

Research Notes

Alikhanian, S.I. A case of a different phenotypical manifestation of a lethal mutation.

In the course of study of the mutation process in the left end of the X-chromosome of *D. melanogaster*, a lethal given the number 76 was obtained. As long as this stock was carried on through attached-X's line ( $176 y v f B/Dp215 x$

$yy$ ) nothing exceptional was noted. (For a detailed description of this investigation see my works in *Zoolog. Zhurnal*, vol. 16, No. 2, 1937 and *Doklady Ak. N. USSR*, No. 9, 1938).--However, as soon as this line was made homozygous, it was detected that besides the expected  $176 y v f B/Dp215$  males and  $176 y v f B/176 y v f B/Dp215$  females, there appeared as well yellow daughters, i.e. females homozygous for the chromosome  $176 y v f B$  but having no *Dp215*. The duplication 215 "covers" the region of the left end of the X-chromosome where  $176$  is located. The stock  $176$  was further carried on in se and always produced beside gray males and females a certain number of yellow females, i.e. females homozygous for  $176$ . --A careful study of the phenomenon revealed that females homozygous for the lethal can survive without the duplication. In attempting to obtain sons from these females, such were in fact obtained but only as exceptions.

Table 1

$176 y v f B/176 y v f B x$  Florida  $\sigma^{\sigma}$

$176 y v f B/Florida$	$yy y v f B$ non-dis- junctional	$\sigma^{\sigma}$ wild type non-dis- junctional	$\sigma^{\sigma} y v f B (176)$
831	34	38	12

--The non-disjunctional females  $176 y v f B/176 y v f B$  were mated to Florida males. The offspring obtained was analogous to that given in table 1.

Table 2

$176 y v f B/Florida$	$176 y v f B$ non-dis- junctional	$\sigma^{\sigma}$ wild type non-dis- junctional	$\sigma^{\sigma} y v f B (176)$
145	23	29	---

--Different results are obtained from the mating of  $176 y v f B/176 y v f B Dp215 x 176 y v f B Dp215$ .

Table 3

$176 y v f B$ $176 y v f B$ Dp215	$176 y v f B$ $176 y v f B$	$176 y v f B$ Dp215	$176 y v f B$
382	58	138	---

--As seen from this table, females homozygous for  $176$  carrying no dupl. 215 emerge in a lower number than females carrying the dupl. 215. It seems probably that this difference is not due to the character of the lethal but is depending on the frequency of disjunction of the duplication present in the gametes of both parents. A more striking feature of the data presented

is the lack of  $1^{76}yvfb$  males without the dupl. 215. While it was possible to obtain sons with  $1^{76}$  chromosome but without Dp215 from mothers homozygous for  $1^{76}$  and carrying no duplication, such males were never produced by mothers homozygous for  $1^{76}$  and carrying Dp215. Another phenomenon has still more drawn our attention.  $1^{76}yvfb$  males obtained in the table 1 when mated to normal flies (y as well as ordinary) behave as quite normal males. Of no less interest is the physiological character of the lethal mutation 76. The larvae of this line possess tumours which cause the death of such larvae on different stages of development. A more detailed communication concerning this phenomenon will be given soon.

Belgovsky, M.L. Deletion frequency in the BSL chromosome.

$B^S$  males were X-rayed, the dosage being 3500 r, and mated to  $y v f car$  females. Individuals carrying a deleted BSL chromosome were recorded among the  $F_1$  females. In a total of 36,638 females the following aberrant types were found:  $v B\phi-4$ ,  $v car\phi-1$ ,  $vf B\phi-1$ ,  $v f car\phi-3$ ,  $v f\phi-1$ ,  $y v f\phi-1$ . Evidently all of them, except of the last one, carried a deleted BSL chromosome, the first two types having received this chromosome with the right break to the left of f, while the third, fourth and fifth types received a chromosome broken to the right of f. Thus the frequency of deletions in the BSL chromosome was approximately  $1/3760$  at the dose of 3500 r. Deleted chromosomes with a break occurring to the left and to the right of f are recorded with equal frequency. The 5 females carrying a deleted chromosome with a break to the left of f were bred in order to secure the deleted chromosome, but they proved of very low viability and highly sterile. Only one of them had produced progeny consisting of 2  $y v f car\phi$ , while the other 4 died. It seems to indicate that the very low deletion frequency recorded is at least partly due to the unviability of the flies hyperploid both for the y and f regions.

Belgovsky, M.L. Symbols & characteristics of mutants and valuations.

T(1;3) Del 143-3. Found by Neuhaus and genetically identified as a deletion. Studied in metaphase plates by Gershenson, who found no free deleted chromosome, but discovered that one of the autosomes had been broken in two parts. A genetical analysis by Belgovsky showed that the distal end of the X, broken between sc and br, became attached to the proximal end of 3L (broken between st and the spindle fibre attachment), while the distal end of 3L was evidently attached to the XR.

Neuhaus, M. The influence of separate arms of the Y-chromosome on secondary non-disjunction in the females of *D. melanogaster*.

When single crossing-over between any arm of the Y-chromosome and the X-chromosome takes place in the females of the constitution  $XY^{SX}$  or  $XY^{LX}$ , separate or attached, short or long arms of the Y are produced. The obtaining of parts of the Y-chromosome makes it possible to study the influence of separate arms of the Y on secondary non-disjunction. Following results were obtained:

	Regular	Exeptional	%	m
$\phi\phi \frac{y-bb}{y-bb}$ Y x	2183	101	4.42	0.43
$\phi\phi \frac{y-bb}{y-bb}$ Y Long arm x $\phi\phi$	997	28	2.73	0.51
$\phi\phi \frac{y-bb}{y-bb}$ Y short arm x $\phi\phi$	2749	40	1.43	0.23
$\phi\phi \frac{y-bb}{y-bb}$ $Y^{SYS}$ x $\phi\phi$	2674	136	4.8	0.4

Shapiro, N.I. X-rays dosage and the frequency of somatic mosaics.

In 1936, an experiment was carried out according to our suggestion by our student Tcherkassova, V.I. in order to study the dependency of the frequency of arising mosaics upon X-ray's dosage. 3

to 52 hours old larvae were subjected to treatment. They were obtained from the crossing of  $+/+$  females to  $y\ w\ f$  males. The frequency of occurrence of mosaic areas was studied in  $F_1$  males and females. The results of examination of the  $F_1$  females are given in the table.--As seen from the data there is no linear proportionality between the frequency of mosaics and X-ray dosage. If the mosaic frequency is studied for each gene separately, it is possible to notice that a deviation from the proportionality rule is manifested by mosaics for the genes yellow-forked, in case they are both present on the same mosaic area, and for the gene white. No deviation from direct proportionality was obtained for the genes yellow and forked separately. Unfortunately, the work could not be completed. In particular it was not found out what kind of changes (point mutations, deletions, simple breaks, somatic crossing over) are responsible for the deviation from direct proportionality. However, already the given above material, enables us to assert with great probability that chromosomal aberrations play a dominant role in the formation of mosaicism. Somatic gene mutations arise under the influence of X-rays comparatively seldom. Approximately among the same number of males as the number of females studied, there were obtained 12 somatic mutations of yellow, white and forked under the same X-ray's dosage. --Somatic crossing over which can as well produce mosaicism can hardly be considered as its chief cause. This is proved by the fact, that forked mosaics (including  $y\ f$ ) are more numerous than  $y$ -mosaics. Forked mosaics arise chiefly as the result of breakages in the inert region of the chromosome. The fact that most forked-mosaics are not accompanied by the manifestation of the yellow gene points out to the important role played by deletions in the formation of mosaicism.

Table

Frequency of Somatic Mosaics in Females Raised from X-rayed  $y\ w\ f/+$  Larvae

Dosage	300 r.	600 r.	900 r.	1200 r.	1500 r.	1800 r.
Number of females studied	7520	6695	2284	4120	1404	1081
Total number of mosaics	104	104	67	203	1404	1081
	0.58 <sup>±</sup> 0.089	1.55 <sup>±</sup> 0.15	2.94 <sup>±</sup> 0.35	4.93 <sup>±</sup> 0.34	7.20 <sup>±</sup> 0.69	9.62 <sup>±</sup> 0.89
Mosaics for:						
white	16	45	30	106	56	51
	0.21 <sup>±</sup> 0.052	0.67 <sup>±</sup> 0.10	1.31 <sup>±</sup> 0.24	2.57 <sup>±</sup> 0.25	3.98 <sup>±</sup> 0.52	4.72 <sup>±</sup> 0.64
yellow	15	20	12	29	13	22
	0.20 <sup>±</sup> 0.051	0.30 <sup>±</sup> 0.067	0.53 <sup>±</sup> 0.15	0.70 <sup>±</sup> 0.13	0.93 <sup>±</sup> 0.25	2.03 <sup>±</sup> 0.42
forked	12	33	16	44	16	18
	0.16 <sup>±</sup> 0.046	0.49 <sup>±</sup> 0.085	0.70 <sup>±</sup> 0.17	1.07 <sup>±</sup> 0.16	1.14 <sup>±</sup> 0.27	1.67 <sup>±</sup> 0.39
yellow-forked	1	6	9	24	16	13
	0.01 <sup>±</sup> 0.011	0.09 <sup>±</sup> 0.036	0.40 <sup>±</sup> 0.13	0.60 <sup>±</sup> 0.11	1.14 <sup>±</sup> 0.27	1.20 <sup>±</sup> 0.33

#### Technical Notes

Sarkissian, S.M. A new method of selection of virgin females in *Drosophila*.

While studying the genetics of the domestic fly (*Musca domestica*) we encountered the necessity of elaborating some proper method of selection of virgin females in *Musca domestica*. This method has been found in the course of a series of experiments. It is based on the discovery of the fact, that if newly emerged flies are deprived of food, females can live during the whole course of their

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are deprived of food, females can live during the whole course of their

starved life together with males and remain virgin.--After this method was proved to hold true for Musca domestica, it was surmized that this phenomenon can be used for other taxonomically related to Musca domestica groups of insects. Therefore experiments were undertaken with D. melanogaster. --Approximately 220 females have been studied. They were all kept without any food together with males from the time of their emergence through 52, 48, 42, 28 and 17 hours. Not a single female gave offspring in the experiment, while the control females gave normal progeny.

Sarkissian, S.M. Description of the method of preparing cultures for selection of virgin females.

In order to obtain virgin females it is necessary to put a scroll of paper (a) in the vial (b) containing larvae, in such a manner that its lower border should touch the food with larvae and its sides cling close to the inner surface of the vial. It is better to choose the period when the first adult larvae begin to pupate.--As soon as the desired number of larvae have pupated on the surface of the scroll of paper, it must be transferred to an empty vial (c), containing no food, where it will remain for the rest of the time. In order that no food whatever should be transferred in the vial "c", it is necessary to cut off the lower part of the scroll.--The duration of life of starving flies varies greatly and is dependent chiefly on temperature and humidity conditions. Our experiments were carried out at 22°C.--Experiments are being continued.

