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Simulation of Quantitative Characters from Qualitatively Acting Genes[1]

II. Orthogonal Subdivision of Hereditary Variance in two-locus Genetic Systems

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Summary. The phenotypes associated with the nine genotypes in a quantitative genetic system consisting of two loci, each having two alleles can be described in terms of nine parameters, giving a system of nine linear equations. Populations with desired magnitudes and known nature of intra- and interlocus interactions are obtained by the use of this linear combination model. The total sums of squares for genotypes in these populations are partitioned into orthogonal components denoting additive and dominance effects of the two loci and the four types of nonallelic interactions between them. In most cases, the relative magnitudes of dominance and epistatic variances are found to be considerably smaller than the actual proportions of these genetic effects. Duplicate interaction produces larger epistatic variance than complementary type of gene interaction. At the higher levels of epistasis, dominant epistasis yields much larger epistatic variance than recessive epistasis. No epistatic variance is produced in the absence of epistatic effects. But, appreciable contributions of additive and dominance gene actions to the total genotypic variability are obtained even in the complete absence of these effects, if additive × dominance and dominance × dominance epistatic effects, respectively, are present. It is concluded that in elucidating the nature of gene action in simplified genetic systems, the estimates of first degree parameters obtained from the linear combination model are more useful than the orthogonal components of genotypic sum of squares.

Through a series of elegant experiments Thoday and his colleagues (1961 and later) have demonstrated that at least some of the polygenes controlling metrical characters in organisms can be located with sufficient accuracy and their individual effects can be recognized. The results of several other investigations (see Stewart, 1969 for a review) strengthen the assertion that continuous variation in quantitatively inherited characters may often be controlled by only a few gene loci. These discoveries point out the far reaching implications of biometrical analysis with locatable polygenes in elucidating our knowledge of quantitative genetic systems.

Conventional biometrical analysis of a simplified two-locus genetic system was reported by Fasoulas and Allard (1962). They used a factorial genetic model given in Cockerham (1954) for partitioning the total genotypic sum of squares into orthogonal components specifying additive, dominance and epistatic gene actions. Since then several other studies on two- or three-locus genetic systems have been published where orthogonal scales were used to separate additive, dominance and epistatic variances (Lee, Cockerham and Smith, 1968; Russell and Eberhart, 1970; Russell, 1971; von der Pahlen and Goldenberg, 1971 and Wilson and Lee, 1971). Orthogonality has the appeal to the users of second degree statistics because of the desirable mathematical properties. However, the estimates of the first degree parameters reveal that in all the six studies reviewed above, the relative role of additive gene action tends to be inflated and that of dominance and epistasis deflated (Jana, 1971; and Jana, in press). An important point never seems to have been made is that restricting our choice to orthogonal scales is merely a mathematical convenience, which needs to be examined from biological considerations. This paper purports to examine the consequences of applying Cockerham's (1954) orthogonal scales to estimate components of genetic variation in two locus genetic systems with biologically interpretable interlocus interactions.

Models and Methods

The phenotypic values associated with the nine genotypes obtained by all combinations of a pair of alleles (*I-i* and *J-j*) at each of the *i*th and *j*th loci can be conveniently expressed in the form of a 9×9 coefficient matrix and a 9-space parameter vector as follows (Seyffert, 1966):

$$\begin{bmatrix} 1 & 1 & 1 & 0 & 0 & 1 & 0 & 0 & 0 \\ 1 & 1 & 0 & 0 & 1 & 0 & 1 & 0 & 0 \\ 1 & 1 & -1 & 0 & 0 & -1 & 0 & 0 & 0 \\ 1 & 0 & 1 & 1 & 0 & 0 & 0 & 1 & 0 \\ 1 & 0 & 0 & 1 & 1 & 0 & 0 & 0 & 1 \\ 1 & 0 & -1 & 1 & 0 & 0 & 0 & -1 & 0 \\ 1 & -1 & 1 & 0 & 0 & -1 & 0 & 0 & 0 \\ 1 & -1 & 0 & 0 & 1 & 0 & -1 & 0 & 0 \\ 1 & -1 & -1 & 0 & 0 & 1 & 0 & 0 & 0 \end{bmatrix} \times \begin{bmatrix} Y \\ a_i \\ a_j \\ d_i \\ d_j \\ aa_{ij} \\ ad_{ij} \\ da_{ij} \\ dd_{ij} \end{bmatrix} = \begin{bmatrix} 22 \\ 21 \\ 20 \\ 12 \\ 11 \\ 10 \\ 02 \\ 01 \\ 00 \end{bmatrix} \quad [A]$$

