorable genes present.

With complementary epistasis (Viana, 2004), only in relation to simpler polygenic systems with up to ten genes, 50% of which interact, and in populations with intermediary frequencies of the dominant genes, the correlation between the number of genes that increase the trait expression and the genotypic value of the individual assumes a value equal or superior to 0.7. (Table 2). Regardless of the type of epistasis, $\rho_{N,A} = 1$, assuming $p_i = p$ for all i. In the case of duplicate epistasis (Viana, 2004), only for polygenic systems with up to 100 genes, of which at most 20% interact, and in populations with low frequencies of the dominant genes, the correlation value is greater than or equal to 0.7.

Table 2 - Correlation values between the number of genes that increase the trait expression and the genotypic value of the individual, considering four types of epistasis, for different polygenic systems (k is the number of genes and k' is the number of genes that interact) and populations (p is the frequency of the dominant genes).

k	k'	p	Complementary epistasis	Duplicate epistasis	Dominant and recessive epistasis	Duplicate genes with cumulative effects
2	2	0.1	0.5500	0.9208	0.8511	0.2452
10	2	0.1	0.8150	0.9621	0.9447	0.4896
10	5	0.1	0.4243	0.8760	0.4990	0.1762
10	10	0.1	0.2166	0.6789	0.2368	0.0841
100	20	0.1	0.3227	0.8095	0.3357	0.1288
100	50	0.1	0.1334	0.4945	0.1357	0.0511
100	100	0.1	0.0668	0.2741	0.0674	0.0255
1000	200	0.1	0.1051	0.4088	0.1055	0.0401
1000	500	0.1	0.0422	0.1769	0.0422	0.0160
1000	1000	0.1	0.0211	0.0895	0.0211	0.0080
2	2	0.5	0.7559	0.5164	0.6963	0.7878
10	2	0.5	0.8032	0.7161	0.7875	0.8105
10	5	0.5	0.7071	0.4082	0.6667	0.7620
10	10	0.5	0.5164	0.2144	0.4815	0.6325
100	20	0.5	0.6389	0.3154	0.6239	0.7213
100	50	0.5	0.3621	0.1329	0.3551	0.4867
100	100	0.5	0.1952	0.0668	0.1929	0.2828

Table 2 (Cont.)

k	k'	p	Complementary epistasis	Duplicate epistasis	Dominant and recessive epistasis	Duplicate genes with cumulative effects
1000	200	0.5	0.2955	0.1048	0.2939	0.4109
1000	500	0.5	0.1251	0.0421	0.1248	0.1850
1000	1000	0.5	0.0631	0.0211	0.0630	0.0943
2	2	0.9	0.4253	0.0600	0.4222	0.4261
10	2	0.9	0.4262	0.1292	0.4256	0.4263
10	5	0.9	0.4243	0.0426	0.4218	0.4259
10	10	0.9	0.4170	0.0202	0.4118	0.4240
100	20	0.9	0.4224	0.0310	0.4212	0.4254
100	50	0.9	0.4022	0.0122	0.3997	0.4198
100	100	0.9	0.3482	0.0061	0.3448	0.4016
1000	200	0.9	0.3891	0.0096	0.3881	0.4159
1000	500	0.9	0.2836	0.0038	0.2827	0.3709
1000	1000	0.9	0.1734	0.0019	0.1730	0.2820

Admitting dominant and recessive epistasis of a gene with the other (k' - 1) and complementary interaction for the other k'(k' - 1)/2 pairs (Viana, 2004), where k' is the number of genes that interact, the correlation value tends to be greater than or equal to 0.7 only in simpler genetic systems (with up to 10 genes, of which at most 50% interact, and in populations with low to intermediate frequencies of the dominant genes). Assuming duplicate genes with cumulative effects (Viana, 2004), the correlation will also be greater than or equal to 0.7 in genetic systems with up to 10 genes, of which at most 50% interact, and in populations with intermediate frequencies of the dominant genes. The correlation values in the case of duplicate genes with cumulative effects were obtained by assuming, for each pair of interacting genes, that the parameter a is equal to the epistatic genetic value of the homozygote for the genes that decrease the trait expression (Viana, 2004). Generally, the correlation is inversely proportional to the relation between a and that epistatic value. When considering $a_i \approx a$ for all i, the correlation values with recessive epistasis are close to those with complementary epistasis; the values with dominant epistasis are close to those with duplicate gene action; and the values with non-epistatic gene interaction are close to those with duplicate genes with cumulative effects.

