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GENETIC BALANCES AS DETERMINANTS OF BIOLOGICAL SIMILARITY AND VARIATION

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ABSTRACT

Postulates: Pairwise structures are the basic components of living systems. As the lowest level of living structures, they obligatory must offer more than one opportunity of survival (1/1, 1/2, 2/2), minimizing the possibility to disappear under Darwinian selection. Functionally monomorphic molecules, as minimal and basic structure (A1 or A2) may extremely change their frequency under natural selection, and disappear in non-convenient conditions, being a non-satisfactory basic component of living systems. Basic relationships, not basic structures, are adaptive units and targets of natural selection. Not genes, but their cumulative homo/heterozygous relations, control the variation of quantitative traits.

INTRODUCTION

Quantitative-genetic variation is based on cumulative homo/heterozygous relationships of corresponding genes. Consequently, not individual genes, but their relationships, are determining variation of quantitative (fitness) characters. When a class with a maximum of homozygotes, or of heterozygotes, survives, their allelogenic constituents are approximately the same. This enables a quick recovery of a reduced size population, in following generations (see, also, Marinkovic 1977, 2016). This approach should be necessarily added to the classical theory of additive gene's determination of quantitative traits (Nilson-Ehle 1909 Emerson&East 1913), that accepts a loss of genetic variation in the cases of survival of extreme classes of a population, under Darwinian selection. Cumulative genetic homo/heterozygous ratios determine especially fitness characters. Survived classes in extreme environmental conditions maintain approximately equal allelogenic variation, which is crucially important for their recovery after bottle neck conditions. Table 1 presents the classical (A) and a proposal of a combination-genetic homo/hetero- cumulative approaches (B,C,D) to the determination and control of quantitative characters in F2 progenies of hybrid parents.

The numerical ratios so far verified to prove the complexity of observed quantitative characters are approximately the same. Only on the basis of three-hybrid crosses (C), it can be seen that to four alternative 'channels' of homo / hetero balances could be reduced 64 potential genotypes, whose 'phenotypes' could be hardly all «adaptive». It means that the role of balancing versus structural relationships in adaptive reductions during reproductive and developmental processes should be studied and estimated in this new analytical way. The above emphasized approach (see, also, Marinkovic 1977, 2016) has a special importance when we try to explain a fascinating *similarity* among individuals within a species, as a result of a restriction of adaptive variation. Biologists, and especially population geneticists, more than a century are analyzing the sources and consequences of *biological variation*, not being involved in a crucial problem how to treat the facts of *enormous individual similarities* that can't be explained by second Mendelian rule of free combinations of chromosomal allelogenes. Figure 1 presents our analysis of involvements of eight gene-enzyme systems from phosphor-sugar metabolic cycle in rates of development of *Drosophila melanogaster*, showing that gene-enzyme relationships (full line-significantly positive, dashed line- sign. negative

correlation in enzyme activity) are determining the variation of this fitness character (Marinkovic 2004). In our study of ten gene-enzyme systems from phosphor-sugar metabolic cycle of 400 *D.melanogaster* individuals (e.g., Marinkovic 1999, 2002, 2009), it was evidenced that out of 78000 potential genotypes only 160 have been realized, i.e. 0.2%.

Table 1. Quantitative traits genetically controlled according to cumulative effects: A/ of additive genes, or B, C, D/ of their homo- versus heterozygous combinations. The progenies of F1 hybrids

A/	0	1	2	3	4	cumul. alleles
	1	: 4	: 6	: 4	: 1	
	aabb	Aabb aAbb aa Bb aa bB	aaBB AAbb AaBb AabB aAbb aAbB	AaBB aABB AABb AAbb	AABB	
B/	homo	ho / he	hetero	cumul. ratios		
	1	: 2	: 1			
	aabb aaBB AAbb AABB	Aabb; AaBB aAbb; aABB aa Bb; AaBB aa bB; AaBB	AaBb Aabb aAbb aAbB			
C/	1	: 3	: 3	: 1		
	aabbcc aabbCC aaBBcc aaBBCC AAbbcc AABBcc AAbbCC AABBCC	Aabbcc; AaBBcc AabbCC; AaBBCC aAbbcc; aABBcc aAbbCC; aABBCC aaBbcc; AaBBcc aaBbCC; AaBBCC aaBBCC; AaBBCC aabbCc; AabbCC aabbCC; AaBBCC aaBBCC; AaBBCC aaBBCC; AaBBCC	AaBBCC; AabbCc AaBBcC; AabbCc aABBCC; aAbbCc aABBcC; aAbbCc AaBBCC; aabbCc AaBBCC; aaBbCc AaBBCC; aaBbCc AaBBCC; aabbCc AaBBCC; AaBbcc AaBBCC; AaBbcc aABBCC; aAbbCc aABBCC; aAbbCc aABBCC; aAbbCc	AaBbCc AaBbcC AaBbCc AaBbCc aAbBcC aAbBcC aAbBcC aAbBcC		
D/	1	: 4	: 6	: 4	: 1	
	aabbccdd	aAbbccdd	AabbCcdd	aaBbCcDd	aAbBcCdD	

