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Relative importance of the epistatic components of genotypic variance in non-inbred populations

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ABSTRACT - *There are theoretical approaches about genic interaction in polygenic systems and methodologies to confirm its occurrence. However, some relevant aspects as the relative importance of the epistatic components of genotypic variance deserve further investigation. Considering complementary, duplicate, recessive, dominant, dominant and recessive epistasis, duplicate genes with cumulative effects and non-epistatic genic interaction, relative magnitudes of the epistatic components of genotypic variance assuming digenic epistasis were analyzed. Regardless of the type of epistasis and gene frequencies, the magnitudes of the epistatic components are proportional to the complexity of the polygenic system, the number of interacting genes and the magnitude of the epistatic effects relative to the deviations* ***a*** *(difference between the genotypic value of the homozygote with greater expression and the mean of the genotypic values of the homozygotes) and* ***d*** *(due to dominance). Only in simple genetic systems or in those where complementary, recessive, dominant and recessive, duplicate genes with cumulative effects, or non-epistatic gene interaction types of epistasis predominate, with high frequencies of dominant genes, epistatic components can be of reduced or negligible magnitude.*

Key words: *genic interaction, digenic epistasis, genetic parameters, non-inbred populations.*

INTRODUCTION

Although from the biological point of view the presence of epistasis is the rule and not the exception, as has been shown in many studies on the inheritance of qualitative traits, its study in quantitative genetic systems is one of the most complex problems faced by geneticists. This is not due to lack of theoretical knowledge, that was fully established almost 50 years ago by Cockerham (1954) and Kempthorne (1955), nor to the lack of methodologies, such as generation mean analysis (Mather and Jinks 1974) and triple test cross (Kearsey and Pooni 1996), but rather to the infinite possibilities of genetic systems considering the combinations of degrees of dominance, gene frequencies and number of genes. Without exception, in the top technical publications on Quantitative Genetics (Kempthorne 1973, Hallauer and Miranda Filho 1988, Wricke and Weber 1986, Comstock 1996, Lynch and Walsh 1996) and in other published studies of quantitative trait inheritance that include epistasis, there is no assessment of relative importance of the epistatic components of genotypic variance, or of their contribution to gains from selection or of the bias in estimating genetic parameters due to fitting the simple additive-dominant model. The objective of this study was to contribute with theoretical information on the relative magnitudes of the epistatic components of genotypic variance, considering digenic epistasis.

METHODS

All theory of polygenic systems that include interaction effects between non-allelic genes was developed by Cockerham (1954) and Kempthorne (1955), although some differences in modeling exist. In those two articles, the components of genotypic variance assuming epistasis were described. Furthermore, the authors developed expressions describing the covariance between genotypic values of relatives. Cockerham´s model is the regression of the genotypic value of an individual (G) as function of the

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number of genes that increase the trait expression (X_1 and X_2), which can be defined as

$$G = \beta_0 + \beta_1 X_1 + \beta_2 X_1^2 + \beta_3 X_2 + \beta_4 X_2^2 + \beta_5 X_1 X_2 + \beta_6 X_1 X_2^2 + \beta_7 X_1^2 X_2 + \beta_8 X_1^2 X_2^2$$

In matrix terms

$$\begin{bmatrix} G_{22} \\ G_{12} \\ G_{02} \\ G_{21} \\ G_{11} \\ G_{01} \\ G_{20} \\ G_{10} \\ G_{00} \end{bmatrix} = \begin{bmatrix} 1 & 2 & 4 & 2 & 4 & 4 & 8 & 8 & 16 \\ 1 & 1 & 1 & 2 & 4 & 2 & 4 & 2 & 4 \\ 1 & 0 & 0 & 2 & 4 & 0 & 0 & 0 & 0 \\ 1 & 2 & 4 & 1 & 1 & 2 & 2 & 4 & 4 \\ 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 & 1 \\ 1 & 0 & 0 & 1 & 1 & 0 & 0 & 0 & 0 \\ 1 & 2 & 4 & 0 & 0 & 0 & 0 & 0 & 0 \\ 1 & 1 & 1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix} \begin{bmatrix} \beta_0 \\ \beta_1 \\ \beta_2 \\ \beta_3 \\ \beta_4 \\ \beta_5 \\ \beta_6 \\ \beta_7 \\ \beta_8 \end{bmatrix}$$

where G_{ij} is the genotypic value of the carrier of i copies of gene A and j copies of gene B (i, j = 0, 1 or 2) which increase trait expression.

Orthogonalizing the columns of X (Q) by the orthogonal polynomials technique, results in

$$P_0 = Q_0$$

$$P_1 = Q_1 - \frac{Q_1' A P_0}{P_0' A P_0} \cdot P_0$$

$$P_2 = Q_2 - \frac{Q_2' A P_0}{P_0' A P_0} \cdot P_0 - \frac{Q_2' A P_1}{P_1' A P_1} \cdot P_1$$

...

$$P_8 = Q_8 - \frac{Q_8' A P_0}{P_0' A P_0} \cdot P_0 - \frac{Q_8' A P_1}{P_1' A P_1} \cdot P_1 - \frac{Q_8' A P_2}{P_2' A P_2} \cdot P_2 - \frac{Q_8' A P_3}{P_3' A P_3} \cdot P_3 - \frac{Q_8' A P_4}{P_4' A P_4} \cdot P_4 - \frac{Q_8' A P_5}{P_5' A P_5} \cdot P_5 - \frac{Q_8' A P_6}{P_6' A P_6} \cdot P_6 - \frac{Q_8' A P_7}{P_7' A P_7} \cdot P_7$$

where A is a diagonal matrix of probabilities of the genotypes (f_{ij}). Assuming non-inbred population in Hardy-Weinberg equilibrium and in linkage equilibrium,

$$\begin{aligned} f_{22} &= p_a^2 p_b^2 \\ f_{12} &= 2 p_a q_a p_b^2 \\ f_{02} &= q_a^2 p_b^2 \\ f_{21} &= 2 p_a^2 p_b q_b \\ f_{11} &= 4 p_a q_a p_b q_b \\ f_{01} &= 2 q_a^2 p_b q_b \\ f_{20} &= p_a^2 q_b^2 \\ f_{10} &= 2 p_a q_a q_b^2 \\ f_{00} &= q_a^2 q_b^2 \end{aligned}$$

where p is the frequency of the gene that increases the trait expression.