where 22, 21, . . . , 00 are phenotypic values corresponding to *IIJJ*, *IIJj*, . . . , *iijj* genotypes, respectively, and the parameters are described as,

a_i = additive effect of *I/i*
a_j = additive effect of *J/j*

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d_i = dominance effect of I/i
d_j = dominance effect of J/j
aa_{ij} = interaction between a_i and a_j
ad_{ij} = interaction between a_i and d_j
da_{ij} = interaction between d_i and a_j
dd_{ij} = interaction between d_i and d_j
Y = effect of residual genotype and environment

The relationships among the first eight parameters giving various classical gene interactions are (Jana, 1971):

Relationship among parameters	Classical epistasis	F_2 ratio	
$a_i = d_i$, $a_j = d_j$, $aa_{ij} = ad_{ij} = da_{ij} = dd_{ij}$		9:3:3:1	
$a_i = d_i = a_j = d_j = aa_{ij} = ad_{ij} = da_{ij} = dd_{ij}$	Complementary	9:7	
$a_i = d_i \neq d_j$, $a_j = d_j = aa_{ij} = ad_{ij} = da_{ij} = dd_{ij}$	Recessive epistasis	9:3:4	
$a_i = d_i = a_j = d_j = -aa_{ij} = -ad_{ij} = -da_{ij} = -dd_{ij}$	Duplicate	15:1	[B]
$a_i = d_i \neq d_j$, $a_j = d_j = -aa_{ij} = -ad_{ij} = -da_{ij} = -dd_{ij}$	Dominant epistasis	12:3:1	
$a_i = d_i = -a_j = -d_j = aa_{ij} = ad_{ij} = da_{ij} = dd_{ij}$	Inhibitory	13:3	

By the use of the coefficient matrix in [A] it is possible to simulate two-locus quantitative genetic systems with desired amounts of additive, dominance and epistatic effects. The relationships in [B] can be used to produce such genetic systems which are characterized by classically interpretable nonallelic interactions.

The parameter Y in [A] is defined as the effect due to environment and the genotype excluding the ith and jth loci. The residual genotype provides the common genetic background in which the phenotypic difference among the nine genotypes at the ith and jth loci are considered. Hence. the results of the present investigations are independent of the sign and magnitude of Y.

The eight parameters in [B] are attributed to the genetic effects of I/i and J/j. The size of a parameter measures the relevant effect as deviation from Y, either in positive or negative direction. The sum of the absolute values of the eight parameters is described as the total genetic effect. The absolute value of a parameter, expressed as percentage of the total genetic effect, is considered as a reliable estimate of the relative magnitude of the effect specified by the parameter.

The nine genotypes produced by all possible combinations of two pairs of genes, *I-i* and *J-j*, provide a situation analogous to a two-factor experiment with three equispaced quantitative levels. Hence, the main effects of I/i and J/j can be subdivided into linear and quadratic orthogonal comparisons, which are equivalent to additive and dominance effects, respectively, in genetic terminology. Cockerham (1954) proposed four orthogonal subdivisions of the interaction effect. Applied to the nine genotypes in the above two-locus model, Cockerham's orthogonal scales have the following form:

	22	21	20	12	11	10	02	01	00	
a_i =	1	1	1	0	0	0	−1	−1	−1	
a_j =	1	0	−1	1	0	−1	1	0	−1	
d_i =	1	1	1	−2	−2	−2	1	1	1	
d_j =	1	−2	1	1	−2	1	1	−2	1	
aa_{ij} =	1	0	−1	0	0	0	−1	0	1	[C]
ad_{ij} =	1	−2	1	0	0	0	−1	2	−1	
da_{ij} =	1	0	−1	−2	0	2	1	0	−1	
dd_{ij} =	1	−2	1	−2	4	−2	1	−2	1	

Since the above contrasts are mutually orthogonal, the sum of squares due to genotypes can be subdivided into eight orthogonal components. Let C_k be the kth orthogonal contrast in [C], then the sum of squares for that contrast is given by

$$SSc_k = \frac{C_k^2}{\sum_l \frac{c_{lk}^2}{n_l}} \quad \begin{matrix} k = 1, 2, \ldots, 8 \\ l = 1, 2, \ldots, 9 \end{matrix}$$

where c_{lk} is the coefficient of the lth genotype and n_l is the number of individuals giving the mean phenotypic value of the lth genotype. In the present studies on simulated populations n_l's are always 100. The relative magnitudes of additive, dominance and epistatic variances are determined as the percentage of total sum of squares for genotypes.

Results

a) Complete dominance producing the 9:3:3:1 ratio in the F_2

Table 1 contains the proportions of additive, dominance and epistatic effects in two-locus genetic systems with complete dominance and equal magnitudes of interaction effects as in the first group in [B]. The results of factorial genetic analysis of the populations simulated from these proportions of genetic effects are given in Table 2. When epistasis is absent, 75%

Table 1. *Genetic effects attributed to the ith and jth loci giving the 9:3:3:1 ratio in the F_2 generation. The total additive, dominance and epistatic effects are expressed as percentage of the total genetic effect*

Effect	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)
a_i	30.0	25.0	25.0	22.2	15.0	10.0	3.0
a_j	20.0	20.0	15.0	11.1	10.0	5.0	2.0
d_i	30.0	25.0	25.0	22.2	15.0	10.0	3.0
d_j	20.0	20.0	15.0	11.1	10.0	5.0	2.0
aa_{ij}	0.0	2.5	5.0	8.3	12.5	17.5	22.5
ad_{ij}	0.0	2.5	5.0	8.3	12.5	17.5	22.5
da_{ij}	0.0	2.5	5.0	8.3	12.5	17.5	22.5
dd_{ij}	0.0	2.5	5.0	8.3	12.5	17.5	22.5
Additive	50.0	45.0	40.0	33.3	25.0	15.0	5.0
Dominance	50.0	45.0	40.0	33.3	25.0	15.0	5.0
Epistasis	0.0	10.0	20.0	33.3	50.0	70.0	90.0
i/a	0.0	0.2	0.5	1.0	2.0	4.7	18.0

$i = |aa_{ij}| + |ad_{ij}| + da_{ij}| + |dd_{ij}|$
$a = |a_i| + |a_j|$

Table 2. *Sums of squares for orthogonal comparisons expressed in percent of the total genotypic sum of squares in seven relationships described in Table 1. V_D/V_A is 0.333 in all cases*

Comparison	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)
a_i	51.9	45.2	52.7	53.3	39.0	29.4	12.7
a_j	23.1	29.4	20.6	16.4	21.3	13.7	10.4
d_i	17.3	15.1	17.6	17.8	13.0	9.8	4.2
d_j	7.7	9.8	6.9	5.5	7.1	4.6	3.4
aa_{ij}	0.0	0.3	1.2	3.9	11.0	23.9	38.9
ad_{ij}	0.0	0.1	0.4	1.3	3.7	8.0	13.0
da_{ij}	0.0	0.1	0.4	1.3	3.7	8.0	13.0
dd_{ij}	0.0	0.0	0.2	0.4	1.2	2.7	4.3
Additive	75.0	74.6	73.3	69.7	60.3	43.1	23.1
Dominance	25.0	24.9	24.5	23.3	20.1	14.4	7.7
Epistasis	0.0	0.5	2.2	7.0	19.6	42.5	69.2
V_I/V_A	0.0	0.01	0.03	0.10	0.33	0.99	2.99

