vials without food, r = 0.58, iv) *gaucha* females, controls vs reduced space plus food, r = 0.89; controls vs vials without food, r = 0.69. Correlation (r) coefficients under 0.7 indicate very poor fit between the pairs of matrices which are compared; r between 0.7 and 0.8 indicates a poor fit; r between 0.8 and 0.9 indicates a good fit; and r equal or over 0.9 indicates a very good fit between the matrices (Rolhf, 1995). Thus, the results of the Mantel's test above obtained a good agreement with the ethograms (Figure 1).

In the absence of food, adult flies of *D. pavani* and *D. gaucha* change their behaviors, as shown by new transitions that occur between the behavioral elements recorded, while other behaviors exhibited in the presence of food disappear. We also found that in the same environment, adults of *D. pavani* may react differently than *D. gaucha* adult flies. This is in agreement with Clark (1998) in the sense that different genetic systems may respond in different ways to the same environmental changes. However, only changes in grooming behavior reflect stress (Spruijt *et al.*, 1992; Eguibar *et al.*, 1997; Lawer and Cohen, 1988). Grooming represents a nervous activity behavior that is variably expressed when "located" among a well known environment or a stressing one. Changes in transitions locomotion ↔ still could merely represent adjustment of each sex of the sibling species to a new environment which is not necessarily stressing (Beerda *et al.*, 1999; Clark *et al.*, 1997). Our findings indicate that only males of *D. gaucha* exhibited recurrence and transitions for grooming behavior. Thus, this sex of *D. gaucha* could be representative indicators of stress when they are transferred to an environment where food and water are not available.

References: Beerda, B., M.B. Schilder, J.A. van Hooff, H.W. de Vries, and J.A. Mol 1999, Physiol. Behav. 66: a) 233-242, b) 243-254; Chrousos, G.P., and P.W. Gold 1992, JAMA 267:1244-1252; Clark, A.G., and C.D. Fucito 1998, Heredity 81:514-527; Clark, J.D., D.R. Rager, and J.P. Calpin 1997, Lab Anim. Sci. 47:571-579; Djawdan, M., A.K. Chippindale, M.R. Rose, and T.J. Bradley 1998, Physiol. Zool. 71:584-594; Eguibar, J.R., and A. Moyaho 1997, Pharmocol. Biochem. Behav. 58:317-322; Hoffmann, A.A., and P.A. Parsons 1994, In: *Evolutionary Genetics and Environmental Stress*, Oxford University Press Inc. New York, U.S.; Lawer, C.P., and P.S. Cohen 1988, Ann. NY Acad. Sci. 525:417-419; Rolfh FJ NTSYS-pc Numerical Taxonomy and Multivariate Analysis System. Version 1.80. State University of New York, Stony Brook, NY. Exeter Software; Sokal, R.R., and F.J. Rolhf 1995, In: *Biometry*, second edition, Freeman & Company, New York; Spruijt, B.M., J.A. van Hooff, W.H. Gispen 1992, Physiol. Rew. 72:825-852; Zhivotovsky, L.A., 1997, EXS 83:241-254.

A case of direct measurement of coefficients of selection in nature.

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The mathematical theory of selection developed by Fisher, Haldane and Wright assumes that coefficients of selection are constant. The usual result thereof, in the absence of heterozygotes' advantage, is elimination and fixation of alleles. The latter is considered as an elementary act, one of those which result in speciation that is considered as transmutation (Timofeev-Resovsky *et al.*, 1973). In contrast to this, observations of many researchers have resulted in establishing that 1) there is no allele fixation in populations, and polymorphism is conserved in each locus; 2) the polymorphism is

supported by selection which always favors a rare, disappearing allele (Luchnikova, 1978), and therefore multiple alleles that create polymorphism are in fact equivalent and may be called tolerable (normal), unlike the destructive (mutant) ones; 3) mutations have a destructive character since they destroy the high organization characteristic of tolerable alleles and are discarded by selection (Ivanov, 1991, 1998, 1999; Ivanov and Ivannikov, 1997); 4) results of these phenomena are genetic homeostasis, species invariance and implausibility of selectogenesis (Jenkin, 1867; Agassiz, 1874; Danilyevsky, 1885).

Direct measurements of the magnitude of selection in nature confirm these ideas. Coefficients of selection against the anomaly *abnormal abdomen* in *Drosophila melanogaster* have been measured. Larvae and pupae collected from nature have been studied under more favorable laboratory conditions with the goal of estimating among them the frequency q_0 of phenotype *abn. abd.* The frequency q_1 of this abnormality among imagines trapped in nature has also been estimated. From their values it is easy to obtain a formula for calculation of coefficient of selection at the stage of late larva and pupa

$$s = \frac{q_0 - q_1}{q_0(1 - q_1)}$$

and estimate its value, which has been done on three populations whose data are presented in Table 1.