The transformed X matrix is

$$\begin{bmatrix} 1 & 2q_a & 2q_a^2 & 2q_b & 2q_b^2 & 4q_a q_b & 4q_a q_b^2 & 4q_a^2 q_b & 4q_a^2 q_b^2 \\ 1 & q_a - p_a & -2p_a q_a & 2q_b & 2q_b^2 & 2(q_a - p_a) q_b & 2(q_a - p_a) q_b^2 & -4p_a q_a q_b & -4p_a q_a q_b^2 \\ 1 & -2p_a & 2p_a^2 & 2q_b & 2q_b^2 & -4p_a q_b & -4p_a q_b^2 & 4p_a^2 q_b & 4p_a^2 q_b^2 \\ 1 & 2q_a & 2q_a^2 & q_b - p_b & -2p_b q_b & 2q_a (q_b - p_b) & -4q_a p_b q_b & 2q_a^2 (q_b - p_b) & -4q_a^2 p_b q_b \\ 1 & q_a - p_a & -2p_a q_a & q_b - p_b & -2p_b q_b & (q_a - p_a)(q_b - p_b) & -2(q_a - p_a) p_b q_b & -2p_a q_a (q_b - p_b) & 4p_a q_a p_b q_b \\ 1 & -2p_a & 2p_a^2 & q_b - p_b & -2p_b q_b & -2p_a (q_b - p_b) & 4p_a p_b q_b & 2p_a^2 (q_b - p_b) & -4p_a^2 p_b q_b \\ 1 & 2q_a & 2q_a^2 & -2p_b & 2p_b^2 & -4q_a p_b & 4q_a p_b^2 & -4q_a^2 p_b & 4q_a^2 p_b^2 \\ 1 & q_a - p_a & -2p_a q_a & -2p_b & 2p_b^2 & -2(q_a - p_a) p_b & 2(q_a - p_a) p_b^2 & 4p_a q_a p_b & -4p_a q_a p_b^2 \\ 1 & -2p_a & 2p_a^2 & -2p_b & 2p_b^2 & 4p_a p_b & -4p_a p_b^2 & -4p_a^2 p_b & 4p_a^2 p_b^2 \end{bmatrix}$$

The genotypic values are

$$\begin{aligned} G_{22} &= (m_a + a_a) + (m_b + a_b) + I_{22} = (M_a + A_{AA} + D_{AA}) + (M_b + A_{BB} + D_{BB}) + I_{22} \\ G_{12} &= (m_a + d_a) + (m_b + a_b) + I_{12} = (M_a + A_{Aa} + D_{Aa}) + (M_b + A_{BB} + D_{BB}) + I_{12} \\ G_{02} &= (m_a - a_a) + (m_b + a_b) + I_{02} = (M_a + A_{aa} + D_{aa}) + (M_b + A_{BB} + D_{BB}) + I_{02} \\ G_{21} &= (m_a + a_a) + (m_b + d_b) + I_{21} = (M_a + A_{AA} + D_{AA}) + (M_b + A_{Bb} + D_{Bb}) + I_{21} \\ G_{11} &= (m_a + d_a) + (m_b + d_b) + I_{11} = (M_a + A_{Aa} + D_{Aa}) + (M_b + A_{Bb} + D_{Bb}) + I_{11} \\ G_{01} &= (m_a - a_a) + (m_b + d_b) + I_{01} = (M_a + A_{aa} + D_{aa}) + (M_b + A_{Bb} + D_{Bb}) + I_{01} \\ G_{20} &= (m_a + a_a) + (m_b - a_b) + I_{20} = (M_a + A_{AA} + D_{AA}) + (M_b + A_{bb} + D_{bb}) + I_{20} \\ G_{10} &= (m_a + d_a) + (m_b - a_b) + I_{10} = (M_a + A_{Aa} + D_{Aa}) + (M_b + A_{bb} + D_{bb}) + I_{10} \\ G_{00} &= (m_a - a_a) + (m_b - a_b) + I_{00} = (M_a + A_{aa} + D_{aa}) + (M_b + A_{bb} + D_{bb}) + I_{00} \end{aligned}$$

where, for each gene, **m** is the mean of the genotypic values of the homozygotes, **a** is the deviation between the genotypic value of the homozygote with greatest expression and **m**, **d** is the deviation due to dominance, **M** is the population mean, **A** is additive genetic value and **D** is genetic value due to dominance. Regarding one gene, M = m + (p – q)a + 2pqd, A = 2qα and D = $-2q^2$d, if the individual is homozygous for the gene that increases the trait expression, or A = (q – p)α and D = 2pqd, if the individual is heterozygous, or A = $-2p\alpha$ and D = $-2p^2$d, if the individual is homozygous for the gene that decreases the trait expression, where α is the average effect of a gene substitution (Falconer and Mackay 1996).

Assuming in relation to the epistatic genetic values (I),

$$\begin{aligned} \bar{I}_{2.} &= p_b^2 I_{22} + 2p_b q_b I_{21} + q_b^2 I_{20} = \bar{I}_{1.} = \bar{I}_{0.} = 0 \\ \bar{I}_{.2} &= p_a^2 I_{22} + 2p_a q_a I_{12} + q_a^2 I_{02} = \bar{I}_{.1} = \bar{I}_{.0} = 0 \\ \bar{I}_{..} &= p_a^2 p_b^2 I_{22} + \cdots + q_a^2 q_b^2 I_{00} = 0 \end{aligned}$$

the reductions in the total sum of squares are

$$R(\beta_0) = \frac{\left(P_0' A Y\right)^2}{P_0' A P_0} = M^2$$

$$R(\beta_1 | \beta_0) = \frac{\left(P_1' A Y\right)^2}{P_1' A P_1} = 2p_a q_a \alpha_a^2 = \sigma^2_{A_a}$$

$$R(\beta_2 | \beta_0, \beta_1) = \frac{\left(P_2' A Y\right)^2}{P_2' A P_2} = 4p_a^2 q_a^2 d_a^2 = \sigma^2_{D_a}$$