The victors in this competition are those individuals that come from groups in which the dominance of normal alleles and consequently contamination of the genotype with harmful mutations is the least, because in such groups harmful mutations could more readily be eliminated and useful mutations more readily preserved by natural selection.--A decrease in dominance brings about an increase in mutability, the latter in its turn leads to a new decrease in dominance and so on. It is this conflict of the processes of increase and decrease in dominance that determines the degree of dominance of genes in the population.--The disturbance of the species system that is possible under conditions of complete isolation, during the process of domestication or transfer to laboratory conditions, and sometimes even in nature, is accompanied by an increase in dominance and by a decrease in mutability.--There were two populations studied: one in Uman (Kiev Region, Ukraine) during the summer and fall of 1937 and the other in the Nikita Botanical Gardens (near Yalta, Crimea) during the fall of 1937 and the summer and fall of 1938.--A study of the frequency of phenotypical aberrations (i.e. deviations from the normal phenotype) in flies from six different centers of propagation (wineries, fruit depots, etc.) has shown that a population, even when it is rapidly growing, still consists of distinct micropopulations.--The frequency of aberrations in the phenotypes of flies belonging to different micro-populations is different and can be correlated with the intensity of selection, there being more aberrations wherever selection is less intense. There are less aberrants among migrant than among non-migrant flies. As the population becomes more numerous, the percentage of aberrants increases. They are more numerous among flies recently hatched from pupae (collected either as still unpigmented, i.e. just hatched flies or as pupae which complete their development later in vials) than among flies that had already been subjected to the action of selection during the imaginal stage. Aberrants are more frequent among females than among males. As the population grows this difference is smoothed over.--These conclusions are based on statistically significant data. 35,551 flies were collected in nature (either as imagoes by means of traps or as pupae) and 3,125 aberrants were found among them.--358 aberrants were studied with the purpose of determining the character of their inheritance. 248 (68.2%) proved to be hereditary.--Of the hereditary aberrations 173 (69.8%) proved to be dominant, 75 (30.2%) recessive. All the sex-linked mutations (26 cases studied) proved to be recessive.--Among phenotypically similar aberrations there were observed dominant aberrations as well as recessive ones. In heterozygotes as well as in homozygotes penetrance and expressiveness of mutations are very variable. They are higher among males than among females. The more pronounced manifestation of harmful characters in females seems to be correlated with the existence of an extra factor of selection among males (competition among males for females). It was proved experimentally (in cooperation with Alexandryskaja) that mutant males have less changes in this competition than normal ones.--Several observations have shown that there exists a possibility of a rapid modification of a dominant mutant character into a recessive.--Most mutations are however dominant to some extent.--This has been proved for lethals by R.A. Mazing.--This dominance of mutations makes them accessible to the action of selection in heterozygotes.--The concentration of heterozygotes for lethals, visible and sterility mutations is nevertheless very high. Thus in the population of Nikita Botanical Garden each fly is heterozygous for two mutations producing marked deviations from the norm.--276 lethals were detected among 1,905 chromosomes, 87 visible mutations among 1,627 chromosomes (X, 2 and 3) and 29 sterility mutations among 187 chromosomes (3) studied. Among 531 chromosomes investigated at Uman there was found one translocation (involving the second and the third chromosomes), lethal when homozygous.--41 third-chromosome lethals detected in one of micropopulations of the Nikita Botanical Gardens

Russian Abstracts

Belgovsky, M.L., Inst. of Gen. of the Acad. of Sci. of the USSR, Moscow. Frequency of minute chromosome rearrangements in relation to X-ray dosage.

We have studied the frequency-dosage relation of the lethal yellow "mutations" occurring in treated scute<sup>8</sup> chromosomes. These changes were chosen as, in our previous investigations (Belgovsky, 1938), we had come to the conclusion that they represent mostly minute rearrangements.

Two doses of X-rays, namely 1000 r and 4000 r were used. Treated scute<sup>8</sup> males were crossed to yellow females and 1 yellow daughters recorded. A total of 14,329 F<sub>1</sub> females in the heavy dose series and 45,167 F<sub>1</sub> females in the light dose series were counted. The ratio of "mutation" frequency at 4000 r to that at 1000 r, corrected for being 4.00. This result does not contradict the hypothesis that minute rearrangement frequency is proportional to the dosage. The difference between the frequency-dosage relations of gross and minute rearrangements thus established shows that the mechanisms of their origination are different. Assuming chromosome breaks to be induced by X-rays directly, we might explain his result on the basis of the independent breaks hypothesis in the case of gross rearrangements, and of the contact hypothesis applied to minute chromonema coils in the case of minute rearrangements. If, however, chromosome breaks are secondary effects of radiation and contact between chromosomes is a prerequisite of any break whatever, then it seems difficult to put forward now an explanation of the difference found.--The similarity of frequency-dosage relations in cases of lethals and minute chromosome rearrangements shows that either the former are mostly minute rearrangements, or the frequency-dosage curves of point mutations and minute rearrangements are similar. The first possibility can evidently be neglected, as not all lethals cytologically studied by different authors were found to be efficient and as it is very improbable that point mutations are unable to cause the death of the organism. Therefore we must adopt the second explanation and conclude that there is some fundamental similarity in the mechanisms of origination of point mutations and minute chromosome rearrangements. If the frequency-dosage curves of point mutations and minute chromosome rearrangements are really very similar or even identical, we must conclude that their relative frequencies at different doses are the same, and consequently the relative frequency of minute rearrangements in nature (at zero dose) must be as high as at different X-ray doses. This being the case, they must evidently play a rather important role in evolution.

Berg, R.L., Leningrad State University, Leningrad. Genetical analysis of wild populations and differences between wild and laboratory lines of *D. melanogaster*.

Increase in dominance of normal alleles (and correlated decrease in mutability) is gradually developed in the course of evolution in the interest of the individual. However, this makes possible the accumulation of harmful mutations in the genotype of the individual and the formation of systems of balanced lethals, and it limits the

possibilities for the species to make use of favorable mutations. Thus a conflict results between the interests of the individual and those of the species. --A lowering of dominance is possible in a species that has a structure adapted thereto, allowing competition between incompletely isolated groups.

spontaneous "mutations" (rather frequent in the scute<sup>8</sup> chromosome), proved to be

proved to be changes of 29 different genes, 21 sterility mutations--of 11 different genes. The high concentration of mutant genes may be partly as the result of the recurrent mutation of genes.--The frequency of occurrence of lethals and visible mutations in all the chromosomes per generation is 12.5% in the population of the Nikita Garden.--The mutability of the X, 2 and 3 chromosomes was studied. In the progeny of y w f females crossed with wild males the percentage of males carrying sex-linked mutations proved to be nearly the same (0.2% as in the wild state (0.297%)).--The mutation process and selection are apparently of primary significance in determining the gene composition of the population, while genetical-automatic processes are only of secondary significance. Wild populations differ from laboratory stocks by their high mutability and low dominance of normal allele. This difference is discussed more fully in another communication.--The detection of these distinctions between wild populations and laboratory stocks may serve as an indirect proof of the existence of a species structure of characteristically adaptive type.

Various lines of *D. melanogaster*, differing as regards their geographical origin, were compared with respect to different properties, such as dominance, mutability, etc. Such a comparison made it possible to establish correlations between the properties studied. That such correlations exist has been pointed out by Muller, Haldane, Plunkett, Wright, and Goldschmidt. There are grounds for assuming that these correlations result from the activity of the gene in ontogeny, which activity may differ in different lines. High activity of the gene in ontogeny (which depends on gene dosage, i.e., on excess gene dosage) leads to the rapid attainment of the margin of safety, the stability of form-Genesis reactions, and, consequently higher dominance, low mutation rate, and but slight ability to form phenocopies. From this it follows that the mutation rate should be the less affected by external factors, the lower the mutability.--The level at which intergenic and interchromosomal balance is established rises with an increase in gene activity. If balance lies at the basis of correlation and compensation in ontogeny, they (and consequently, viability under extreme conditions of treatment) must be the greater, the higher is gene activity.--The difference in stability between genes of the sex chromosome and those of the autosomes indicates a difference in the activity of these genes and, consequently, in their role in interchromosomal balance. This is indicated likewise by the different level of interchromosomal balance in sex chromosomes and autosomes (Muller, 1930) and by the presence of a mechanism of gene-dosage compensations in the X-chromosome (Muller, 1932, 1935). If the intensity of selection is a factor in gene stabilization, the difference in mutability between genes of the sex chromosome and of the autosomes is greater, the greater the dominance of the autosomal genes, since it is this dominance that creates the difference in intensity of selection between sex-linked and autosomal genes. The higher the level of intergenic and interchromosomal balance of the individuals of any given line and the greater the difference between the sex chromosome and the autosomes in this line, the less should be the viability of individuals with disturbed balance (super-females). The writer made a study of the differences between lines as regards: (1) the frequency of occurrence of spontaneous mutations, both lethal and visible, in the sex chromosomes and autosomes; (2) the dominance in them of mutations obtained from wild populations; (3) the viability of super-females appearing in the progeny from crosses between males from various lines and y w f females (in these experiments Edna Brissenden participated); and (4) the relative frequency of occurrence of mutations as between the sex chromosomes and the autosomes. The frequency of occurrence of phenocopies in different lines was studied by Lobashev and Solodovnikov, the dependence of the mutation rate on the physiological condition of the organism (the aging of the soma) by Zuitin and Pavlovets, and viability under extreme conditions of

treatment (with temperature, narcotics, etc.) by Vladimírsky.---The character of the correlations between these properties confirms their dependence on gene activity during ontogeny. The possibility of establishing correlations for the whole genotype and not merely for single genes shows that changes in the activity of different genes apparently take place in the same direction. The existence of correlations points to the possibility of the coordinated evolution of different properties. Wild lines differ, as a rule, from laboratory lines in their higher mutability, lower dominance of the normal allelomorphs, less difference in mutability between sex-linked and autosomal genes, and greater viability of the super-females in the progeny of the males of these lines. The difference in dominance is due both to differences of the normal allelomorphs and of the genotype as a whole.---The mutation rate among flies caught in nature is not lower than among flies of the same wild line kept in the laboratory for a few generations. The production of an isogenic line of one of the autosomes also does not affect the mutation rate in the autosomes. This indicates that high mutability is a property of the genes themselves rather than of the genotype as a whole. Excess gene dosage is apparently greater, as a rule, in laboratory than in wild lines. This may be due to the disturbance of the species system in laboratory lines resulting from isolation.

Dubinín, N.P., Inst. of Experimental Biol., Moscow:USSR.  
The genetic structure and the evolution of species.