Table 2. Eight possible 2nd-chromosomal types for three allozyme markers in gametes of 400 *D.melanogaster* individuals, and their probable evolutionary origin

Probable evolutionary origin	2 nd Chromosomal gametic types			Estimated frequency in 400 ind.
	Gpd 2-20.5	Adh 2-50.1	Hk 2-73.5	
I.	1	F	1	328
II.	93 mut	F	1	139
III.	1	S mut	1	137
IV.	93 rec	S	1	73
V.	1	F	103 mut	57
VI.	93	F rec	103	33
VII.	1	S rec	103	19
VIII.	93 rec	S rec	103	14
				800 chr.

Table 3. Twelve (out of 24) most frequent 3rd-chromosomal types, present at five allozymic loci in 390 *D.melanogaster* individuals. Decrease in the frequency of observed chromosomal types suggests the sequence of the appearance of their mutational markers during evolutionary biosynthesis

Sod 3-24.6	Pgm-1 3-43.4	Est-C 3-47.7	Odh 3-49.2	In(3R)	
				Acph-1 3-101.1	Nchr.
100	100	100	100	100	447
		103mut			163
	96mut				64
			98mut		28
90mut					16
	96rec	103			12
90rec		103rec	98		10
				97mut	9
				94mut	9
		103rec		97	5
	96	103rec		97	8

Out of 800 'second chromosomes' with observed aGpd, HK and Adh loci, more than forty percent had the original, i.e. initial allele at all these loci, another 40% had a mutation at one of these loci, suggesting that more than 80% of individual carriers have such almost identical constitution of evolutionary basic alleles (Table 2, Marinkovic 2002). The minor rest of here studied sample contains two or three additional replacements of available alleles, being called « genetic load » of this population. The similar picture is obtained when 800 «third chromosomes» were observed with five loci studied (Table 3, Marinkovic 2005), with original alleles present in a great majority of chromosomal carriers, and « genetic loads » as normal constituents of a population.

Conclusion

These real population samples show that rare mutations are part of normal constitution of a population, used for its survival in specific conditions. Due to the fact that a majority of such mutations are of recessive origin, their incorporation in the balance of homo- and heterozygotes (as well as of the other epistatic relationships) could be the main mechanism that may maintain the constitution of a population, and which basically determines and controls the similarity and variation of quantitative traits. It narrows and directs the enormous potential of combined .variations of apparently restricted no. of GL mutations to highly adaptive proportion of homo / hetero and epistatic relationships (Marinkovic 1977, 2016). Some initial comments on our earlier expressed approach on the influence of the genetic and developmental balances in life processes have been expressed in the papers of Petanovic & Marinkovic 1978, Zivotovski 1984, Cluster *et al.* 1988, Kovac & Marinkovic 1999, Zivanovic *et al.* 2000, etc. As homo / hetero balance also determines the constitution of chromosomes selected in meiotic divisions to be involved in

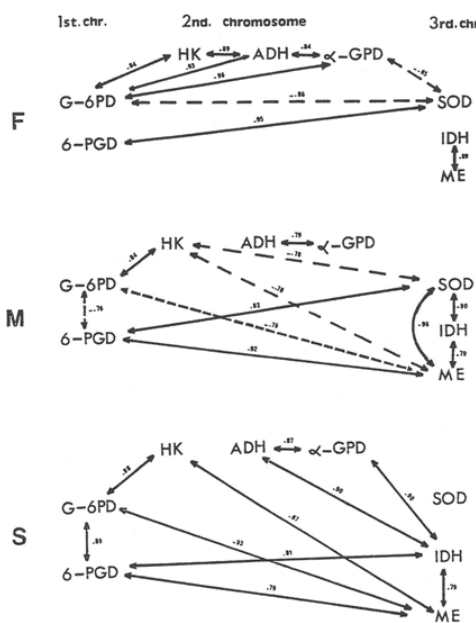


Figure 1. Positive (full lines) and significant negative correlations (dashed lines) among enzyme activities of fosphor-sugar metabolic cycle of *D.melanogaster* with Fast, Medium and Slow pre-adult developmental rate

reproductive cells (see, also, Marinkovic 2016), GAMETES, NOT ZYGOTES, should be considered as INITIAL FOUNDERS OF NEW GENERATIONS in Eucaryotes !

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