$$R(\beta_3 | \beta_0) = \frac{\left(P_3' A Y\right)^2}{P_3' A P_3} = 2p_b q_b \alpha_b^2 = \sigma^2_{A_b}$$

$$R(\beta_4 | \beta_0, \beta_3) = \frac{\left(P_4' A Y\right)^2}{P_4' A P_4} = 4p_b^2 q_b^2 d_b^2 = \sigma^2_{D_b}$$

$$R(\beta_5 \mid \beta_0, \beta_1, \beta_3) = 4p_a q_a p_b q_b \left(p_a p_b e_{22} + p_a q_b e_{21} + q_a p_b e_{12} + q_a q_b e_{11}\right)^2 = \sigma^2_{AA}$$

$$R(\beta_6 \mid \beta_0, \beta_1, \beta_3, \beta_4, \beta_5) = 2p_a q_a p_b^2 q_b^2 \left(p_a e_{22} - p_a e_{21} + q_a e_{12} - q_a e_{11}\right)^2 = \sigma^2_{AD}$$

$$R(\beta_7 \mid \beta_0, \beta_1, \beta_2, \beta_3, \beta_5) = 2p_a^2 q_a^2 p_b q_b \left(p_b e_{22} + q_b e_{21} - p_b e_{12} - q_b e_{11}\right)^2 = \sigma^2_{DA}$$

$$R(\beta_8 \mid \beta_0, \beta_1, \beta_2, \beta_3, \beta_4, \beta_5, \beta_6, \beta_7) = p_a^2 q_a^2 p_b^2 q_b^2 \left(e_{22} - e_{21} - e_{12} + e_{11}\right)^2 = \sigma^2_{DD}$$

where

$$e_{22} = G_{22} - G_{21} - G_{12} + G_{11} = I_{22} - I_{21} - I_{12} + I_{11}$$
$$e_{21} = G_{21} - G_{20} - G_{11} + G_{10} = I_{21} - I_{20} - I_{11} + I_{10}$$
$$e_{12} = G_{12} - G_{11} - G_{02} + G_{01} = I_{12} - I_{11} - I_{02} + I_{01}$$
$$e_{11} = G_{11} - G_{10} - G_{01} + G_{00} = I_{11} - I_{10} - I_{01} + I_{00}$$

The genotypic variance is

$$\sigma^2_G = Y'AY - R(\beta_0) = \sigma^2_A + \sigma^2_D + \sigma^2_{AA} + \sigma^2_{AD} + \sigma^2_{DA} + \sigma^2_{DD}$$

Considering k genes,

$$\sigma^2_{AA} = 4\sum_{i=1}^{k}\sum_{j=1}^{k} p_i q_i p_j q_j \left(p_i p_j e22_{ij} + p_i q_j e21_{ij} + q_i p_j e12_{ij} + q_i q_j e11_{ij}\right)^2$$

$$\sigma^2_{AD} = 2\sum_{i=1}^{k}\sum_{j=1}^{k} p_i q_i p_j^2 q_j^2 \left(p_i e22_{ij} - p_i e21_{ij} + q_i e12_{ij} - q_i e11_{ij}\right)^2$$

$$\sigma^2_{DA} = 2\sum_{i=1}^{k}\sum_{j=1}^{k} p_i^2 q_i^2 p_j q_j \left(p_j e22_{ij} + q_j e21_{ij} - p_j e12_{ij} - q_j e11_{ij}\right)^2$$

$$\sigma^2_{DD} = \sum_{i=1}^{k}\sum_{j=1}^{k} p_i^2 q_i^2 p_j^2 q_j^2 \left(e22_{ij} - e21_{ij} - e12_{ij} + e11_{ij}\right)^2$$

where σ^2_{AA}, σ^2_{AD}, σ^2_{DA} and σ^2_{DD} are the additive x additive, additive x dominant, dominant x additive and dominant x dominant epistatic genetic variances, respectively.

Types of Epistasis

The complexity of the polygenic systems makes any theoretical study of epistasis very difficult because there are infinite combinations of gene frequencies and degrees of dominance. To simplify, known types of digenic epistasis will be considered, characterized as follows:

· Complementary epistasis (9:7): $a_i = d_i = a$ and $p_i = p$, for every i, and

$$I_{00} = 2\left(1 - q^2\right)a$$
$$I_{20} = I_{10} = I_{02} = I_{01} = -2q^2 a$$
$$I_{22} = I_{21} = I_{12} = I_{11} = \frac{2q^4 a}{1 - q^2}$$

· Duplicate epistasis (15:1): $a_i = d_i = a$ and $p_i = p$, for every i, and

$$I_{00} = -\frac{2\left(1 - q^2\right)^2 a}{q^2}$$
$$I_{20} = I_{10} = I_{02} = I_{01} = 2\left(1 - q^2\right)a$$
$$I_{22} = I_{21} = I_{12} = I_{11} = -2q^2 a$$

· Dominant and recessive epistasis (13:3): $a_a = d_a = -a_b = -d_b$, $p_a = p_b = p$ and

$$I_{00} = I_{22} = I_{21} = I_{12} = I_{11} = a_a$$
$$I_{20} = I_{10} = -a_a$$
$$I_{02} = I_{01} = \theta a_a$$

where θ is a coefficient of proportionality.

· Recessive epistasis (9:3:4): $a_a = d_a = \theta' a_b = \theta' d_b$, where θ' is a coefficient of proportionality, different from 1, $p_a = p_b = p$ and

$$I_{00} = 2\left(1 - q^2\right)a_b$$
$$I_{20} = I_{10} = I_{02} = I_{01} = -2q^2 a_b$$
$$I_{22} = I_{21} = I_{12} = I_{11} = \frac{2q^4 a_b}{1 - q^2}$$

· Dominant epistasis (12:3:1): $a_a = d_a = \theta' a_b = \theta' d_b$, $p_a = p_b = p$ and

$$I_{00} = -\frac{2\left(1 - q^2\right)^2 a_b}{q^2}$$
$$I_{20} = I_{10} = I_{02} = I_{01} = 2\left(1 - q^2\right)a_b$$
$$I_{22} = I_{21} = I_{12} = I_{11} = -2q^2 a_b$$

· Duplicate genes with cumulative effects (9:6:1): $a_i = d_i$ e $p_i = p$, for every i, and

$$I_{00} = \theta a$$
$$I_{20} = I_{10} = I_{02} = I_{01} = -\frac{q^2 \theta a}{1 - q^2}$$
$$I_{22} = I_{21} = I_{12} = I_{11} = \frac{q^4 \theta a}{\left(1 - q^2\right)^2}$$

where θ is a proportionality coefficient.