(I) Introduction: The progress of modern genetics has shown the main hypothesis of Darwinism to be correct. Following the investigations of Charles Darwin genetics has opened up entirely new prospects for the study of evolution. We are indebted to S. Wright,

R.A. Fisher, J.B.S. Haldane and others for a number of fundamental works in the field of theoretical genetics and evolution.---The experimental and theoretical trends of our studies in evolution have started from the works of several Russian investigators--S.S. Tschetverikoff (1927), N.P. Dubinín (1931 etc.), D.D. Romashov (1931 etc.).---(II) Theoretical Analysis of the Problem: The genetic structure and evolution of the species has been studied theoretically in connection with the analysis of the significance of the system of species formed under the influence of ecological, biological, geographical and other factors. These studies have led Romashov and the author (1931, 1932) to show that the role played by casual stochastic deviations in the ratio of the gene frequencies (automatic genetic processes) is one of the most important phenomena of evolution which relates the mutation phenomena to the zone of action of natural selection.---During recent years these investigations have brought us to a series of conclusions regarding the form and nature of the automatic genetic processes in connection with various forms of isolation, migration, numerical fluctuations of populations and disproportion of sexes. The special significance of all these phenomena in the course of primary processes of species formation has been established. Several of these problems have been mathematically worked out by A.N. Kolmogorov, V.J. Glivenko and N.V. Smirnov.---(III) Experimental Study of the Problem (A) (The Mutation Process): The variability of the genes has been investigated in visible mutations (the sex chromosome) and lethal genes (the sex and second chromosomes) of a number of populations of D. melanogaster. The general pattern of gene variability in nature has been studied. The results show that all kinds of morphological and physiological hereditary changes occur as mutations. Certain peculiarities of the mutation process, which are connected with the distribution of the genes which change the mutation frequency, have been discovered.---The variability of chromosome structure has been studied in natural populations in a number of species of the Drosophila genus. An

attempt to elucidate the peculiarities of chromosome mutations of different species has been made by means of X-ray treatment and a comparative study of *D. melanogaster* and *D. funobris*.--(B) (Characteristics of Hereditary Variability in Populations): Populations contain an enormous hereditary variability which constitutes the basis for the evolution of separate populations and of the species as a whole. The most widespread mutation types are small physiological and morphological mutations, and recessive lethal genes, and then structural chromosome mutations (mainly inversions). Many gene mutations found in nature are but slightly specific. When crossed they show weak and unstable characters, and when analyzed, they disclose a very complicated hereditary picture. Clearly inherited and well pronounced mutations are rarely met with. The great majority of hereditary changes are recessive and are concealed in the populations in heterozygous condition, although a whole group of changes is manifested in a semi-dominant state. A study of small physiological mutations on such characters as fertility, duration of life, general viability etc., has led to the discovery of the great plasticity of natural populations with regard to a number of most significant physiological characters.--However, in spite of the enormous diversity of hereditary variability in populations, our investigations have disclosed a few specific and limited directions in the variability of single characters of the species, which directions proved to be parallel in a number of species.--Mass non-hereditary variability among populations is often found parallel with hereditary variability.--(C) (Distribution of the Mutations Among the Populations of Species): A study of the genetic structure of single populations has shown that it is caused by the mutation process, by the action of the automatic genetic processes which multiply some of the mutations up to the beginning of selection, and by the action of selection.--Of special interest are the distribution and the frequency of mutation of lethal genes; since their analysis discloses the real connection existing between the mutation process and negative selection.--Single populations (ecogenotypes) display (1) a great variety of small mutations, so that every female taken from nature gives rise to a stock differing from others by this or that character, (2) a number of pronounced gene mutations and chromosome changes, multiplied to a certain degree and limited as to diversity and (3) a great number of different lethal mutations.--As to the distribution of marked mutations throughout the system of populations, some genes were found to be inherent only to a given ecogenotype; whereas others have a certain area of distribution and, finally, some are found throughout the whole species.--In comparison with the complex distribution of the gene mutations, the structure of the species with regard to chromosome mutations is simpler.--Inversions, several endemic, a number widespread, and some distributed throughout the entire area of the species (USSR, Western Europe, North America), have been found in *D. melanogaster*. The automatic genetic processes which have operated throughout the entire history of the species play a great role in the distribution of the chromosome mutations. A comparative study of various *Drosophila* species has shown that they vary greatly in the distribution of chromosome mutations and in the phylogenic history of single chromosomes; all this has its counterpart in the specificity of the systems of different species. One *Drosophila* species was found to represent a permanent structural heterozygous condition and to display a peculiar picture of karyotype variability and evolution.--A study of the distribution of mutations within separate populations and within a system of populations makes possible an experimental analysis of the genetic basis of the system of species and of their evolution.--(D) (The Phenotypic Realization of the Genetic Structure of Populations): Investigations of the phenotypic realization of hereditary variability discloses the primary material which lies at the foundation of natural selection. This phenomenon is conditioned by homozygosity for recessive mutations and by heterozygosity and homozygosity

for dominant changes and for chromosome mutations. The great diversity of the gene content of populations makes for an enormous phenotypic variety for the action of natural selection at any given moment during the life of the population.--The specific character of the systems of different species plays a decisive role in the phenotypic realization of the genetic structure of populations.--The study of polymorphism of hereditary aberrations in Drosophila melanogaster makes it possible to elucidate both the selective role played by different mutation types and the correlation existing between the genetic constitution of populations and its phenotypic manifestation in nature.--(IV) Conclusion: The main factors determining the genetic structure within the system of the species are: variability of the genes, variability of the structure and of the number of chromosomes, variability due to crossing and hybridization, automatic genetic processes and natural selection.--Our theoretical investigations have disclosed the evolutionary role played by chance fluctuations in the ratio of the gene frequencies.--However, natural selection remains the principal factor which determines the adaptive evolution of populations.--Our experimental studies are in complete accord with theoretical analysis. They bring out experimentally the main factors which determine the structure and the evolution of populations.--The results obtained indicate the lines of further investigations.

Kerkis, J., Inst. of Gen., Acad. of Sci. of the U.S.S.R., Moscow.  
The effect of low temperature on the mutation frequency in D. melanogaster with consideration about the causes of mutations in nature (15:25 in original DIS)

Brown ebony males were treated with low temperature (ca. -5.5 - -6.0°C.) during 1.5 - 2 hours. A total of about 34,000 males were treated from which only 53 survived and gave offspring. They were crossed with ClB females immediately after treatment. Two series of F<sub>2</sub> offspring from ClB F<sub>1</sub> females crossed with y fa v f dl-49 males and from F<sub>1</sub> males crossed

with bw e females, were used for detecting the X-chromosome lethals and for studying the translocation frequency between the Y, 2, and 3 chromosomes respectively.--Of the 917 treated X-chromosomes, 13 sex-linked lethals were detected, while only 5 lethals were found out of 1934 X-chromosomes in the control series. The rate of mutation is 1.42% and 0.26% respectively, the difference being equal to 1.16% ± 0.318%, i.e. highly significant. No translocation was found among the 1254 cultures of treated series or in the control series.--A relatively high percentage of lethals in the progeny of the males treated by low temperature seems to indicate that the coincidence of the type of action of positive temperatures on mutation rate with the Vant Hoff rule for the rate of chemical reactions is apparently an accidental one. Mutations or changes in the very complex molecular structure of the gene occur as a result of breaks of any kind of the biochemical balance of the cell or chromosome, and these changes of structures of the gene can be induced by any physiological or physico-chemical factor external in relation to this structure. These conclusions are supported not only by our own results, but also by those of M. Navashin, obtained in 1933 et seq., on the frequency of chromosome mutations in aged seeds. In this sense it is hopeless, in our opinion, to search for any definite factor responsible for spontaneous mutational processes in nature. The cause of the production of mutations is therefore the same as that of the variability of matter of any kind, i.e. the interaction of genic substance with the surroundings in the broad sense of the word. The endless variety of kinds of interaction of the organism with surroundings together with the intercellular metabolism are the very causes of mutational process in nature. The short wave radiation has its own peculiarities because of its strong ability to induce strictly

local changes in the molecular structures. Numerous experiments have already indicated clearly enough that the role of this factor in the spontaneous mutational process in nature is very limited.

Khvostova, W.W., Inst. of Experimental Biol., Moscow. The influence of the proximal chromosome regions on the position effect of the cubitus interruptus gene in *D. melanogaster* (15:25 - 26 in original DIS)

The gene for cubitus interruptus, located near the spindle fibre of chromosome 4 of *D. melanogaster*, gives a position effect which consists in the dominance of the normal allelomorph growing feebler as it is removed from the inert region of chromosome 4 and as it becomes attached to the active regions (Dubinin and Sidorov, 1933; Khvostova and Gavrilova,

1935).--This paper describes 193 cytogenetically studied cases of the position effect of *ci+*. The following aberrations were found: 1 inversion in chromosome 4, 161 translocations between chromosome 4 and other autosomes; 12 translocations between the fourth and the X chromosomes, and 19 between the fourth and the Y chromosomes. The breaks were found along almost the entire length of chromosomes 2 and 3, except in the inert and in the adjacent active regions, which occupy about 20-25 per cent of the length of the arms.--The absence of breaks in these regions may be due to the specific methods used for detecting the translocations. The proximal regions do not give the position effect of *ci+*. This is proved by 11 aberrations with chromosome 4 (which did not give the position effect of *ci+*) showing 4 aberrations as translocations with the inert regions of chromosomes 2 and 3, and 2 with their proximal regions. In 5 cases the breaks occurred to the left of the *ci* gene, which was therefore not removed from the proximal inert region of chromosome 4.--A study of the manifestation of *ci* in translocations with breaks in different chromosome regions showed that two translocations with the most proximal breaks gave the most feeble manifestation of *ci*. This fact confirms the conclusion that the chromosome regions located close to the spindle fibre do not decrease the dominance of *ci+* (or do so only to a slight extent).--A study of translocations with the X chromosome (which gave the position effect of *ci+*) showed that translocations with breaks in the proximal parts of the active region and in the inert part to the left of *bb* give the position effect of *ci+*.--A study of translocations with the Y chromosome (which gave the position effect of *ci+*) showed that the breaks in the Y chromosome occur only in the distal regions in both the short and the long arms. In the proximal parts of both arms (about 1/3 of the length of each arm) no breaks were found.--The following conclusions are thus indicated: only the proximal part of the region of the X chromosome adjacent to the spindle fibre, and the proximal regions of both arms of the Y chromosome, are analogous to the inert regions of the autosomes in their action on the *ci+* gene (they do not weaken its dominance). The distal regions of the Y chromosome and a fragment of the inert region of the Y chromosome (the fragment located to the left of the *bb* gene) are analogous in their action to that of the active parts of the autosome (they weaken the dominance of *ci+*).--A study of 6 translocations which gave the position effect of *ci+* showed that chromosome 4 is attached to the proximal regions of chromosome 2 and 3 (regions 4L, 80, 81 on Bridge's map), which were removed through inversion from the spindle fibre. This shows that the proximal regions which normally do not give the position effect of *ci+* acquire this capacity when removed from the spindle fibre. The data obtained may be explained in the following way: (1) The inert substance of the chromosomes has a specific influence on the action of the *ci+* gene and this influence spreads to the adjoining parts of the active regions; when the active regions are removed from the inert regions, the influence of the inert substance stops. In this

case the inert substance of the X and Y chromosomes becomes differentiated into regions which have various actions on the  $ci+$  gene. (2) The position of the  $ci+$  gene in relation to the attachment point of the spindle fibre influences its action; the regions (both active and inert) lying near the attachment point of the spindle fibre do not decrease the dominance of  $ci+$ ; the remote regions do decrease it.--This specific influence of those chromosome regions located near the spindle fibre on the function of the gene may be explained by their peculiar structure and by the peculiar intergenic connections which affect the action of the  $ci+$  gene.