If there is dominance, but no epistasis, the lower the degree of dominance, the closer the $\rho_{N,G}$ and $\rho_{N,A}$ values (Table 1). Except for the gene systems with predominantly overdominance interaction and in populations with high frequencies of the dominant genes, the values are close, indicating that, generally, it is not problematic to assess the efficiency of selection of individuals and progenies based on the broad-sense heritability. The predicted genetic gain, however, will have an additional bias, proportional to the variance due to dominance.

When there is digenic epistasis, if the selection units are half sib progenies, the additive variance of the additive-dominant model is:

$$\sigma_A^{2*} = 4\sigma_{Ga}^2 = \sigma_A^2 + \frac{1}{4}\sigma_{AA}^2$$

where σ_{Ga}^2 is the among family genotypic variance.

With full sib families the value is:

$$\sigma_A^{2*} = 3\sigma_{Ga}^2 - \sigma_{Gw}^2 = \sigma_A^2 - \frac{1}{2}\left(\sigma_{AD}^2 + \sigma_{DA}^2\right) - \frac{3}{4}\sigma_{DD}^2$$

where σ_{Gw}^2 is the within family genotypic variance.

Regardless of the type of epistasis, the bias in the estimation of the additive variance tends to be considerable (Table 3). The very high magnitudes are mainly the reflection of the assumption of equality of the gene frequencies at all the loci. The values are generally lower when two genes and different gene frequencies are considered (Table 4). With complementary, recessive, and dominant and recessive epistasis, duplicate genes with cumulative effects and non-epistatic gene interaction, the bias is generally less than 10% only in simple polygenic systems, with up to ten genes, of which at most 20% interact, and in populations with intermediate to high frequencies of the dominant genes. In the case of duplicate gene action and dominant epistasis, the bias is only less than 10% for simpler polygenic systems, with up to ten genes, of which at most 20% interact, and in populations with low frequencies of the dominant genes. Consequently, the narrow-sense heritabilities at the half sib family mean, plant within family and plant levels are overestimated. With full sib families, the narrow-sense heritabilities at the progeny mean, individual within family and individual levels are underes-

Table 3 - Bias (%) in the estimate of the additive variance because of the fitting of the additive-dominant model when there is digenic epistasis, considering half sib (HS) and full sib (FS) progenies, for different types of epistasis, polygenic systems (k is the number of genes and k' is the number of genes that interact) and populations (p is the frequency of the dominant genes).