· Non epistatic genic interaction (9:3:3:1): $a_a = d_a = \theta' a_b = \theta' d_b$, $p_a = p_b = p$ and

$$I_{00} = \theta a_a$$
$$I_{20} = I_{10} = I_{02} = I_{01} = -\frac{q^2 \theta a_a}{1 - q^2}$$
$$I_{22} = I_{21} = I_{12} = I_{11} = \frac{q^4 \theta a_a}{\left(1 - q^2\right)^2}$$

RESULTS AND DISCUSSION

Assuming complementary epistasis and population with reduced frequencies of dominant genes (Table 1), the additive x additive epistatic genetic variance is, in general, the most important component of the genotypic variance independently of the polygenic system, surpassing the additive and dominance genetic variances. Only in simpler systems, with few interacting genes, the additive variance can be the component of largest magnitude. In spite of complete dominance, the variances due to dominance and additive x dominant, dominant x additive, and dominant x dominant epistatic effects represent at most 10% of the genotypic variance. In populations with intermediate allelic frequencies (Table 1) and in polygenic systems with up to 100 genes, if about 20% of these genes interact, the additive variance is the component of largest magnitude of the genotypic variance. In more complex systems, it is surpassed by the additive x additive variance. Comparatively to the populations with low frequencies of dominant genes, there is, in general, a decrease in the magnitude of the additive x additive epistatic variance and an increase in the magnitude of the other components of the genotypic variance. Even so, it continues to be the most important component of the epistatic variance. Magnitudes of the epistatic variances are lower. If the frequencies of dominant genes in the population are high (Table 1), with the exception of the very complex polygenic systems with at least 1000 genes, of which at least 50% interact, the variance due to dominance is the component of largest magnitude of the genotypic variance. The most relevant component of the epistatic variance is the dominant x dominant variance. With complementary epistasis, the magnitude of the epistatic variance is maximized when the frequencies of the dominant genes are reduced and minimized when the frequencies of the dominant genes are high. When the gene frequencies are intermediary, the additive x additive variance has the same magnitude as the sum of the additive x dominant and dominant x additive epistatic variances. Regardless of the gene frequencies, the increase in the number of genes in the polygenic system and in the number of interacting genes will increase the magnitude of the epistatic variance, which can represent practically 100% of the genotypic variance.

Admitting duplicate epistasis (Table 2), if the frequencies of the dominant genes in the population are small, only in simpler polygenic systems of at most 100 genes, with about 20% of them interacting, the magnitude of the additive variance

Table 1. Relative magnitudes (%) of the components of genotypic variance, considering complementary epistasis, in relation to different polygenic systems and populations

k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
2	2	0.1	30.252	1.681	61.091	6.788	0.189	68.067
10	2	0.1	66.421	3.690	26.826	2.981	0.083	29.889
10	5	0.1	18.000	1.000	72.698	8.078	0.224	81.000
10	10	0.1	4.694	0.261	85.304	9.478	0.263	95.046
100	20	0.1	10.411	0.578	79.888	8.876	0.247	89.011
100	50	0.1	1.780	0.099	88.064	9.785	0.272	98.121
100	100	0.1	0.447	0.025	89.327	9.925	0.276	99.528
1000	200	0.1	1.104	0.061	88.705	9.856	0.274	98.835
1000	500	0.1	0.178	0.010	89.582	9.954	0.276	99.812
1000	1000	0.1	0.044	0.002	89.709	9.968	0.277	99.953
2	2	0.5	57.143	28.571	6.349	6.349	1.587	14.286
10	2	0.5	64.516	32.258	1.434	1.434	0.358	3.226
10	5	0.5	50.000	25.000	11.111	11.111	2.778	25.000
10	10	0.5	26.667	13.333	26.667	26.667	6.667	60.000
100	20	0.5	40.816	20.408	17.234	17.234	4.308	38.776
100	50	0.5	13.115	6.557	35.701	35.701	8.925	80.328
100	100	0.5	3.810	1.905	41.905	41.905	10.476	94.286
1000	200	0.5	8.734	4.367	38.622	38.622	9.656	86.900
1000	500	0.5	1.566	0.783	43.401	43.401	10.850	97.652
1000	1000	0.5	0.398	0.199	44.179	44.179	11.045	99.403
2	2	0.9	18.090	81.407	0.017	0.150	0.336	0.503
10	2	0.9	18.163	81.736	0.003	0.030	0.068	0.101
10	5	0.9	18.000	81.000	0.033	0.298	0.669	1.000
10	10	0.9	17.391	78.261	0.144	1.294	2.911	4.348
100	20	0.9	17.839	80.278	0.062	0.560	1.261	1.883
100	50	0.9	16.180	72.809	0.364	3.276	7.371	11.011
100	100	0.9	12.121	54.545	1.102	9.917	22.314	33.333
1000	200	0.9	15.139	68.124	0.553	4.980	11.204	16.737
1000	500	0.9	8.045	36.201	1.843	16.588	37.323	55.754
1000	1000	0.9	3.008	13.534	2.759	24.831	55.869	83.459

σ^2_I is the epistatic genetic variance; k is the number of genes and k' is the number of genes that interact; p is the frequency of the dominant genes.

will be equal or larger to the additive x additive variance. In more complex systems, the additive x additive variance is the largest component of the genotypic variance. Regarding populations with intermediary allelic frequencies, the additive x additive variance is the component of largest magnitude. Exceptions are simple systems of up to 10 genes, with about 20% of them interacting, where the additive x additive variance will be smaller than the additive variance (Table 2). In populations with high frequencies of dominant genes, the variance of largest magnitude is the dominant x dominant regardless of the polygenic system, representing 61 to 67% of the genotypic variance (Table 2). Contrary to that observed with complementary epistasis, the variance due to dominance is of negligible magnitude. With duplicate epistasis, an increase in the frequencies of dominant genes will increase the magnitude of the epistatic variance, which can represent almost 100% of the genotypic variance. In populations with intermediary allelic frequencies, the additive x additive variance is also equal to the sum of the additive x dominant and dominant x additive variances. As described for complementary epistasis, an increase in the trait control complexity and in the number of interacting genes can bring the proportion of the genotypic variance closer to 100% due to the differences among the epistatic genetic values of the individuals, regardless of the gene frequencies.