Kirssanow, B.A., Inst. of Experimental Biol., Moscow.

Relation between changes of crossing-over and X-ray dosages and certain problems of the process of crossing-over

(15:26 - 27 in original DIS)

Crossing-over was studied in chromosome 3 in D. melanogaster. X-rays increase crossing-over in the mid-region of chromosome 3 during the first three days after irradiation. The maximum changes of crossing-over in chromosome 3 are produced by X-ray treatment on the seventh day after irradiation. Crossing-over increases in the mid-region and decreases in the distal parts of the chromosomes. Increases in the X-ray dosages increase the changes of crossing-over at every stage of the experiment, but only to a certain limit, which varies for each stage. When the limit has been reached an increase of the dosage of X-rays either does not cause any considerable changes in crossing-over or even (in the case of very large dosages) decreases these changes. This top limit of the changes of crossing-over increases from the first to the seventh day after irradiation and reaches its maximum in the st-p region.--A comparison of the increase of crossing-over in the mid-region and of the decrease of crossing-over in the chromosome ends has shown that the predominance of the first over the second is not great; on the average it amounts to about 5 per cent of crossing-over (with certain fluctuations in some experiments). This predominance (we shall call it residual crossing-over) does not differ significantly from the data published on the frequency of translocations between chromosomes 2 and 3 in females and from the frequency of crossing-over in chromosome 3 in males. The approximate coincidence of the frequency of the above-mentioned processes and their similarity in genetic behavior and in reactions to external factors makes it possible to assume that the nature of these processes is similar.--The less the initial crossing-over frequency per unit of cytological chromosome length, the more crossing-over frequency may grow in comparison with the initial frequency.--The increase in the frequency of crossing-over in the regions h-st, st-p, and p-ss, caused by inversions in non-homologous chromosomes, takes place mainly at the expense of single crossovers. Irradiation of flies of the h st p ss/+++ structure increases the frequency of crossing-over within the regions h-st, st-p, and p-ss at the expense of multiple crossovers to a considerably higher degree than when there is an accumulation of inversions. X-rays decrease the frequency of crossing-over in the distal regions of the X-chromosome, whereas inversions in non-homologous chromosomes increase this frequency.

Koltzoff, N.K., Inst. of Experimental Biol., Moscow.

The participation of the chromosomes in cell-metabolism

(15:27 - 28 in original DIS)

(In the present paper are discussed the results of the author's investigations on the growing large oocytes of Triton, chick and pigeon). The individual chromosomes (from leptotene till the reduction metaphase) can be seen in the growing large oocytes of Triton, chick and pigeon.--The author holds that there is a marked difference between the stable genotype of the chromosome and its phenotype, which undergoes some

changes in the same chromosome during the various stages of development. Only the genonema belongs to the genotype, whereas chromoplasm, chromatin, vacuoles, etc. are phenotypical structures whose changes do not influence the hereditary qualities of the genonema.--The genonema is a fine filament with a series of enlargements--elementary chromomeres. It is present in every chromosome during all the developmental stages and is stretched out at the stage when the chromosomes attain their maximum growth. During metaphase, the genonema is coiled into a spiral or screw inside the chromoplasm; at this stage the chromosome is not divided into visible segments. In the initial stages of oogenesis, the genonema is gradually unrolled in length; this unrolling is accompanied by the appearance of the segments of the chromoplasm, i.e., of chromomeres. Later on the primary large chromomeres separate into many secondary ones. During the fourth and fifth periods of oogenesis, the reverse process takes place, i.e., the genonemata are coiled again and the secondary chromomeres are fused into larger tertiary chromomeres or even into a joined indivisible mass of chromoplasm.--The genonema and its genes remain chemically stable throughout oogenesis, and do not undergo any metabolic changes, viz. oxidation and reduction. The genonema represents the resistant genotype of the chromosome and can be changed only through mutation. The growth of the genonema in width is effected through apposition from the surrounding chromoplasm; its longitudinal splitting is to be looked for during the period of maximum length, i.e., during interkinesis. This splitting of genonemata can be separated by a long interval from the moment of division of the chromosomes, which can be observed in mitosis or meiosis.--Chemical metabolism takes place in the surrounding chromoplasm in the immediate vicinity of the genonema. Chromoplasm, together with chromatin which impregnates it during some stages or envelops it as a protective sheath, is continuously modified throughout oogenesis; it either accumulates large masses of nutritive substances from the Karyoplasm and nucleoli (especially in the primary or tertiary chromomeres) or is reduced to a fine, possibly monomolecular, film.--Chromatin is primarily a solid protective sheath of the chromosome which at some stages isolates the genonema from the karyoplasm and determines the definite form of the chromosomes which is convenient for karyokinetic movements.--Although the genonema does not take an immediate part in the chemical metabolism, its participation in the process of physico-chemical assimilation is very important. The genonema contains the ready patterns of all complicated proteids or other substances specific to the species or the individual, which had once been produced during the long process of evolution. It is improbable that such complex substances could be synthesized every time anew without the ready patterns. The genonema and its single parts, the genes, are "touch-standards"; in their neighborhood the process of assimilation is begun which from a physico-chemical point of view is an autocatalytic crystallization process. A new genonema is precipitated alongside the existing inherited genonema by apposition. The material necessary for the construction of new genonemata is yielded by aminoacids, polypeptids, carbohydrates, etc. which are contained in the chromoplasm; the energy necessary for crystallization is derived from chemical metabolism proceeding in the chromoplasm and the karyoplasm.--The process of assimilation around the genonema is especially intense during oogenesis, when the genonema is free from the chromatin-sheath and lies in the karyoplasm, being surrounded only by a very thin film of chromoplasm. With the appearance of the "lamp-brushes", the colloidal transverse threads arise in immediate connection with the elementary chromomeres; the whole nucleus is divided into sections corresponding to the "lamp-brush" chromosomes. When the process of crystallization is completed the transverse threads leave the chromosome and remain lying free in karyoplasm, which during meiosis joins the protoplasm of the egg. In this way, the egg is enriched with inherited gene-substances of

Chromatin, i.e., thymonucleic acid, completely disappears from the chromosomes during the second period of oogenesis and reappears again only in the fifth period; the chromatin should, therefore, by no means be included in the genotype of the chromosome and hence it cannot be regarded as an ingredient of the genes.

maternal origin. The same process of crystallization, i.e., assimilation of the gene-substances, very probably precedes each cell division during the interkinesis phase which is a truly active period of cell-life.--Only during the third period of oogenesis can the minute elementary chromomeres be noted, perhaps even genes, which are unrolled alongside of the straightened "backbone" thread of the genonema. At the earlier and later stages of oogenesis we observe fewer compound chromomeres; each of them including a lot of elementary, minute chromomeres. The chromoplasm of the compound primary and tertiary chromomeres functions as an isolated laboratory, where the products of assimilation of the neighboring genes undergo different chemical reactions. We can thus explain the phenomenon of the "position effect", by him first in 1927, that the genonema is a gigantic chain-molecule in which the radicals play the role of genes. During cell division two genonema-molecules (after conjugation--four molecules) unite, forming an "elementary micellary body".

Kozhevnikov, B. Th., Inst. of Experimental Biol., Moscow. Experimental study of crossing-over between non-sister genes in sister chromatids.

It follows from a number of studies that in sister chromatids no crossing-over occurs between sister genes. It has also been shown by Sturtevant and Muller that double Bar arises only if there is unequal crossing-over between non-sister chromatids. Bridges has described Bar as a duplicated section. The conclusion

is thus indicated that crossing-over between sister chromatids does not take place even in those cases where it ought to occur between non-sister genes.--The purpose of the present study was to verify this conclusion on chromosome structures in which the duplication is sufficiently great.--In order to obtain the necessary stocks the inversions B25 and B26, where the left break had occurred near Bar and the right one in the inert region, were used and the  $sc^8$  inversion whose left break is near the left of  $sc$  and the right one in the inert region in front of the  $bb$  gene. Females heterozygous for  $sc^8$  B25 or B26 were obtained which were crossed with males carrying the translocation Bar Stone. A long chromosome forms in these females, as a result of crossing-over on the B- $bb$  region (i.e. in the region which is common to both inversions). The proximal part of the long chromosome consists of the whole chromosome  $sc^8$  (except a small region at the left end containing the  $y$   $ac$  genes), whereas its distal part consists of chromosome Bar 25 (or B26), which is devoid of the right region containing the inversion and the spindle fibre. The  $F_1$  flies which had received the long chromosome should be females, but they are non-viable because they are hypoploid for a considerable region of the X-chromosome. However, when the ovum containing the long chromosome is fertilized by a spermatozoon which carries only the right region of the X-chromosome from the translocation Bar Stone, the flies prove to be quite viable. In this structure there is hypoploidy for only a minute region of  $y$   $ac$ . These females may be bred as a constant stock when they are crossed with males carrying the translocation Bar Stone. Two such stocks were formed: 25/8 and 26/8; the figures indicate which inversions were used for this line. In the stocks thus obtained the long chromosome contained a recessive allomorph in its distal part and a dominant allomorph in the proximal part, or vice versa. Double crossing-over between the distal half of the long chromosome and the proximal region of the sister chromatid was studied in this structure. This was proved by the occurrence of homozygous recessives (the  $ct$  gene served as indicator in the 25/8 stock, and the gene vermilion in the 26/8 stock).--The structure of the 26/8 stock was checked cytologically.

Malinovsky, A.A., Inst. of Experimental Biology, Moscow.

The role played by chromosome inversions in the evolution of populations.