of the total gene controlled variability is attributed to additive effects and 25% to dominance deviations. The average degree of dominance expressed as the ratio of total dominance variance and total additive variance (V_D/V_A) is 1/3. As long as dominance is complete ($a_i = d_i$ and $a_j = d_j$), the dominance ratio remains unchanged irrespective of the magnitude and nature of epistasis. Although with an increase in the values of epistatic parameters an increasing role for epistatic variance is revealed, the contribution of epistasis to the total variability among genotypes is always less than the actual amount of epistasis present. Like V_D/V_A, the ratio between total interaction variance and additive genetic variance (V_I/V_A) is a measure of the average nonallelic interactions. Since V_D/V_A is constant (.333), the interaction ratio increases with the increase in the amount of epistasis. It may be pointed out here that all effects in Table 1 are considered as positive deviations from Y. A change in the direction of effect will alter the results in Table 2 as the phenotypic differences associated with the nine genotypes will be different. For example, if the interaction terms are negative, an increased role for epistatic variance will be detected.

b) Complementary, duplicate and inhibitory interactions

It is evident from the relationships in [B] that epistasis, dominance and additive effects constitute 50%, 25% and 25% of the total genetic effect, respectively, in genetic systems characterized by above three types of classical interactions. The orthogonal subdivisions of the genotypic sum of squares reveal no change in the degree of dominance $(V_D/V_A = 1/3)$, but the relative contributions of interaction components are different for the three types of epistasis (Table 3). Since definite relationships among the eight parameters exist for any of the three classical interactions, a change in the direction of an effect does not affect the results given in Table 3. In all the three cases the variance components indicate an inflated role for additive gene action. The overestimation is caused at the expense of both dominance and epistasis in complementary and inhibitory systems. With duplicate interaction 50% of the genotypic variance is attributed to epistasis which is same as the relative magnitude of the first degree epistatic parameters.

Table 3. *Genetic effects with classical interactions and sums of squares due to orthogonal comparison expressed in percent of total sum of squares for genotypes. V_D/V_A is 0.33 in all cases*

Effect	Absolute value of the effect	Variance for the effect		
		Complementary	Duplicate	Inhibitory
a_i	12.5	30.00	18.75	42.86
a_j	12.5	30.00	18.75	10.71
d_i	12.5	10.00	6.25	14.29
d_j	12.5	10.00	6.25	3.57
aa_{ij}	12.5	11.25	28.13	16.07
ad_{ij}	12.5	3.75	9.37	5.36
da_{ij}	12.5	3.75	9.37	5.36
dd_{ij}	12.5	1.25	3.12	1.79
Additive	25.0	60.00	37.50	53.57
Dominance	25.0	20.00	12.50	17.86
Epistasis	50.0	20.00	50.00	28.57
i/a or V_I/V_A	2.0	0.33	1.33	0.53

c) Recessive and dominant epistasis

When dominance of the ith and jth loci are not equal, the complementary interaction breaks down to recessive epistasis. The extent of nonallelic interaction would then depend on the size of d_j. It is then theoretically possible to have more than 50% epistasis (limit 66.6%). Table 4 contains proportions of the eight genetic parameters at various levels of dominance at the jth locus. Considering positive d_i and d_j, populations are simulated and the components of genetic variance in such populations are presented in Table 5. As before, the components of variance show diminished roles for dominance and epistasis. The average degree of interaction measured as V_I/V_A increases steadily with the increase in epistasis as the variance ratio V_D/V_A is always 1/3.