Compared to the previous cases, the analysis of duplicate genes with cumulative effects is more troublesome, because for each pair of interacting genes there is a proportionality between the **a** deviation, constant for every gene, and the epistatic genetic value of the homozygote for the genes that reduce the trait expression (θ). Assuming $\theta = 0.1$ and a population with reduced frequency of dominant genes, the additive x additive variance is the component of genotypic variance of largest magnitude in complex polygenic systems of at least 100 genes, with about 50% of them interacting. In simpler systems, the additive variance has a larger value (Table 3). When the allelic frequencies are intermediary, the additive variance is the component of largest magnitude followed by genetic variance due to dominance, which both correspond to practically 100% of the genotypic variance (Table 4), regardless of the polygenic system. In populations with high frequencies of dominant genes, the additive and dominance variances also represent practically 100% of the genetic variance, but the second component is the one of largest magnitude (at least 80%), independently of the polygenic

Table 2. Relative magnitudes (%) of the components of genotypic variance, considering duplicate epistasis, in relation to different polygenic systems and populations

k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
2	2	0.1	84.792	4.711	9.421	1.047	0.029	10.497
10	2	0.1	92.566	5.143	2.057	0.229	0.006	2.292
10	5	0.1	76.737	4.263	17.053	1.895	0.053	19.000
10	10	0.1	46.088	2.560	46.088	5.121	0.142	51.351
100	20	0.1	65.531	3.641	27.669	3.074	0.085	30.828
100	50	0.1	24.458	1.359	66.580	7.398	0.205	74.183
100	100	0.1	7.512	0.417	82.634	9.182	0.255	92.070
1000	200	0.1	16.715	0.929	73.916	8.213	0.228	82.357
1000	500	0.1	3.131	0.174	86.785	9.643	0.268	96.696
1000	1000	0.1	0.802	0.045	88.991	9.888	0.275	99.154
2	2	0.5	26.667	13.333	26.667	26.667	6.667	60.000
10	2	0.5	51.282	25.641	10.256	10.256	2.564	23.077
10	5	0.5	16.667	8.333	33.333	33.333	8.333	75.000
10	10	0.5	4.598	2.299	41.379	41.379	10.345	93.103
100	20	0.5	9.950	4.975	37.811	37.811	9.453	85.075
100	50	0.5	1.766	0.883	43.267	43.267	10.817	97.351
100	100	0.5	0.446	0.223	44.147	44.147	11.037	99.331
1000	200	0.5	1.098	0.549	43.712	43.712	10.928	98.353
1000	500	0.5	0.178	0.089	44.326	44.326	11.082	99.734
1000	1000	0.5	0.044	0.022	44.415	44.415	11.104	99.933
2	2	0.9	0.360	1.620	3.240	29.163	65.617	98.020
10	2	0.9	1.668	7.506	3.003	27.023	60.801	90.826
10	5	0.9	0.182	0.818	3.273	29.455	66.273	99.000
10	10	0.9	0.041	0.183	3.298	29.685	66.792	99.776
100	20	0.9	0.096	0.433	3.288	29.595	66.588	99.471
100	50	0.9	0.015	0.067	3.303	29.728	66.887	99.918
100	100	0.9	0.004	0.017	3.305	29.746	66.928	99.980
1000	200	0.9	0.009	0.042	3.304	29.737	66.908	99.949
1000	500	0.9	0.001	0.007	3.306	29.750	66.937	99.992
1000	1000	0.9	0.000	0.002	3.306	29.751	66.941	99.998

σ^2_I is the epistatic genetic variance; k is the number of genes and k' is the number of genes that interact; p is the frequency of the dominant genes.

Table 3. Relative magnitudes (%) of the components of genotypic variance, considering duplicate genes with cumulative effects and populations with reduced frequency of dominant genes (p), in different polygenic systems

θ	k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
0.1	2	2	0.1	82.551	4.586	11.545	1.283	0.036	12.863
0.1	10	2	0.1	92.020	5.112	2.574	0.286	0.008	2.868
0.1	10	5	0.1	73.143	4.063	20.458	2.273	0.063	22.794
0.1	10	10	0.1	40.685	2.260	51.207	5.690	0.158	57.055
0.1	100	20	0.1	60.692	3.372	32.253	3.584	0.100	35.936
0.1	100	50	0.1	20.521	1.140	70.310	7.812	0.217	78.339
0.1	100	100	0.1	6.067	0.337	84.003	9.334	0.259	93.595
0.1	1000	200	0.1	13.780	0.766	76.696	8.522	0.237	85.455
0.1	1000	500	0.1	2.504	0.139	87.378	9.709	0.270	97.357
0.1	1000	1000	0.1	0.638	0.035	89.146	9.905	0.275	99.326
1	2	2	0.1	6.011	0.334	84.056	9.340	0.259	93.655
1	10	2	0.1	23.970	1.332	67.042	7.449	0.207	74.698
1	10	5	0.1	3.104	0.172	86.810	9.646	0.268	96.724
1	10	10	0.1	0.708	0.039	89.080	9.898	0.275	99.253
1	100	20	0.1	1.659	0.092	88.179	9.798	0.272	98.249
1	100	50	0.1	0.261	0.015	89.503	9.945	0.276	99.724
1	100	100	0.1	0.065	0.004	89.689	9.965	0.277	99.932
1	1000	200	0.1	0.161	0.009	89.598	9.955	0.277	99.830
1	1000	500	0.1	0.026	0.001	89.726	9.970	0.277	99.973
1	1000	1000	0.1	0.006	0.000	89.745	9.972	0.277	99.993
2	2	2	0.1	1.578	0.088	88.256	9.806	0.272	98.335
2	10	2	0.1	7.396	0.411	82.744	9.194	0.255	92.193
2	10	5	0.1	0.795	0.044	88.997	9.889	0.275	99.160
2	10	10	0.1	0.178	0.010	89.582	9.954	0.276	99.812
2	100	20	0.1	0.420	0.023	89.352	9.928	0.276	99.556
2	100	50	0.1	0.065	0.004	89.689	9.965	0.277	99.931
2	100	100	0.1	0.016	0.001	89.735	9.971	0.277	99.983
2	1000	200	0.1	0.040	0.002	89.713	9.968	0.277	99.957
2	1000	500	0.1	0.006	0.000	89.745	9.972	0.277	99.993
2	1000	1000	0.1	0.002	0.000	89.749	9.972	0.277	99.998