(15:29 - 31 in original DIS)

sometimes primary in their nature? What is the typical fate of an inversion in a population?--2. The fate of an inversion in a population has been studied in connection with (1) the cessation of crossing-over in an individual heterozygous for the inversion, and (2) the appearance, distribution and selection of gene mutations. Inversion signifies the one of the two orders of genes which is primarily of the lowest frequency.--3. The number of useful mutations spreading in every chromosome form is approximately proportional to the concentrations of this form, and normal forms therefore accumulate more useful mutations. (a) If a useful mutation has on the average a higher coefficient of selection in homozygous condition than in heterozygous (recessiveness), the inversion is gradually eliminated by the better adapted normal form. Two possibilities exist (1) A normal homozygote is better adapted than a heterozygous inversion (heterosis is low), inversions are eliminated instantaneously. (2) Heterozygous inversions are better adapted than normal form (heterosis is great). An equilibrium of frequencies results, but the accumulation of new useful mutations shifts the equilibrium to the advantage of normal forms. Both these cases occur apparently very often. They explain the presence of low inversion frequencies in species with extensive populations where selection predominates over change frequency fluctuations (b) But if every separate mutation is on the average more useful in heterozygotes than in homozygotes a frequency equilibrium becomes established for inversions. Equilibrium is shifted with the accumulation of new mutations and attains a limit of 0.5.--4. The occurrence, chance frequency fluctuations and the selection of harmful mutations lead to frequency equilibrium (according to Sturtevant and Mather) owing to selection in favour of heterozygous inversions. However the point of view of these authors that selection tends to equalize inversion frequency and normal forms is erroneous. Equilibrium only fixes the initial frequency, although not very firmly.--5. (a) The conclusions of Sturtevant and Mather may be justified only in case it is assumed that harmful mutations displace their normal allelomorphs by chance so that (1) separate harmful mutations invade the whole class of chromosomes (inverted or normal) and become fixed there; (2) The number of harmful mutations in chromosomes of different mutations is equal. But such conditions are hardly possible (except in small populations). (b) But if this takes place permanent heterozygotes may be formed. Disturbances of crossing-over which hinder the passing of harmful mutations from one chromosome class into another prevent complete degeneration which is possible in small populations (Wright, etc.) and assure the preservation of adapted heterozygous forms.--Conclusions: 1. As selection does not favor classes small in number (sec. 3a) the spreading of inversions is mainly the result of chance causes (undirected frequency fluctuations, chance inclusion of a specially useful mutation, etc.).--2. In more isolated smaller populations greater inversion frequencies may be expected.--3. Permanently heterozygous species (Dubinin, Sokolov) may apparently arise in three ways: (1) If the accumulating mutations are more useful in heterozygotes than homozygotes (sec. 3b); (2) If harmful mutations practically attain 100 per cent in relation to the inversion frequency or the normal chromosome form (sec. 5a and b); (3) If the forms differ from one another by inversions and give hybrids which have high heterosis.--4. Inversions may contribute to the divergence of varieties in two cases: (1) if two varieties

1. Many related species differ from one another by inversions. On the other hand, many species are heterozygous for inversions (Dubinin, Sokolov and Tiniakov, Sturtevant and Dobzhansky, Dobzhansky and Quaal, etc.). Is the divergence of karyotypes always the result of the divergence of species, or are the karyotype changes

What is the typical fate of an inversion

in a population?--2. The fate of an inversion in a population has been studied in connection with (1) the cessation of crossing-over in an individual heterozygous for the inversion, and (2) the appearance, distribution and selection of gene mutations. Inversion signifies the one of the two orders of genes which is primarily of the lowest frequency.--3. The number of useful mutations spreading in every chromosome form is approximately proportional to the concentrations of this form, and normal forms therefore accumulate more useful mutations. (a) If a useful mutation has on the average a higher coefficient of selection in homozygous condition than in heterozygous (recessiveness), the inversion is gradually eliminated by the better adapted normal form. Two possibilities exist (1) A normal homozygote is better adapted than a heterozygous inversion (heterosis is low), inversions are eliminated instantaneously. (2) Heterozygous inversions are better adapted than normal form (heterosis is great). An equilibrium of frequencies results, but the accumulation of new useful mutations shifts the equilibrium to the advantage of normal forms. Both these cases occur apparently very often. They explain the presence of low inversion frequencies in species with extensive populations where selection predominates over change frequency fluctuations (b) But if every separate mutation is on the average more useful in heterozygotes than in homozygotes a frequency equilibrium becomes established for inversions. Equilibrium is shifted with the accumulation of new mutations and attains a limit of 0.5.--4. The occurrence, chance frequency fluctuations and the selection of harmful mutations lead to frequency equilibrium (according to Sturtevant and Mather) owing to selection in favour of heterozygous inversions. However the point of view of these authors that selection tends to equalize inversion frequency and normal forms is erroneous. Equilibrium only fixes the initial frequency, although not very firmly.--5. (a) The conclusions of Sturtevant and Mather may be justified only in case it is assumed that harmful mutations displace their normal allelomorphs by chance so that (1) separate harmful mutations invade the whole class of chromosomes (inverted or normal) and become fixed there; (2) The number of harmful mutations in chromosomes of different mutations is equal. But such conditions are hardly possible (except in small populations). (b) But if this takes place permanent heterozygotes may be formed. Disturbances of crossing-over which hinder the passing of harmful mutations from one chromosome class into another prevent complete degeneration which is possible in small populations (Wright, etc.) and assure the preservation of adapted heterozygous forms.--Conclusions: 1. As selection does not favor classes small in number (sec. 3a) the spreading of inversions is mainly the result of chance causes (undirected frequency fluctuations, chance inclusion of a specially useful mutation, etc.).--2. In more isolated smaller populations greater inversion frequencies may be expected.--3. Permanently heterozygous species (Dubinin, Sokolov) may apparently arise in three ways: (1) If the accumulating mutations are more useful in heterozygotes than homozygotes (sec. 3b); (2) If harmful mutations practically attain 100 per cent in relation to the inversion frequency or the normal chromosome form (sec. 5a and b); (3) If the forms differ from one another by inversions and give hybrids which have high heterosis.--4. Inversions may contribute to the divergence of varieties in two cases: (1) if two varieties

live in adjacent regions but under different conditions. Here the difference in respect to an inversion inhibits the diffusion of useful genes from one region into another where they are harmful; the inversion unites small mutations into a large complex which perishes much sooner when it enters a region to which these mutations are not adapted. The mutations perish sooner because the coefficients of the selection of mutations are summed up. This prevents the levelling of genotypes of contiguous genotypes. (2) if an inversion and a normal form have accumulated recessive useful mutations the heterozygote for the inversion is less well adapted. In this case it is not the best adapted form which necessarily spreads but the one whose product of frequency and coefficient of selection is higher ( $\bar{P}, \bar{P}$ ).

As inversion frequency varies some populations are invaded by a normal chromosome and others--by an inversion. Mutation complexes containing an inversion differ from those with a normal form.--Appendix--1. Selection of the inversion (I)  $\Delta P = P \alpha [P(\alpha - \gamma) - Q(\beta - \gamma)]$

$$(II) \bar{P} = \frac{\beta - \gamma}{\alpha + \beta - 2\gamma}$$

$\bar{P}$  stable when  $\gamma > \alpha; \gamma > \beta$ ; unstable when  $\alpha < \alpha; \gamma < \beta$ . In the latter case  $\Delta P > 0$  if  $\bar{P} > \bar{P}$  and  $\Delta P < 0$  if  $\bar{P} < \bar{P}$ . 2. The influence of the accumulation of useful mutation (III)  $\Delta d = \gamma \bar{P}; \Delta \beta = \gamma Q$ . If the initial coefficient of selection is  $d = 0; \beta = 0; \gamma = 0$ , then (IV)  $P = \frac{1 - K - F}{1 - \theta + 2\theta(1 - K)}$  If the initial mutations are  $\alpha \neq 0; \beta \neq 0$  and  $\gamma \neq 0$  but  $\gamma > \alpha; \gamma > \beta$  and  $P = \bar{P}$  then (V)  $\bar{P} = \frac{P(1 - \theta) + \theta(1 - K)}{1 - \theta + 2\theta(1 - K)}$

$$\text{where } \theta = \frac{\Psi}{\alpha + \beta - 2\gamma}$$

3. The accumulation of harmful mutations: A. It is assumed that mutations undergo chance frequency fluctuations, which are equal in both chromosome classes ( $P_i$  attains an equal percentage of mutations which have arisen in the inversion and in the normal chromosome).--(VI)  $E d_i = \gamma; P_i(2d_i + \frac{P_i(\beta - 2d_i)}{P}); E \beta_i = \gamma; P_i(2\beta_i + \frac{P_i(\alpha - 2\beta_i)}{Q});$

In this case (II)  $\bar{P}' = \bar{P}$

B. It is assumed that harmful mutations may undergo chance distribution and fixation in an inversion and in a normal chromosome without any inhibition from selection.--(VII)  $E d = \frac{\Psi}{2N}; E \beta = \frac{\Psi}{2N}; E \gamma = \frac{\Psi K}{N}$

In this case (II)  $\bar{P} = \bar{P}$

$\bar{P}$  - initial inversion frequency,  $\bar{P}$  - inversion frequency of equal value,  $\bar{P}$  - same in the next generation.

$Q, Q, Q'$  - corresponding values for a normal chromosome;  $\alpha$  - coefficient of selection for a homozygous inversion;  $\beta$  - coefficient of selection for a heterozygous form;  $\gamma$  - coefficient of selection for a normal form. (the coefficients of selection have been taken as regards the conditional level of initial adaptability).  $\bar{\alpha} = \alpha - \gamma; \bar{\beta} = \beta - \gamma$  and  $\alpha_i; \beta_i$  coefficients of selection of the same chromosome classes in so far as they depend on the mutations of the  $i$  category ( $E d_i = \alpha; E \beta_i = \beta; E \gamma_i = \gamma$ ).

$P_i$  - random frequency (as regards the population as a whole).--Harmful mutations by chance of this frequency are placed into category  $i$ .  $\delta$  - average coefficient of selection of mutations belonging to category  $i$  in homozygous condition;  $\delta_i$  - average coefficient of selection of mutations belonging to category  $i$  in heterozygous condition;  $\Delta$  - growth of value during one generation;  $K$  - average gene manifestation in a heterozygote; as compared to a homozygote.--  $\Psi$  - coefficient of proportionality showing the increment of adaptability in its dependence on the occurrence of useful mutations;

$\gamma_i$  - number of mutations in category  $i$ .--  $\Psi$  - average number of harmful mutations per chromosome arising during one generation multiplied by their average coefficient of selection in homozygous condition.

Masing, R.A., Pavlov Biol. Inst.  
Koltushi, Leningrad.  
Different viability of heterozygotes for lethals in *D. melanogaster*.  
(15:31 - 32 in original DIS)

Comparative viability of flies heterozygous for different sections of chromosome (with the lethal and without) obtained as a result of crossing-over, has been studied in three stocks. In all the stocks flies heterozygous for a section of the chromosome con-

taining no lethal showed no increase in viability. Increased viability of flies heterozygous for a section of the chromosome containing the lethal was observed in the progeny of all individually studied crossover males carrying the lethals in the stocks 627 and 15MF. This suggested that in these stocks either the lethal itself was a dominant gene of increased viability with recessive lethal effect or that there was a dominant modifier located near the lethal.--Apparently the increased viability of flies heterozygous for the lethal in stock T201 is due to interaction between the lethal gene and a dominant modifier located so far from the lethal that crossing-over between both genes is frequent.--In two sets of experiments increased viability of heterozygotes for lethals was met with in four cases. Semidominant lethal effect (i.e. low viability of heterozygotes for lethals) was observed in 24 cases.--14 stocks studied contained different lethals which originated spontaneously and under the influence of a chemical agent in laboratory stocks. The viability of heterozygotes was determined by means of crossing Cy/l males with ebony and normal females from inbred stocks.--Not a single case of decreased viability of heterozygotes for lethals was observed. Increased viability in the heterozygous state was observed in seven stocks, the results being similar when crossing with ebony females and normal females. In 5 stocks increased viability of heterozygotes was observed only when crossing these males wither with ebony females or with normal females. The results obtained in this work are not sufficient to explain this peculiar effect.--In some cases lethals are not indifferent to the life of heterozygotes. This suggests the important role of selection among heterozygotes in alteration of concentration of lethals in populations, such selection being either positive or negative.