Table 4. *Genetic effects attributed to the ith and jth loci with recessive epistasis. The total additive, dominance and epistatic effects are expressed as percentage of the total genetic effect*

Effect	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)
a_i	42.50	35.00	25.00	14.00	5.00	1.85	0.50
a_j	2.50	5.00	8.33	12.00	15.00	16.05	16.50
d_i	42.50	35.00	25.00	14.00	5.00	1.85	0.50
d_j	2.50	5.00	8.33	12.00	15.00	16.05	16.50
aa_{ij}	2.50	5.00	8.33	12.00	15.00	16.05	16.50
ad_{ij}	2.50	5.00	8.33	12.00	15.00	16.05	16.50
da_{ij}	2.50	5.00	8.33	12.00	15.00	16.05	16.50
dd_{ij}	2.50	5.00	8.33	12.00	15.00	16.05	16.50
Additive	45.00	40. 0033.33		26.00	20.00	17.90	17.00
Dominance	45.00	40.00	33.33	26.00	20.00	17.90	17.00
Epistasis	10.00	20.00	33.33	48.00	60.00	64.20	66.00
i/a	0.22	0.50	1.00	1.85	3.00	3.59	3.88

$i = |aa_{ij}| + |ad_{ij}| + |da_{ij}| + |dd_{ij}|$
$a = |a_i| + |a_j|$

Table 5. *Sums of squares for orthogonal comparisons expressed in percent of total genotypic sum of squares in genetic systems with recessive epistasis given in Table 4. In all cases V_D/V_A ratio is 0.333*

Comparison	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)
a_i	74.34	71.46	60.48	34.32	10.71	5.26	3.54
a_j	0.44	2.36	9.68	27.12	42.86	46.49	47.64
d_i	24.78	23.82	20.16	11.44	3.57	1.75	1.18
d_j	0.15	0.78	3.26	9.04	14.29	15.50	15.88
aa_{ij}	0.16	0.88	3.63	10.17	16.07	17.44	17.86
ad_{ij}	0.06	0.29	1.21	3.39	5.36	5.81	5.96
da_{ij}	0.06	0.29	1.21	3.39	5.36	5.81	5.96
dd_{ij}	0.02	0.10	0.40	1.13	1.78	1.94	1.98
Additive	74.78	73.82	70.16	61.44	53.57	51.75	51.18
Dominance	24.93	24.06	23.39	20.48	17.86	17.25	17.06
Epistasis	0.29	1.56	6.45	18.08	28.57	31.00	31.76
V_I/V_A	0.004	0.021	0.092	0.294	0.533	0.599	0.621

Duplicate gene interaction reduces to dominant epistasis if $d_i \neq d_j$. The proportions given in Table 4 can be used to simulate genetic systems with dominant epistasis when the signs of aa_{ij}, ad_{ij}, da_{ij} and dd_{ij} are opposite to that of d_j. Letting d_i and d_j positive and interactions negative, populations with three phenotypes of dominant epistatic systems are produced. The orthogonal subdivisions of the genotypic sums of squares in these populations are presented in Table 6. It is evident from the results that at a low level of epistasis, there is no difference between recessive and dominant epistasis with regard to the relative contributions of various gene actions. This is expected, because at the lower levels of epistasis either system approximates complete dominance with no epistasis described in Table 1 column (I). The differences, however, become obvious at the higher levels of epistasis. At these levels dominant epistasis yields larger epistatic variance than recessive epistasis. When total epistatic effect is about 60%, the components of genetic variance method attributes a larger relative role to epistasis. With still larger epistasis, little or no difference between the relative magnitude of total epistatic effect and epistatic variance is detected. The corresponding variance component with recessive epistasis is one-half of the former. These differences are reflected in the interaction variance ratios (V_I/V_A) as V_D/V_A is constant (1/3). An interesting feature of the results given in Table 6, column (V) is that although there are 5% additive and 5% dominance effect of the ith locus, the sums of squares for these two effects are zero. Such results readily lead to the erroneous conclusion that additive and dominance gene actions are absent at this locus.