σ^2_I is the epistatic genetic variance; k is the number of genes and k' is the number of genes that interact.

system (Table 5). If the value of θ is at least 1.0, in the populations with reduced frequencies of dominant genes the component of largest magnitude of the genotypic variance is the additive x additive variance, regardless of the polygenic system (Table 3). If the allelic frequencies are intermediary, the additive variance is the component of greatest value in the simpler system with up to 100 genes, with about 20% of them interacting. However, the additive x additive variance is the largest component in the other cases (Table 4).

In the populations with high frequencies of dominant genes, the dominance variance is the most important component of the genotypic variance, followed by the additive variance (Table 5). In the case of duplicate genes with cumulative effects, an increase in the proportion between the **a** deviation and the epistatic value of the homozygote for the genes that reduced the trait expression coupled with low frequencies of the dominant genes, a high number of genes determining the trait, and a high number of interacting genes will increase the magnitude of the epistatic variance, which can attain approximately 100% of the genotypic variance. Also in this case, when the allelic frequencies are intermediate, the additive x additive variance has the same magnitude as the sum of the additive x dominant and dominant x additive variances.

The case of dominant and recessive epistasis is even more complex. Considering three genes and digenic epistasis, only in relation to two of the three pairs it is possible to define this type of epistasis; for the third pair the epistasis is complementary, duplicate or duplicate genes with cumulative effects. The values presented in Table 6 were obtained assuming dominant and recessive epistasis of a single gene with all the (k' – 1) genes remaining, theta (**θ**) equal to – 1 and complementary epistasis in relation to the k'(k' – 1)/2 other pairs. In populations with reduced frequencies of dominant genes, the additive x additive variance is the component of largest magnitude of the genotypic variance, except in the simpler polygenic systems with no more than

Table 4. Relative magnitudes (%) of the components of genotypic variance, considering duplicate genes with cumulative effects and populations with intermediary allelic frequencies (p), in different polygenic systems

θ	k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
0.1	2	2	0.5	66.617	33.309	0.033	0.033	0.008	0.074
0.1	10	2	0.5	66.657	33.328	0.007	0.007	0.002	0.015
0.1	10	5	0.5	66.568	33.284	0.066	0.066	0.016	0.148
0.1	10	10	0.5	66.225	33.113	0.294	0.294	0.074	0.662
0.1	100	20	0.5	66.480	33.240	0.125	0.125	0.031	0.281
0.1	100	50	0.5	65.478	32.739	0.792	0.792	0.198	1.782
0.1	100	100	0.5	62.112	31.056	3.037	3.037	0.759	6.832
0.1	1000	200	0.5	64.758	32.379	1.273	1.273	0.318	2.864
0.1	1000	500	0.5	56.268	28.134	6.933	6.933	1.733	15.599
0.1	1000	1000	0.5	38.314	19.157	18.902	18.902	4.725	42.529
1	2	2	0.5	62.069	31.034	3.065	3.065	0.766	6.897
1	10	2	0.5	65.693	32.847	0.649	0.649	0.162	1.460
1	10	5	0.5	58.065	29.032	5.735	5.735	1.434	12.903
1	10	10	0.5	40.000	20.000	17.778	17.778	4.444	40.000
1	100	20	0.5	52.023	26.012	9.762	9.762	2.441	21.965
1	100	50	0.5	23.684	11.842	28.655	28.655	7.164	64.474
1	100	100	0.5	8.000	4.000	39.111	39.111	9.778	88.000
1	1000	200	0.5	16.886	8.443	33.187	33.187	8.297	74.672
1	1000	500	0.5	3.422	1.711	42.163	42.163	10.541	94.867
1	1000	1000	0.5	0.889	0.444	43.852	43.852	10.963	98.667
2	2	2	0.5	51.429	25.714	10.159	10.159	2.540	22.857
2	10	2	0.5	62.937	31.469	2.486	2.486	0.622	5.594
2	10	5	0.5	41.860	20.930	16.537	16.537	4.134	37.209
2	10	10	0.5	18.182	9.091	32.323	32.323	8.081	72.727
2	100	20	0.5	31.359	15.679	23.539	23.539	5.885	52.962
2	100	50	0.5	8.072	4.036	39.063	39.063	9.766	87.892
2	100	100	0.5	2.198	1.099	42.979	42.979	10.745	96.703
2	1000	200	0.5	5.211	2.606	40.970	40.970	10.243	92.183
2	1000	500	0.5	0.890	0.445	43.851	43.851	10.963	98.665
2	1000	1000	0.5	0.224	0.112	44.295	44.295	11.074	99.663

σ^2_I is the epistatic genetic variance; k is the number of genes and k' is the number of genes that interact.

10 genes, with about 20% of them interacting (Table 6). Even with complete dominance in all the loci, the genetic variances due to dominance, additive x dominant, dominant x additive and dominant x dominant epistatic effects represent together at most 10% of the genetic variance. In populations with intermediary allelic frequencies, the additive x additive variance is the component with the largest value in polygenic systems with at least 100 genes, with about 50% of them interacting (Table 6). In the other cases, it is the additive variance that represents the largest fraction of the genotypic variance, followed by dominance variance.