Neuhaus, M. E., Genetical Lab.,  
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Relation between the length of the X-chromosome of *D. melanogaster* and the frequency of X-ray translocations.

(15:32 in original DIS)

Two hypotheses were proposed to explain the mechanism of structural changes of chromosomes induced by X-rays. One is the breakage hypothesis of Stadler (1932); the other is the crossing-over or contact hypothesis proposed by Serebrovsky (1929). Some indirect data speak for the second hypothesis.--In studying the mutation process induced by

X-rays (dosage 4000 r) in X, XY<sup>S</sup> and XY<sup>L</sup> spermatozoa (Neuhaus, in press) there was found 2.93±0.65% of translocations between the X-chromosome and the autosomes in X-bearing spermatozoa, 6.98±1.04% in XY<sup>S</sup>-bearing spermatozoa, and 10.00±1.25% in XY<sup>L</sup>-spermatozoa. The percent of translocations between the autosomes in X-bearing spermatozoa was 10.59 (n=670), while in XY<sup>S</sup> spermatozoa it was 9.65 (n=601) and in XY<sup>L</sup> spermatozoa 11.89% (n=580).--The above data are difficult to explain from the stand-point of the breakage hypothesis. According to this hypothesis breaks occur independently from one another and, the breaks having occurred, the fragments rejoin at random. If this is the case we must expect a decrease of the percentage of translocations between the autosomes in XY<sup>L</sup> spermatozoa as compared with that in XY<sup>S</sup> and X spermatozoa, and in XY<sup>S</sup> as compared with X-bearing spermatozoa. But this is not the case.--The above data are easily explained by the contact hypothesis. The increase in the length of the X-chromosome leads to an increase in the volume of the nucleus of the spermatozoon, therefore more

quanta of X-rays are absorbed. The increase in the length of the X-chromosome does not influence the interrelation and the frequency of mutual contacts of the autosomes, hence the percentage of translocations between the autosomes remains the same. In  $XY^L$  and  $XY^S$  spermatozoa the frequency of contacts between the X-chromosome and the autosomes is increased and so is the number of quanta absorbed, and therefore it is to be expected that the percentage of translocations between the X-chromosome and the autosomes must rise.

Olenov, I.M., and Kharmac, I.S.,  
Leningrad: State Roentgeno. Inst.  
Transformation of normal genotype in wild D. melanogaster populations.

(15:32 - 33 in original DIS)

The results of the analysis of a wild population of D. melanogaster together with existing literary data led us to suggest that the process of inclusion of mutant genes into a normal genotype is of common occurrence in wild D. melanogaster populations. Our aim was to verify the exactness of this suggest-

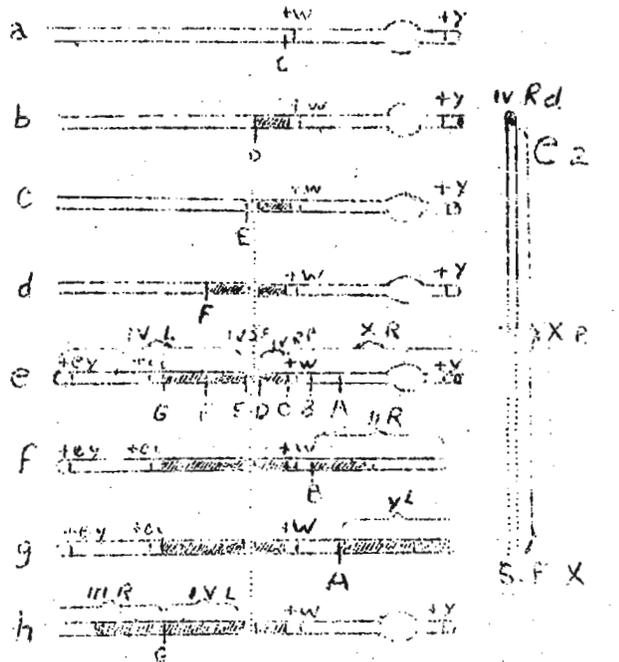
ion. With this purpose we studied all the visible mutations belonging to the 2 chromosome, that in the summer and autumn of 1938 came to our observation in the Uman population as well as all the visible mutations in the Bobrov population (Voroneg region) and in that of Simferopol (Crimea) analyzed at the same time.--We paid special attention to such characters which, while appearing in some strains as the sole recorded change occur at the same time as a peculiarity accompanying the principal manifestation of other genes, because many visible mutations belong to the number of activators of the gene, included as an effect of selection into the normal genotype. Additional to the changes in the posterior crossvein five alterations, presenting an analogous picture of inheritance, were brought into evidence. Four of these (not unfolded wings, divergent wings, indentations in the inner edge of the wing, dachsoid legs) are incompletely manifested similar to the abnormality in the posterior crossvein. The same four alterations we frequently have noted as a part of the pleiotropic effect of other visible mutations. The strains in which all these five alterations have been observed together with changes in the posterior crossvein are found in 50 to 60% of all chromosomes that contained visible mutations.--We consider that by natural selection mutant genes are simultaneously included into the normal genotype of wild D. melanogaster populations, that the transformation of the normal genotype is a mass process continuous in its occurrence. The transformation of the normal genotype occurs no doubt not only at the expense of visible mutations; of great importance are the transgenerations which produce no phenotypical changes recognizable on application of the sampling method habitually employed. Such a conception of the normal genotype represents, we should say, a certain progress as compared with the generally accepted view according to which the normal genotype is considered to represent a relatively stable system. The origination of new varieties and races, the starting point of the species producing process, appears to us not as a movement beginning in every case from some stationary point but as an occasionally originating and necessary result of the action of natural selection which uninterruptedly transforms the normal genotype.

Panshin, I.B., Inst. of Experimental Biol., Moscow.

The role of heterochromatin in the position effect of the white (mottled) and cubitus interruptus genes.

(15:33 - 34 in original DIS)

A translocation (involving the X and 4 chromosomes, causing the  $w^{mt}$  position effect) favorable for the study of the  $w^{mt}$  and  $ci$  position effect in the same chromosome was used for obtaining (through X-ray treatment) secondary rearrangements which change the mode of variegation of the white gene and call forth the position effect of  $ci$ . The structure of this translocation (designated as  $w^{mtll}$ ) is shown in the diagram (e1, e2). It is clear that a mutual translocation has occurred between the X (break to the right from the  $3C_2$  double band) and the right arm of chromosome 4. The diagram shows that rearrangements of different structures were tested (obtained from  $w^{mtll}$  original rearrangements and more complicated cases derived from X-raying of secondary rearrangements, in all 45 rearrangements) in which the quantity of heterochromatin connected with the white gene was changed. It was found that the more heterochromatin is associated with the  $w$  gene within one chromosome, the fewer pigmented cells are present in the eye, i.e. there is an inverse proportionality between the quantity of heterochromatin and the number of cells in which the action of the  $w$  gene remains unchanged (diagram). Diagram a represents the normal X or the absolute reverse from  $w^{mtll}$ ; b and c--the almost normal eye, d--the dark  $w^{mt}$ ; f and g--few pigmented cells; h--absolutely pigmentless stock which give  $w^{mt}$  with an extra Y.--Note that variegation does not depend on the position of the  $w$  gene as regards the chromocenter, but only on the quantity of heterochromatin connected with the gene within the chromosome, since (1) the mode of variegation is equal in the structures b and c, whereas in b white is removed from the chromocenter and in c--connected with the spindle fiber (i.e. with the whole chromocenter), and (2) in c, d, e, f, g, h--the position of the gene with respect to the chromocenter is the same, but the number of pigmented cells decreases with the increase of the quantity of heterochromatin within the chromosome connected with the gene.--It is clear that all the heterochromatic regions of the chromosomes are identical (except X and Y) in their action on the  $w$  gene and each heterochromatic region can be divided without any loss of its qualitative properties. The inert region of the X and probably the Y-chromosome consists of two parts; the proximal regions being homologous to the autosomal heterochromatic region, the distal ones to the active regions.--A comparison of the causes underlying the secondary position effect of the  $w$  gene (variegation changes as a function of heterochromatin quantity) and the primary position effect of the  $ci$  gene (decrease of dominance of the normal allomorph) shows that these causes are similar, or even identical. In both cases the changes of the gene effect are caused by changes of the connection between the gene and the heterochromatin within the chromosome. This conclusion, which has also been reached by W.W. Khvostova for the  $ci$  gene, is confirmed by the fact that in the  $w^{mtll}$  chromosome the breaks in the IVL inert region with subsequent translocation to the active region almost always change both genes ( $w$  and  $ci$ ). This illustrates the dependence



e--(the  $w^{mtll}$  original stock) the relatively light  $w^{mt}$ ;

of both genes on a third element (IVL inert region). The action of the *ci* gene should also depend on the quantity of heterochromatin connected with the gene, since qualitative observations indicate that an inverse correlation takes place between the degree of reversion from  $w^{mt}$  to the normal phenotype and the manifestation of *ci* in translocations of the  $w^{mtll}$  chromosome.--The single quantitative interdependence between the gene action and the quantity of heterochromatin connected with the gene can be considered as an indication of the chemical reaction between the genes and heterochromatin products involved, and corresponds in general to the chemical hypothesis of Muller and Offerman of the position effect. This conclusion corresponds to the findings of Schultz and Caspersson that nucleic acid is of great significance for the position effect caused by the heterochromatic regions.

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Cytological properties of inert  
regions and their bearing on the  
mechanisms of mosaicism and  
chromosome rearrangement.