Table 6. *Sums of squares for orthogonal comparisons expressed in percent of total genotypic sum of squares in genetic systems with dominant epistasis derived from the relationship given in Table 4 by setting all interactions negative. In all cases V_D/V_A ratio is 0.333*

Comparison	(I)	(II)	(III)	(IV)	(V)	(VI)	(VII)
a_i	74.64	72.81	63.16	25.68	0.00	2.58	4.83
a_j	0.12	0.73	3.95	16.44	25.00	24.14	23.39
d_i	24.88	24.27	21.05	8.56	0.00	0.86	1.61
d_j	0.04	0.24	1.32	5.48	8.33	8.05	7.80
aa_{ij}	0.18	1.09	5.92	24.66	37.50	36.21	35.08
ad_{ij}	0.06	0.36	1.97	8.22	12.50	12.07	11.69
da_{ij}	0.06	0.36	1.97	8.22	12.50	12.07	11.69
dd_{ij}	0.02	0.12	0.66	2.74	4.17	4.02	3.90
Additive	74.76	73.54	67.10	42.12	25.00	26.72	28.22
Dominance	24.92	24,51	22.37	14.04	8.33	8,91	9.41
Epistasis	0.32	1.94	10.52	43.84	66.67	64.37	62.37
V_I/V_A	0.004	0.026	0.157	1.041	2.667	2.409	2.210

The above results (Table 6) are produced by considering dominance effects of both loci in the positive direction. The general trend remains unchanged when either d_i or d_j is negative. If d_i is negative and d_j positive, a slightly larger estimate of epistatic variance is obtained with recessive epistasis. Under similar conditions dominant epistatic system has a smaller epistatic variance than when both d_i and d_j are positive. However, the reduction is so small at higher degrees of epistasis that the relative magnitude of epistatic variance for dominant epistasis is still considerably larger than that for the former.

d) No dominance, partial dominance and overdominance

The classical interactions dealt with in the previous sections are defined under the condition of complete dominance. The departure from complete dominances opens numerous possible relationships among the genetic parameters even when $aa_{ij} = ad_{ij} = da_{ij} = dd_{ij}$. The factorial genetic analysis of some of the relationships given in Table 7 are considered meaningful in the present context. It can be seen that incomplete dominance generally leads to a larger reduction in epistatic variance than overdominance. Clear cases of overdominance may not be reflected in the variance ratio (V_D/V_A) because of the inflated role for additive effects. On the other hand, the absence of dominance of either or both loci does not yield a zero variance for the effects if dd_{ij} has a nonzero value. Similarly, if $ad_{ij} \neq 0$ and $da_{ij} \neq 0$, there is additive variance at the ith and jth loci, respectively, even in the complete absence of additive gene action. The discrepant results are due to the differences between the orthogonal comparisons in [B] specifying additive and dominance effects and the unique solutions for the first degree parameters derived from [A] (given in Jana, 1971). The additive effects, a_i and a_j have nonzero coefficients for the single heterozygotes in [B], which are absent in the unique solutions. The inclusion of the single heterozygotes in the additive comparisons leads to the contribution of ad_{ij} and da_{ij} to the variances for a_i and a_j, respectively. The additional nonzero coefficient (−2) for the double heterozygote in the dominance comparisons in [B] results in an equal contribution of dd_{ij} gene action to variances due to d_i and d_j.

No variation due to any of the epistatic gene actions is produced if the value for the effect in [A] is zero. The reason for the coincidence can be found in the similarity of the coefficients of the nine genotypes for the four epistatic effects in [B] and the unique solutions of the linear equations given in Jana (1971).

Discussion

A variety of biometrical methods are available for the genetic analysis of metrical traits. Most of these techniques are based on the implicit assumption that the continuous variations in quantitatively inherited characters are mediated by a large number of genes, each having such a small effect that they can only be studied *en masse*, in terms of average effects of genes or groups of genes, often referred to as the effective

Table 7. *Genetic effects attributed to the ith and jth loci with incomplete dominance and various degrees of nonallelic interactions (Column a) and the sums of squares for orthogonal comparisons expressed in percent of total genotypic sum of squares (Column b)*