In populations with high frequencies of dominant genes, the component of largest magnitude is the variance due to dominance, which is surpassed by the dominant x dominant epistatic variance in the polygenic system with many genes, at least 1,000, with at least 50% of them interacting (Table 6). As in the case of complementary epistasis, the epistatic variance is maximized when the frequencies of the dominant genes are reduced. Regardless of the gene frequencies, an increase in polygenic system complexity and in the number of interacting genes will increase the magnitude of the epistatic variance that may attain almost 100% of the genotypic variance. Once again there is equality in the values of the additive x additive variance and the sum of the additive x dominant and dominant x additive variances when the allelic frequencies are equal.

Assuming $a_i \approx a_j$ (i, j = 1, ..., k; i ≠ j), the relative values of the components of genotypic variance with recessive epistasis came close to those presented for complementary epistasis. Under the same assumption, the relative values of the components of genotypic variance with dominant epistasis came close to those presented for duplicate epistasis and the relative values of the components of the genotypic variance with non-epistasis genetic interaction approach those presented for duplicate genes with cumulative effects.

If the gene frequencies in the population are not all approximately equal, the relative magnitudes of the components of the genotypic variance differ from the presented values (Table 7). In relation to two genes, with complementary epistasis (A_B_ with one genotypic value and A_bb, aaB_ and aabb with different value), there is a tendency for the additive variance to be the component of largest value, followed by the dominance variance. The magnitude of the epistatic components tends to be larger when the frequencies of the dominant genes are reduced. With duplicate epistasis (A_B_,

Table 5. Relative magnitudes (%) of the components of genotypic variance, considering duplicate genes with cumulative effects and populations with high frequencies of dominant genes (p), in different polygenic systems

θ	k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
0.1	2	2	0.9	18.182	81.817	0.000	0.000	0.001	0.001
0.1	10	2	0.9	18.182	81.818	0.000	0.000	0.000	0.000
0.1	10	5	0.9	18.181	81.816	0.000	0.001	0.002	0.003
0.1	10	10	0.9	18.180	81.809	0.000	0.003	0.008	0.012
0.1	100	20	0.9	18.181	81.814	0.000	0.001	0.003	0.005
0.1	100	50	0.9	18.176	81.792	0.001	0.009	0.021	0.032
0.1	100	100	0.9	18.159	81.714	0.004	0.038	0.085	0.127
0.1	1000	200	0.9	18.173	81.776	0.002	0.015	0.034	0.051
0.1	1000	500	0.9	18.124	81.556	0.011	0.095	0.214	0.320
0.1	1000	1000	0.9	17.951	80.779	0.042	0.378	0.851	1.271
1	2	2	0.9	18.158	81.713	0.004	0.038	0.086	0.129
1	10	2	0.9	18.177	81.797	0.001	0.008	0.017	0.026
1	10	5	0.9	18.135	81.608	0.008	0.076	0.172	0.257
1	10	10	0.9	17.973	80.880	0.038	0.341	0.767	1.146
1	100	20	0.9	18.093	81.420	0.016	0.145	0.326	0.487
1	100	50	0.9	17.626	79.315	0.101	0.910	2.048	3.060
1	100	100	0.9	16.125	72.564	0.374	3.365	7.572	11.311
1	1000	200	0.9	17.295	77.828	0.161	1.451	3.265	4.877
1	1000	500	0.9	13.759	61.917	0.804	7.237	16.283	24.324
1	1000	1000	0.9	7.950	35.776	1.860	16.743	37.671	56.274
2	2	2	0.9	18.089	81.399	0.017	0.153	0.343	0.513
2	10	2	0.9	18.163	81.734	0.003	0.031	0.069	0.103
2	10	5	0.9	17.996	80.984	0.034	0.303	0.683	1.020
2	10	10	0.9	17.376	78.192	0.147	1.319	2.967	4.432
2	100	20	0.9	17.833	80.247	0.063	0.571	1.286	1.921
2	100	50	0.9	16.144	72.647	0.371	3.335	7.504	11.210
2	100	100	0.9	12.040	54.179	1.117	10.051	22.614	33.782
2	1000	200	0.9	15.088	67.894	0.563	5.063	11.393	17.019
2	1000	500	0.9	7.955	35.796	1.859	16.735	37.655	56.249
2	1000	1000	0.9	2.957	13.308	2.768	24.913	56.054	83.734

σ^2_I is the epistatic genetic variance; k is the number of genes and k' is the number of genes that interact.

Table 6. Relative magnitudes (%) of the components of genotypic variance, considering dominant and recessive epistasis, in relation to different polygenic systems and populations

k	k'	p	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
2	2	0.1	72.440	4.024	21.123	2.347	0.065	23.536
10	2	0.1	89.243	4.958	5.205	0.578	0.016	5.799
10	5	0.1	24.904	1.384	66.158	7.351	0.204	73.713
10	10	0.1	5.605	0.311	84.440	9.382	0.261	94.083
100	20	0.1	11.269	0.626	79.075	8.786	0.244	88.105
100	50	0.1	1.842	0.102	88.006	9.778	0.272	98.056
100	100	0.1	0.455	0.025	89.320	9.924	0.276	99.520
1000	200	0.1	1.113	0.062	88.696	9.855	0.274	98.825
1000	500	0.1	0.178	0.010	89.582	9.954	0.276	99.812
1000	1000	0.1	0.045	0.002	89.708	9.968	0.277	99.953
2	2	0.5	48.485	24.242	12.121	12.121	3.030	27.273
10	2	0.5	62.016	31.008	3.101	3.101	0.775	6.977
10	5	0.5	44.444	22.222	14.815	14.815	3.704	33.333
10	10	0.5	23.188	11.594	28.986	28.986	7.246	65.217
100	20	0.5	38.929	19.465	18.491	18.491	4.623	41.606
100	50	0.5	12.608	6.304	36.039	36.039	9.010	81.087
100	100	0.5	3.722	1.861	41.963	41.963	10.491	94.417
1000	200	0.5	8.640	4.320	38.685	38.685	9.671	87.040
1000	500	0.5	1.558	0.779	43.406	43.406	10.851	97.663
1000	1000	0.5	0.397	0.199	44.180	44.180	11.045	99.404
2	2	0.9	17.829	80.230	0.064	0.578	1.300	1.942
10	2	0.9	18.110	81.495	0.013	0.117	0.264	0.394
10	5	0.9	17.792	80.065	0.071	0.638	1.435	2.143
10	10	0.9	16.961	76.323	0.222	1.998	4.496	6.717
100	20	0.9	17.742	79.838	0.080	0.720	1.620	2.420
100	50	0.9	15.974	71.884	0.401	3.612	8.128	12.141
100	100	0.9	11.890	53.504	1.144	10.296	23.166	34.607
1000	200	0.9	15.065	67.793	0.567	5.100	11.475	17.142
1000	500	0.9	7.993	35.967	1.853	16.673	37.515	56.040
1000	1000	0.9	2.993	13.468	2.762	24.855	55.923	83.539

σ^2_I is the epistatic genetic variance; k is the number of genes, k' is the number of genes that interact and p in the frequency of the dominant gene.