(15:34 - 35 in original DIS)

The fundamental structural element of a chromosome is a chromiote with the adjacent section of a chromonema, the loci of individual genes being connected with both of them.--"Heterochromatin" and "euchromatin" are not present as specific substances in chromosomes, but any chromosome section can assume a "heterochromatic" or "euchromatic" condition. An intimate

conjugation of chromonemata and the presence of thymonucleic acid in chromiotes only are characteristics of a "euchromatic" condition of a chromosome section. A weakening of the conjugational properties of chromonemata and the appearance of thymonucleic acid in chromonemata are characteristics of a "heterochromatic" condition of a chromosome section. As a result, the latter assumes diffuse structure. The euchromatic condition is usually characteristic of active sections of chromosomes, whereas the regions situated near the spindle fiber attachments ("inert" regions) are more frequently found to be in heterochromatic condition.--An approximation of a euchromatic active region of a chromosome to the region of the spindle fiber attachment leads to its transformation into a heterochromatic region. It can be made to revert again into the euchromatic condition by the influence of internal (additional inert regions) or external (temperature) factors.--A small heterochromatic inert region inserted between two active regions assumes the euchromatic condition. All this indicates that chemical processes responsible for the euchromatic or heterochromatic condition of a chromosome section are reversible. When in euchromatic condition, inert regions are incapable of inducing a heterochromatic condition in the adjacent sections.--A change of the euchromatic condition of an active section into the heterochromatic one is accompanied in mosaic strains by a change in the expression of characters controlled by a given chromosome section, this change being most frequently from the dominant expression to the recessive one. When the section in question again assumes the euchromatic condition (influence of the Y-chromosome, temperature) the expression of the corresponding character also becomes changed and it develops into the dominant form.--Consequently the course of development of a given character in a mosaic individual depends at a particular moment not upon the presence or absence of a chromosome section containing certain genes, but upon its definite condition, the latter having definite morphological and chemical characteristics.--Inert regions are specific chromosome sections containing many loci homologous to one another ("multiplications"). The mutual attractions of these loci is usually described as a non-homologous conjugation of chromosomes.--An investigation of the behavior of different sections of the X-chromosome at conjugation led

to the discovery of the presence of "inert" regions in the following sections: 1A1-3, 2B, 3C3-3C6, 4C3-7, 7C, 8C, 9B, 11A 11CD, 12EF, 15F, 17A, 19E3-4, 20A-F. In contrast to other parts of the X-chromosome, all sections mentioned above have a tendency to conjugate with one another.-- Distal "inert" regions apparently represent a fundamental feature of the primary organization of a chromosome just as the inert regions located near the spindle fiber attachments do. The nature of "interstitial inert regions" is not clear as yet.--A comparison of the distribution of the best-known breaks in the X-chromosome (inversion  $sc^8$ ,  $sc^4$ ,  $y^{3P}$ ,  $w^{M4}$ ,  $rst^3$ ,  $ClB$ ,  $d49$ ,  $B^{M1}$ ,  $B^{M2}$  and nine breaks of the inversions studied by Hoover, 1938; translocations  $w^{M5}$ ,  $w^{M6d}$ , etc.) with that of the inert regions showed that all these breaks had occurred in inert regions or in their immediate vicinity.-- A study of the distribution of 141 cytologically detected X-chromosome breaks obtained in the course of another experiment by N.P. Dubinin, V.V. Khvostova, and V.V. Mansurova, clearly showed that the distribution of breaks was not at random. Although a chromosome can be broken in any point the breaks in inert regions are relatively much more frequent than in the active ones. For example, 8 breaks were found in 11A, 9 in 11CD, 6 in 19E, etc.; the probability of chance occurrence of 6 breaks within just one section was in this experiment 0.00105 and that of 8 breaks only 0.00002. The greatest number of breaks was found in sections 11 and 12 where inert regions represented by sections 11A, 11CD and 12EF are also clustered.--The data obtained lead to the conclusion that the property of inert regions of conjugating with one another must be a very important factor in the mechanism of chromosome rearrangement. The increased breakability of a chromosome in the inert regions ("weak spots" being especially characteristic of sections 11A and 19E) is also of importance. It can be due either to the peculiarities of chemical structure of inert regions, or to a conflict of forces of attraction. Both the property of conjugation and the breakability are evidently important factors in the process of rearrangement and both hypotheses proposed to account for the process - the independent breaks and the contact one - may be correct. The second, however, seems to the author to be more plausible.-- Most of the frequently mutating genes of the X-chromosome are located near the inert regions. Such are: yellow (inert sections 1A-1-3), white (inert section 3C3), cut (inert section 7C), lozenge (inert section 8C), forked (inert section 15F), and bobbed (inert section 20C). This does not seem to be a mere coincidence. Considering this fact in connection with the increased frequency of X-ray mutations in chromosome sections approached to inert regions as a result of chromosome rearrangements, and with the somatic variability of characters depending upon genes located in such sections (mosaicism), we are justified in suggesting that the spontaneous mutability of genes is also somehow related to their position with respect to inert regions.

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Multiple linear repetitions of the Bar gene.

The changes to ultra-Bar, often found by Zeleny in Bar stocks, were explained in 1923 by Sturtevant and Morgan as a double repetition of the Bar locus caused by unequal crossing-over. New proofs of this explanation were given by the subsequent investigations of

Sturtevant (1925-1928). Sturtevant and Thompson did not succeed in their attempts to obtain more complex repetitions than double Bar, and in order to explain this failure Sturtevant assumes that such structures are sterile or poorly viable whereas Thompson assumes that the lower limit of the number of facets of the Bar type eye is attained in double Bar and that the addition of new Bar genes can diminish the eye no more.--As it is possible to compare approximately the action of a definite temperature with that of a definite

number of Bar genes, a prognosis of quadruple-B was made by determining the temperature reactions of  $B^d$ . This prognosis was justified later on.-- However, in view of the possible transgression of  $B^d$  with  $B^q$  the search for  $B^q$  was made not in the stock with free X-chromosome, but in the  $B^d B^d$  stock with attached X's.--It was assumed that if a  $B^q B^+$  female were to arise following unequal crossing-over in this stock (even not being phenotypically different from her  $B^d B^d$  sibs) she could be identified by her  $B^+ B^+$  daughters which are bound to arise from equal crossing-over in the progeny of  $B^q B^+$  females.--Quadruple-B was found after 8237 cultures had been investigated (with 25-35 females in each culture). The homozygous  $B^q$  has 8-12 facets, and the heterozygous 24 facets.--At the beginning the  $B^q$  males were sterile but when the mutation yellow was eliminated fertile  $B^q$  males appeared.-- In extensive experiments on unequal crossing-over in  $B^q/B^d$  stock sextuple-B was found with 17 facets in  $B^s/+$  heterozygotes (no homozygotes were obtained).-- $B^q$  contains 5 and  $B^s$  7 regions 16A 1-4 in the salivary gland chromosome. In extensive experiments on unequal crossing-over in the homozygous stock of  $B^q$ , a crossover female with 4 facets was obtained. Probably it was a heterozygous octuple B. This female left no progeny.-- The structure infra- $B^q$  with 19 facets and a fourfold repetition of a new allelomorph of B with 8 facets was obtained. Structures similar to  $B^d$ ,  $B^q$  etc. may be looked upon as a special kind of chromosome aberrations. The linear repetitions of B are instances of experimental polymery. The position effect which was found by Sturtevant does not increase in more complex Bar structures. An analysis of the curves which characterize the number of facets in different B structures shows that each doubling, each multiple increase of the number of B genes inhibits one or two divisions of the facet forming cells. The coefficient of dominance of different B structures has been determined. The reduction of ocelli in  $B^q$  has been investigated in connection with the problem of the field of action of a gene. The frequency of crossing-over on the distal and proximal limits of  $B^q$  repetitions has proved to be equal, which gives some genetical data on the dynamics of conjugation along the chromosome.--Unequal crossing-over between different allelomorphs of Bar has also been investigated.--Non-crossover reversions of a new B allelomorph caused by a mutability-increasing gene have been studied. On the basis of the data obtained, it is possible to think of the changes of B as not affected by alteration of gene balance, because in the structures which retain several insertions of 16A 1-4 the character B disappears after such a mutation although the gene balance remains intact.--X-rays (more than 20,000 individuals from irradiated  $fB^s/+$  females were studied) did not cause unequal crossing-over between sister chromatids. In order to elucidate the problem of the simultaneous mutation of neighboring genes the effect of X-rays on the  $B^q$  structure was studied. Simultaneous reverse mutations in the four regions of B did not appear. Simultaneous changes in two regions were found and are most frequently a result of the position effect, but not of a simultaneous mutation.--In general the B locus shows changes both of the mutation and of position effect types and there is no ground for explaining all the changes by either of them.--In view of a number of traits common to Bar and Hairy wing, an investigation of unequal crossing-over and a cytological investigation were carried out in the stock  $y Hw$ . Unequal crossing-over does not occur, but a duplication was found in the salivary glands.

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The mutation process in ageing sperm of *D. melanogaster* and the problem of the specificity of the action of the factors of mutation.

In a number of papers (1935, 1936, 1938) the author has expressed his point of view that external mutation factors cannot be looked upon as "action accelerators" only. The question arose as to the specificity of the action of external factors, which remains latent in the qualitative peculiarities of the mutation process. The cytogenetic study of

lethal mutations occurring (1) under the action of X-rays, (2) spontaneously and under the action of chemical factors, showed that the second are devoid of those chromosome aberrations which characterize the first. However, it was necessary to obtain material on spontaneous lethals arising in mature sperm in order to show that the character of the mutation process depends not only on external factors but on the peculiarities of those stages at which they occur. The author chose the mutational action of ageing sperm. ClB females fertilized by forked males were isolated from the males. The experimental and control flies were obtained from the same females, the only difference being that the control consisted of eggs laid during the first days after the female was inseminated while the experimental eggs were laid 30 days and often longer after insemination. Throughout these 30 days the females were placed without the males on a poor diet at 18°C. and were transferred later onto a normal diet.

Table 1

	Number of initial females	Number of analyzed chromosomes	Mutations			Remark
			Lethal	Semi-lethal	Total Percentage	
Experiment	271	2804	18	6 <sup>x</sup> /	24 0.856±0.174	<sup>x</sup> / Among them-- 3 with visible manifestation.
Control	306	6327	11	4 <sup>x</sup> /	15 0.237±0.184	<sup>x</sup> / All 4 with visible manifestation

Difference 0.619-0.184

The increase of mutation frequency of 3.6 times (Ratio = 3.36) and the regularity of the occurrence of mutations leave no doubt that the majority of new mutations had occurred in mature sperm.--A cytogenetic analysis was carried out for 21 cases and a small deficiency was established for one lethal only; in the other 20 cases no cytological changes were found. Lethal and semilethal mutations occurring in mature sperm do not differ apparently by their structure from the general group of spontaneous mutations which might have occurred at all stages of spermatogenesis. Note that the majority of gene mutations occurring when the sperm is ageing are located on the right side of the chromosome, while spontaneous mutations of the usual control (131 lethals) are irregularly arranged along the entire chromosome length.--The author attaches great importance to the accumulation of mutations in ageing sperm. He considers that this process contributes to the comprehension of the mutation processes occurring in wild populations. At present the author is therefore studying the mutation process in the sperm of hibernating males and in the sperm which is retained in the hibernating females. Males and fertilized females (isolated from each other) were kept at 5-7°C. for several months. Under these conditions the flies remain immobile.

The majority of flies perished, a small number of flies survived and gave F<sub>2</sub> when the CLB method was applied. The following results were obtained.

Table 2

	Number of initial flies	Number of chromosomes investigated	Mutations	
			Number	Percentage
Hibernating from 38 to 50 days	179	2238	9	0.402±0.13
Hibernating 62 days	39	1325	5	0.38 ±0.17
Control	104	5504	9	0.164±0.055

The investigation was carried out on the 80th generation of the forked stock carefully inbred by the author, while in table 1 the data are given which describe the 50th generation of the same stock. Note the general decrease of the frequency of spontaneous mutability which has been found by other investigators analyzing the forked stock.

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A study of the nature of mosaicism in the inversion *scute*<sup>8</sup> of *D. melanogaster*.