Effect	(I)		(II)		(III)		(IV)		(V)		(VI)	
	a	*b*	*a*	*b*	*a*	*b*	*a*	*b*	*a*	*b*	*a*	*b*
a_i	0.00	0.22	50.00	72.07	0.00	5.91	12.50	34.28	30.00	58.86	20.00	41.69
a_j	0.00	0.22	30.00	27.07	0.00	5.91	7.50	18.73	20.00	27.55	10.00	12.09
d_i	50.00	70.66	0.00	0.02	12.50	16.28	0.00	1.38	20.00	9.18	30.00	29.69
d_j	30.00	26.54	0.00	0.02	7.50	8.89	0.00	1.38	10.00	2.66	20.00	13.90
aa_{ij}	5.00	1.32	5.00	0.45	20.00	35.45	20.00	24.88	5.00	0.98	5.00	1.48
ad_{ij}	5.00	0.44	5.00	0.15	20.00	5.91	20.00	8.29	5.00	0.33	5.00	0.49
da_{ij}	5.00	0.44	5.00	0.15	20.00	5.91	20.00	8.29	5.00	0.33	5.00	0.49
dd_{ij}	5.00	0.15	5.00	0.05	20.00	3.94	20.00	2.76	5.00	0.11	5.00	0.16
Additive	0.00	0.44	80.00	99.15	0.00	11.82	20.00	53.00	50.00	86.41	30.00	53.78
Dominance	80.00	97.21	0.00	0.05	20.00	25.17	0.00	2.76	30.00	11.85	50.00	43.59
Epistasis	20.00	2.35	20.00	0.80	80.00	63.01	80.00	44.23	20.00	1.74	20.00	2.63
d/a or V_D/V_A	—	220.33	0.00	0.00	—	2.13	0.00	0.05	0.60	0.14	1.67	0.81
i/a or V_I/V_A	—	5.33	0.25	0.01	—	5.33	4.00	0.83	0.40	0.02	0.67	0.05

$a = |a_i| + |a_j|$
$d = |d_i| + |d_j|$
$i = |aa_{ij}| + |ad_{ij}| + |da_{ij}| + |dd_{ij}|$

factors. If, however, some components of a polygenic system can be located and their individual effects identified, it is possible to analyze them one by one like the so-called oligogenes. Formal biometrical analysis of several quantitative characters controlled by two or three loci, or short chromosome segments marked by them, have been undertaken in recent years in the hope that such studies would be useful in clarifying the nature of gene controlled variability in polygenic systems (Fasoulas and Allard, 1962, Lee, Cockerham and Smith, 1968; Persson, 1969 and 1971, Russell and Eberhart, 1970; Russell, 1971; von der Pahlen and Goldenberg, 1971; and Wilson and Lee, 1971). The additive and dominance gene actions of individual loci and interlocus interactions were estimated by the use of the second degree statistics. When phenotypic values associated with all possible genotypes are known, a reliable estimate of the first degree parameters is given by the solutions of simultaneous linear equations (Seyffert, 1966). Such estimates detect much larger roles for intralocus and interlocus gene interactions than revealed by the components of genetic variance (Jana, 1971; and Jana, in press). With the increasing evidence in favour of the view that quantitatively inherited characters are often controlled by a much fewer genes than ordinarily believed, it seems worth while to explore the consequences of standard biometrical analysis of simplified genetic systems with biologically well defined gene interactions. Fisher (1918) introduced the method of partitioning hereditary variance into additive, dominance and epistatic components which was further extended by Cockerham (1954) to the subdivision of epistatic variance into various components of nonallelic interaction, such as, additive × additive, additive × dominance and dominance × dominance, etc. The characteristic technique by which these geneticists sought to separate gene actions involves orthogonal comparisons specifying various genetic effects. It can be seen from the results presented in this paper that the components of hereditary variance lead to an overestimation of the contribution of additive effect, and therefore, cannot be considered as a reliable guide in understanding the nature of gene action underlying phenotypic differences. For example, with complete dominance, variance due to dominance effect is only one-third of the additive variance leading to the usual conclusion of partial dominance. The relative magnitudes of epistatic variance are different for different types of epistasis even when the total epistatic effects are equal. When orthogonal scales are used, duplicate interaction and its deviation, dominance epistasis provide better evaluation of the role of epistasis than complementary gene interaction and recessive epistasis. Much of the epistasis remain undetected by the components of genetic variance when the extent of interaction among loci is relatively small. If, however, an epistatic effect is completely absent, no variation due to that effect is detected. Similar conclusions cannot be extended to additive and dominance gene actions when certain types of nonallelic interactions are present.