Table 7. Relative magnitudes (%) of the components of genotypic variance, considering two genes and different epistasis

Epistasis	p_A	p_B	σ^2_A/σ^2_G	σ^2_D/σ^2_G	σ^2_{AA}/σ^2_G	$(\sigma^2_{AD} + \sigma^2_{DA})/\sigma^2_G$	σ^2_{DD}/σ^2_G	σ^2_I/σ^2_G
Complementary	0.1	0.3	51.825	4.227	34.288	9.252	0.408	43.949
	0.1	0.6	78.767	5.811	8.348	6.725	0.348	15.421
	0.1	0.9	93.613	5.390	0.172	0.783	0.043	0.998
	0.5	0.1	70.810	5.575	14.915	8.286	0.414	23.615
	0.5	0.9	64.607	34.422	0.118	0.588	0.265	0.971
	0.9	0.7	43.166	55.926	0.076	0.432	0.400	0.908
	0.9	0.4	73.271	25.747	0.134	0.648	0.201	0.983
Duplicate	0.1	0.3	71.033	12.900	12.535	3.383	0.149	16.067
	0.1	0.6	47.978	33.686	9.926	7.996	0.414	18.336
	0.1	0.9	14.881	66.156	3.266	14.881	0.817	18.964
	0.5	0.1	56.426	25.705	11.285	6.270	0.313	17.868
	0.5	0.9	5.013	20.551	9.023	45.113	20.301	74.436
	0.9	0.7	2.042	7.787	7.567	42.879	39.726	90.171
	0.9	0.4	6.985	29.426	8.671	41.911	13.007	63.589
Dominant and recessive	0.1	0.3	71.334	12.803	12.376	3.340	0.147	15.863
	0.1	0.6	70.481	20.007	5.149	4.148	0.215	9.512
	0.1	0.9	90.698	8.343	0.165	0.753	0.041	0.959
	0.5	0.1	30.115	6.106	40.282	22.379	1.119	63.780
	0.5	0.9	65.841	33.162	0.121	0.604	0.272	0.997
	0.9	0.7	16.572	74.436	0.755	4.276	3.961	8.992
	0.9	0.4	11.866	52.264	4.891	23.641	7.337	35.870
Duplicate genes with cumulative effects ($\theta = 1$)	0.1	0.3	37.045	2.327	47.301	12.764	0.563	60.628
	0.1	0.6	75.201	4.451	11.015	8.873	0.459	20.347
	0.1	0.9	93.487	5.229	0.221	1.008	0.055	1.284
	0.5	0.1	64.508	3.901	19.952	11.084	0.554	31.591
	0.5	0.9	64.541	34.209	0.152	0.758	0.341	1.250
	0.9	0.7	43.107	55.723	0.098	0.556	0.515	1.170
	0.9	0.4	73.200	25.535	0.173	0.834	0.259	1.265

σ^2_I is the epistatic genetic variance; p is the frequency of the dominant gene.

A_bb and aaB_ with one genotypic value and aabb with another value), the additive x additive epistatic variance should be the most important component of the genotypic variance when the frequencies of the dominant genes are greater than 0.5. With smaller frequencies of the dominant genes, the epistatic variance is minimized and the component of largest value tends to be the additive variance. Also in the case of dominant and recessive epistasis (A_B_, A_bb and aabb with one genotypic value and aaB_ with another value), the additive variance is the most important component, but the dominance variance becomes larger when the frequencies of the dominant genes are high. With duplicate genes with cumulative effects (A_B_ with one genotypic value, A_bb and aaB_ with another genotypic value and aabb with a third value), assuming q = 1, the additive variance tends to be the component of largest magnitude. However, if the frequencies of the dominant genes are high, the additive x additive variance will be the largest component. The values for recessive epistasis (A_B_ with one genotypic value, A_bb with another genotypic value and aaB_ and aabb with a third value), dominant epistasis (A_B_ and A_bb with one genotypic value, aaB_ with another genotypic value and aabb with a third value) and non-epistasis genic interaction (A_B_, A_bb, aaB_ and aabb with different genotypic values) are practically the same as complementary epistasis, duplicate epistasis and duplicate genes with cumulative effects, respectively.

Importância relativa dos componentes epistáticos da variância genotípica em populações não endógamas

RESUMO - *Existem abordagens teóricas sobre interação gênica em sistemas poligênicos e metodologias que permitem confirmar sua ocorrência. Entretanto, alguns aspectos relevantes como as importâncias relativas dos componentes epistáticos da variância genotípica requerem investigação. Foram consideradas epistasia complementar, duplicada, recessiva, dominante, dominante e recessiva, genes duplicados com efeitos cumulativos e interação gênica não-epistática. Independente do tipo de epistasia e das freqüências gênicas, quanto mais complexo o sistema poligênico, quanto maior o número de genes que interagem e quanto maior a magnitude dos efeitos epistáticos, relativamente aos desvios a (diferença entre valor genotípico do homozigoto de maior expressão e média dos valores genotípicos dos homozigotos) e d (devido à dominância), maiores as magnitudes dos*

componentes epistáticos. Apenas em sistemas genéticos mais simples ou naqueles com predominância de epistasia complementar, recessiva, dominante e recessiva, genes duplicados com efeitos cumulativos ou interação gênica não epistática, e freqüências elevadas dos genes dominantes, podem ser reduzidos ou desconsiderados os componentes epistáticos.

Palavras-chave: interação gênica, epistasia digênica, parâmetros genéticos, populações não-endógamas.

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