When calculating the value of selection, it was assumed that the abnormality's penetrance, which is usually incomplete, was 100% in all cases. Due to this, we obtained coefficients of selection minimal with respect to the module. Indeed, if selection works against genotypes predisposed to the abnormality irrespective of the visual manifestation of the latter, then the true coefficient of selection against it is

$$\hat{s} = \frac{Q_0 - Q_1}{Q_0(1 - Q_1)},$$

where Q_0 and Q_1 are the frequences of abnormal genotypes at the preimaginal and imaginal stages, respectively. If α is the penetrance of the abnormality, then $Q_0 = q_0 / \alpha$ and $Q_1 = q_1 / \alpha$. Then

$$\hat{s} = \frac{q_0 - q_1}{q_0(1 - q_1 / \alpha)},$$

and since $0 < \alpha \leq 1$, then $1 - q_1 / \alpha \leq 1 - q_1$; therefore $|\hat{s}| \geq |s|$, *i.e.* true coefficients of selection with respect to the module are no less than the calculated ones. So, *e.g.*, at a penetrance $\alpha = 0.5$, the tabulated coefficients of selection would be substituted by -0.50, 0.79 и 0.80, respectively.

It is noteworthy that 1) coefficients of selection have a too large module; 2) the direction of selection changes sharply and strongly; 3) when selection favored the abnormality, its frequency among imagines was high (Dushanbe), and *vice versa*, selection against the abnormality decreased its frequency (other populations). Thereby, an extremely rare case of an extraordinary increase in the frequency of a very harmful abnormality in populations in a vast territory was studied (Berg, 1972a,b, 1973, 1974), and the role of selection in this phenomenon was demonstrated. The detrimental character of *abn. abd.* consists in a partial or complete sterility of flies when the abnormality affects the last segments of the abdomen and genitals (copulatory apparatus). It is supposed that the factor of selection is a pathogenic microbe to which the flies that carry the mutations *abn. abd.* are immune.

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References. Agassiz, L., 1874, Atlantic Monthly 33:92-111; Berg, R.L., 1972a, Dros. Inf. Serv. 48:67; Berg, R.L., 1972b, Dros. Inf. Serv. 48:67-69; Berg, R.L., 1973, Dros. Inf. Serv. 50:92; Berg, R.L., 1974, Dros. Inf. Serv. 51:37-38; Danilyevsky, N.Ya., 1885(1889), *Darwinism*, St.

Table 1. Occurrence of *abnormal abdomen* at later stages of development and among adult flies and values of selection coefficient against it in populations of *D. melanogaster*.

Population, month and year	Frequency of the anomaly in the sample, %				Coefficient of selection against the anomaly and its standard error
	Larvae and pupae		Imagines		
	q_0	n	q_1	n	
Dushanbe (Middle Asia) May 1975	5.85	427	8.27	701	- 0.45 ± 0.36
Tbilisi (Caucasus) May 1976	11.5	52	3.02	265	0.76 ± 0.14
Siniy Gay (Far East) October 1979	4.49	89	0.966	414	0.79 ± 0.15

In Dushanbe population the coefficient of selection against the normal phenotype $s=(q_1-q_0)/q_1(1-q_0)$ is 0.31 ± 0.17.

Petersburg: M.Ye. Komarov's Publisher, vol. 1, part 1:xii+519, part 2:xvi+530+148, vol. 2:200 (Russ.); Ivanov, Yu.N., 1991, Decomposition of the spontaneous mutation process into the main mutation types in the genome of fruit fly *D. melanogaster*, Abstr. IV All-Union Conf. *"Ecological Genetics of Plants, Animals and Man"*, Kishinyov: Shtiintsa, 461-462 (Russ.); Ivanov, Yu.N., 1998, Dros. Inf. Serv. 81:193-197; Ivanov, Yu.N., 1999, Dros. Inf. Serv. 82:87-90; Ivanov, Yu.N., and A.V. Ivannikov 1997, Dros. Inf. Serv. 80:57-59; Jenkin, F., 1867, The North British Review 46:277-318; Luchnikova, Ye.M., 1978, In: *Drosophila in Experimental Genetics*, Novosibirsk: Nauka, 171-196 (Russ.); Timofeev-Resovsky, N.V., A.V. Yablokov, and N.V. Glotov 1973, *Essay of Population Doctrine*, Moscow: Nauka (Russ.).

Structure, function, and expression of a retinoid binding protein in *rugose* (*rg*) mutants.

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Rugose (*rg*) locus mutant *Drosophila* have rough compound eyes and abnormal cone (Semper) cell numbers due to cone cell specification defects (Renfranz and Benzer, 1989). Figure 1 is a scanning electron micrograph (SEM) showing that *rg*, the *x3* allele, has rough compound eyes but fairly normal ocelli. Despite this slight disarray, the eye seems to be fairly normal. For instance, higher magnifications show that the compound eyes have the usual array of corneal nipples, thought to be an antireflection adaptation (*e.g.*, Stark *et al.*, 1989). Another way to demonstrate that the compound eye is not too badly disorganized is the deep pseudopupil image (Figure 2, rg^{p3}). The deep pseudopupil is virtual image of the magnified rhabdomere tips and has been utilized for decades as a diagnosis of rhabdomere integrity (*e.g.*, Harris *et al.*, 1976). Although, the individual R1-6 and R7 receptors cannot be distinguished in this image (*cf.*, Harris *et al.*, 1976), the image is pretty good.

We were interested in the *rg* mutant with its cone cell phenotype because we had been investigating a protein, RFABG = retinoid and fatty acid binding glycoprotein, expressed in cone cells (Shim *et al.*, 1997). Figure 3 shows a selected confocal micrograph (fluorescein optics) of *w*