Although in rare cases (*eg.* Table 6, column *V*) the relative magnitude of epistasis is less than the proportion of epistatic variance, the reverse situation is most frequent. The general underestimation of the role of epistasis is in no way unique to the Cockerham's (1954) model. The modified analysis of variance of diallel table proposed by Lee *et al.* (1968) also leads to similar results (Jana, 1971, and Jana, in press). What appears to be the absence of epistasis from the regression analysis of array variances and covariances in two sets of diallel crosses reported by Persson (1969 and 1971), there are 16% to 20% epistasis, largely of ad_{ij} and dd_{ij} types (Jana, in press). The

reasons for the failure to detect some of the classical interactions by the graphical analysis of diallel crosses are described in detail by Mather (1967). Determining components of genetic variance from segregating populations by the method of least-squares maximizes additive effect and thus minimizes dominance and epistasis (Crow and Kimura, 1970). Therefore, when segregation at a few gene loci adequately explains the variation in a metrical character, and when all genotypes and associated phenotypes are identified, it seems desirable to use the simpler and more direct method of analysis proposed by Seyffert (1966) than the sophisticated biometrical techniques developed for the analysis of complex polygenic systems. The merit of the former analytical method becomes more evident when the aim of an investigation is to seek for a physiological and biochemical interpretation of dominance and epistasis.

Zusammenfassung

Die in einem quantitativ-genetischen System mit je 2 Allelen an 2 Loci möglichen 9 Phänotypen, die mit den entsprechenden Genotypen assoziiert sind, können durch einen Satz von 9 linearen Gleichungen beschrieben werden. Mit Hilfe dieses Modells der linearen Kombination wurden Populationen mit willkürlich gewählter Dimension und Art der Interaktion innerhalb der und zwischen den Loci konstruiert. Die Gesamtsummen der Abweichungsquadrate für die Genotypen derartiger Populationen werden in orthogonale Komponenten zerlegt, die den additiven und den Dominanz-Effekten bzw. den vier Arten der nichtallelen Interaktion der beiden Loci zugeschrieben werden können. In der Mehrzahl der Fälle sind die relativen Größenordnungen der Dominanz- und Epistasie-Varianzen wesentlich kleiner als die tatsächlichen Anteile dieser Effekte. Eine gegenseitige Vertretbarkeit nichtalleler Gene (duplicate gene action, 15:1-Spaltung) führt zu einer größeren Epistasievarianz als komplementäre Genwirkung (9:7-Spaltung). Bei stark ausgeprägter Epistasie führt die sog. dominante Epistasie (12:3:1-Spaltung) zu einer wesentlich größeren Epistasievarianz als die rezessive Epistasie (9:3:4-Spaltung). In Abwesenheit epistatischer Effekte wird keine Epistasievarianz beobachtet. Jedoch werden bemerkenswerte Beiträge additiver und dominanter Genwirkungen zur genotypischen Gesamtvariabilität auch bei völliger Abwesenheit derartiger Wirkungen beobachtet, wenn Interaktionen des Typs additiv × dominant bzw. dominant × dominant vorliegen. Hieraus wird geschlossen, daß die Aufklärung der Art der Genwirkung in einfachen genetischen Systemen gezeigt hat, daß die Schätzwerte der Parameter 1. Grades, die aus dem zitierten Modell mit linearer Kombination erhalten werden können, brauchbarer sind als die orthogonalen Kombinationen der genotypischen Summe der Abweichungsquadrate.

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