k	k'	p	Complementary epistasis		Duplicate epistasis		Dominant and recessive epistasis		Duplicate genes with cumulative effects	
			HS	FS	HS	FS	HS	FS	HS	FS
2	2	0.1	50.5	-11.7	2.8	-0.6	7.3	-1.7	349.6	-80.9
10	2	0.1	10.1	-2.3	0.6	-0.1	1.5	-0.3	69.9	-16.2
10	5	0.1	101.0	-23.4	5.6	-1.3	66.4	-15.4	699.2	-161.9
10	10	0.1	454.4	-105.2	25.0	-5.8	376.6	-87.2	3146.6	-728.4
100	20	0.1	191.8	-44.4	10.6	-2.4	175.4	-40.6	1328.5	-307.5
100	50	0.1	1236.9	-286.3	68.1	-15.8	1194.5	-276.5	8565.6	-1982.8
100	100	0.1	4998.0	-1156.9	275.0	-63.7	4912.5	-1137.1	34612.1	-8012.1
1000	200	0.1	2009.3	-465.1	110.6	-25.6	1992.1	-461.1	13914.8	-3221.0
1000	500	0.1	12595.9	-2915.7	693.1	-160.4	12552.8	-2905.8	87229.6	-20192.0
1000	1000	0.1	50434.3	-11674.6	2775.0	-642.4	50348.0	-11654.6	349267.9	-80849.0
2	2	0.5	2.8	-7.6	25.0	-68.8	6.3	-17.2	1.2	-3.4
10	2	0.5	0.6	-1.5	5.0	-13.8	1.3	-3.4	0.2	-0.7
10	5	0.5	5.6	-15.3	50.0	-137.5	8.3	-22.9	2.5	-6.8
10	10	0.5	25.0	-68.8	225.0	-618.8	31.3	-85.9	11.1	-30.6
100	20	0.5	10.6	-29.0	95.0	-261.3	11.9	-32.7	4.7	-12.9
100	50	0.5	68.1	-187.2	612.5	-1684.4	71.5	-196.5	30.2	-83.2
100	100	0.5	275.0	-756.3	2475.0	-6806.3	281.9	-775.2	122.2	-336.1
1000	200	0.5	110.6	-304.0	995.0	-2736.3	111.9	-307.8	49.1	-135.1
1000	500	0.5	693.1	-1905.9	6237.5	-17153.1	696.5	-1915.4	308.0	-847.1
1000	1000	0.5	2775.0	-7631.3	24975.0	-68681.3	2781.9	-7650.3	1233.3	-3391.7
2	2	0.9	0.0	-1.8	225.0	-17718.8	0.1	-7.1	0.0	-0.5
10	2	0.9	0.0	-0.4	45.0	-3543.8	0.0	-1.4	0.0	-0.1
10	5	0.9	0.0	-3.6	450.0	-35437.5	0.1	-7.8	0.0	-0.9
10	10	0.9	0.2	-16.3	2025.0	-159468.8	0.3	-25.8	0.1	-4.2
100	20	0.9	0.1	-6.9	855.0	-67331.3	0.1	-8.9	0.0	-1.8
100	50	0.9	0.6	-44.3	5512.5	-434109.4	0.6	-49.5	0.1	-11.3
100	100	0.9	2.3	-179.0	22275.0	-1754156.3	2.4	-189.4	0.6	-45.7
1000	200	0.9	0.9	-72.0	8955.0	-705206.3	0.9	-74.1	0.2	-18.4
1000	500	0.9	5.7	-451.1	56137.5	-4420828.1	5.8	-456.3	1.5	-115.1
1000	1000	0.9	22.9	-1806.0	224775.0	-17701031	23.1	-1816.6	5.8	-460.7

Table 4 - Bias (%) in the estimate of the additive variance because of the fitting of the additive-dominant model when there is digenic epistasis, considering two genes, half sib (HS) and full sib (FS) progenies, for different types of epistasis and populations (p is the frequency of the dominant genes).

Epistasis	p_A	p_B	HS	FS	Epistasis	p_A	p_B	HS	FS
Complementary	0.1	0.3	16.54	-9.52	Duplicate	0.1	0.3	4.41	-2.54
	0.1	0.6	2.65	-4.60		0.1	0.6	5.17	-8.98
	0.1	0.9	0.05	-0.45		0.1	0.9	5.49	-54.12
	0.5	0.1	5.27	-6.29		0.5	0.1	5.00	-5.97
	0.5	0.9	0.05	-0.76		0.5	0.9	45.00	-753.75
	0.9	0.7	0.04	-1.20		0.9	0.7	92.65	-2509.19
	0.9	0.4	0.05	-0.65		0.9	0.4	31.03	-439.66

Table 4 (Cont.)

Epistasis	p_A	p_B	HS	FS		Epistasis	p_A	p_B	HS	FS
Dominant and recessive	0.1	0.3	4.34	-2.50		Duplicate genes with cumulative effects	0.1	0.3	31.92	-18.37
	0.1	0.6	1.83	-3.17			0.1	0.6	3.66	-6.36
	0.1	0.9	0.05	-0.45			0.1	0.9	0.06	-0.58
	0.5	0.1	33.44	-39.94			0.5	0.1	7.73	-9.24
	0.5	0.9	0.05	-0.77			0.5	0.9	0.06	-0.98
	0.9	0.7	1.14	-30.83			0.9	0.7	0.06	-1.54
	0.9	0.4	10.31	-145.99			0.9	0.4	0.06	-0.83

timated. The predicted gains will, therefore, have an additional bias, proportional to that of the heritability.

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