In flies carrying the inversion *sc*<sup>8</sup>, as well as other aberrations connected with chromosome breaks in the inert region, a mosaic manifestation of genes may be noted which, owing to aberrations, approaches the inert substance. In this inversion mosaicism for the yellow and achaete genes is noted. Investiga-

tions on mutations caused by X-raying the *sc*<sup>8</sup> chromosome have led the author to the assumption that mosaicism is due to some kind of hereditary change of the *y ac* loci in the soma (Sidorov, 1936). Patterson's point of view that these changes are deficiencies of the entire left area of the chromosome was refuted by a number of facts. In order to prove my point of view it was necessary to follow up the spontaneous changes of the *y ac* loci in the inversion *sc*<sup>8</sup>.--In the progeny of *sc*<sup>8</sup> males crossed with *y sc*<sup>13</sup> females, a great number of lethal *y ac* mutations was found. The mutations arose with a frequency of 1:5,689 chromosomes. The majority of these mutations were caused by crossing-over between the distal region of the inert substance in the *sc*<sup>8</sup> inversion and the short arm of the Y-chromosome. Of the seven *y ac* mutations only one lacked a piece of the short arm of the Y-chromosome on the distal end of the X-chromosome. An additional class of *sc*<sup>13</sup> was discovered. A special experiment was undertaken to obtain an additional crossover class to the "*y ac* mutations" which consists of a Y-chromosome with a left end of the X-chromosome containing the normal

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 Permanent heterozygosity in  
 drosophila.

In the south of USSR, a D. species was found which was named D. obscura-3. The chromosome set of this species contains five pairs of rod-shaped chromosomes and one pair of microchromosomes. --Forty-one stocks were bred from females fertilized in nature and caught in Baku and Sukhumi in 1936, in Sochi,

Sukhumi and Novorossiisk in 1937, in Simferopol and Sochi in 1938. The investigation of the salivary gland chromosomes showed that every stock contained several chromosome aberrations. The aberration set was identical in all the populations.--The following chromosome mutations were found in the Sochi population:

Chromosome	Chromosome mutations	Percentage of stocks heterozygous for various aberrations	Remark
2	Inversion "A"	66	
3	Inversion "B"	45	
4	Complex rearrangement "A"	91	overlapping
5	Complex rearrangement "B"	100	inversion

All the stocks from the other populations proved also to be 100 per cent heterozygous for the complex rearrangement in chromosome 5. In spite of inbreeding carried out during 7-8 generations no homozygous forms were obtained in the overwhelming majority of stocks. However, a cytological study revealed both hetero- and homozygous chromosomes of every pair in the larvae of every given generation.--Direct proofs of the existence of a mechanism of balanced lethals for one of the chromosomes were obtained in the following way. Among the inbred stocks one proved to be homozygous for the chromosome of the fifth pair. The stock was used as analyzer in a cytological study of segregation. One hundred and one males taken from several stocks of D. obscura-3 were inbred for seven generations and then crossed with females from the stock homozygous for the chromosome of the fifth pair. One hundred males were heterozygous and only one homozygous for the chromosome of the fifth pair. It is therefore clear that the overwhelming majority of individuals is heterozygous for one of the chromosomes. It is only exceptionally, possibly through crossing-over, that homozygous individuals occur. The homozygous forms apparently perish mostly during the larval and pupal stages. If this is right, the introduction of a chromosome devoid of lethals from a homozygous stock will disturb the system of balanced lethals and will cause the occurrence of homozygous forms among imago. This hypothesis has been experimentally proved by us.--The above facts point to the conclusion that we are dealing with permanent heterozygotes. At the bottom of this heterozygosity lies a system of balanced lethals which is connected with intrachromosome aberrations, inversions; this points to the difference existing between permanent heterozygotes Drosophila and complex Cenothera heterozygotes. Owing to the

difficulties connected with catching the flies of a new Drosophila species whose populations are not numerous and whose area of distribution is limited, extensive investigations have not been carried out. However, our study of wild populations from different localities has given the same results for 3 consecutive years. The assumption may therefore be made that the peculiarities of multiplication observed by us characterize not only the stock studied, but the entire species.

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A new case of high spontaneous mutability in D. melanogaster.

The first publications on mutable stocks of D. melanogaster appeared in 1937

(Plough and Holthausen, Demerec, Goldschmidt). In 1938, such a stock was found in Portugal by Valadares, and the author discovered, in 1938, a highly mutable stock in a wild population caught in the fall of 1937 in Khotkovo

(60 km. from Moscow).--The problem of mutable stocks in wild populations is of great importance because the existence of these stocks in nature and their possible reoccurrence in natural populations throws new light on the theoretical problems of populations and on the complicated problems of the nature of the mutation process in general. This greatly changes current views as to the possible mutation frequency in natural populations.--This paper gives some results obtained by the author in one mutable stock studied. This mutable stock was bred from 5 females and 4 males caught in nature. Till the fall of 1938 this stock was bred in mass cultures. The frequency of occurrence of certain change in this stock required additional study. The first investigations showed its high mutability. Up to now 50,437 flies have been individually studied; among them 786 individuals were distinctly changed. The aberrant flies may be divided into 108 independent types (Tables 1 and 2).--The data on the sex chromosome are accurate, because only those characters were taken into account which were found in one individual among 60-70 individuals of a culture. But if 2-3 identically changed individuals were found (a group or "bundle") all the three changes were considered as a single change. Almost all these mutations found in the sex chromosome were identical with corresponding stock mutations.--There may be errors in the frequency of occurrence of separate gene mutations in the autosomes because it is sometimes difficult to know the moment when heterozygous recessives arise. However, the frequency given in the table is in no case higher, but rather lower. In usual wild and laboratory stocks of D. melanogaster (Plough and Ives, 1935) and D. funebris (Spencer, 1935) 5 visible mutations as a rule occur per 100,000 flies. The spontaneous frequency of occurrence of visible mutations in our mutable stock therefore is 41 times greater than the usual frequency (without recurrent cases). If the recurrent cases are taken into account, the frequency of occurrence of visibles will obviously be 313 times greater than the usual frequency. This frequency of occurrence is therefore twice as high as that caused by X-rays. In this mutable stock, too, such rare mutations as tetraptera, rotated, aristapedia, etc. were found. Such mutations as lobular wings, wings with an extra radial vein, horn-like protrusions on the sides of the body, eight and four-legged flies, females with sex combs, etc.,--are also possibly quite new. The cause of this high mutability apparently lies in the occurrence of some kind of autosome factor in this stock. This factor of mutability is not attached to the X-chromosome because often yellow males and other mutations occur in crosses in which both X-chromosomes do not come from the mutable stock though the autosomes do. Somatic mutations were often observed. Lethal mutations in the X-chromosome of the male are considerably less frequent than the visible mutations in other chromosomes. It is noteworthy that mutable stocks are apparently widespread (R.L. Berg, Dusseeva and Sokolov have found mutable stocks in D. melanogaster in many places in the Ukraine and Crimea (data unpublished) and all these stocks are apparently of the

same nature because they invariably follow the occurrence of yellow, plexus, abnormal abdomen, etc. A preliminary cytological analysis of the salivary glands of this stock showed that a disturbance in chromosome conjugation, nonhomologous conjugation and chromosome rearrangements (three cases in our preliminary material) are often found. The majority of the changes obtained are real mutations, because of the 103 types of changes found 39 have already given stocks breeding normally, the majority with 100 per cent manifestation. The newly formed stocks also continue to mutate.

Table 1  
Type and Frequency of Occurrence of Mutations in the Sex Chromosome of the Mutable Stock among 50,437 Flies Examined

No.	Character of mutation	Frequency of Occurrence of Mutation		
		♀	♂	Total
1	yellow	---	57	57
2	lozenge	---	8	8
3	forked	---	7	7
4	miniature	---	7	7
5	white	---	4	4
6	outstretched	---	3	3
7	vermillion	---	2	2
8	scute	---	2	2
9	cut	---	2	2
10	Bar	2	---	2
11	Notch	1	---	1
12	Beaded	1	---	1
		4	92	96

Table 2  
Type and Frequency of Occurrence of Autosome Mutations in the Mutable Stock among 50,437 Flies Examined

No.	Type of Mutation	Frequency of Occurrence of Mutation		
		♀	♂	Total
1	black	24	3	27
2	tetraptera	3	4	7
3	brown	2	3	5
4	aristapedia	2	1	3
5	rotated abdomen	4	2	6
6	plexus	3	2	5
7-47	various wing changes	171	122	293
48-53	various eye changes	38	44	82
54-71	various body and head changes	65	50	115
72-83	various bristle changes	51	31	82
84-91	various leg changes	32	33	65
		395	295	786

Zuitin, A.I., Peterhof Biol. Inst. Leningrad University, Leningrad. The changes in the environment as the principal external factor in natural mutations.

During the last ten years, whenever the problem of the environmental factors producing mutations in nature was taken up for consideration, such unusual factors as the radio-active properties of the earth, the cosmic radiation, or the high tropical temperature, were commonly assumed to be the most probable mutation-producing factors. According to these assumptions cultures of *Drosophila* were subjected to exotic experiments: they were taken down to the depths of the earth (Babcock and Collins, 1929; Hanson and Heys, 1930), or they were taken

high up to the skies (Friesen, 1936; Jollos, 1936 and 1937), or they were exposed to the action of high tropical temperature (37°C) for a more or less long time assuming that it is this temperature or some one thereabouts that must have the expected positive effect in increasing the rate of mutations (Goldschmidt, 1929; Jollos, 1930-1933; Plough and Ives, 1933-1935; Timofeeff-Ressovsky, 1935; and others). An attempt was also made to find a relationship between the Van't Hoff rule and changes in the rate of mutations. (Muller, 1928;

Timofeeff-Ressovsky, 1935; and others).--A few years ago a work was undertaken by the author, based on a different assumption, namely that it is the most common and usual variable ecological factors, such as temperature, humidity, etc., incessantly accompanying the entire animal and plant world in its evolution, that are capable of producing mutations when they acquire such extreme, more or less contrasting, values as are sub-lethal for a given population, acting usually in two different directions at the same time--both as a mutation-producing factor and as a factor of natural selection.--Considering the fact that sudden, contrasting changes of temperature are quite widespread throughout the world, not only at present but also in the past ages of the earth's history, the experiments planned were undertaken first of all with the temperature factor, both in laboratory and in natural surroundings.--These experiments gave quite a definite positive answer to the question in consideration, as can be seen from the tables published in three preliminary papers by the author (C.R. Acad. Sci. USSR, 1937 and 1938). In all the experimental sets a high death rate of the developing individuals and a high sterility of survived ones were observed. It was found that temperature itself was not the main factor in the effect produced, but that it was a more or less sudden and contrasting change from a certain usual temperature or thermal regimen to a different unusual one, which brought the population to the limits of its vitality. The high sterility of surviving individuals indicates that the germ cells also are brought into a sub-lethal state by such temperature changes. And one may suppose that the spontaneous mutation in constant external conditions, usual for the individual organism, can be produced by accidental sub-lethal combinations of physiological micro-conditions in a particular mutating cell. No definite relation to the rule of Van't Hoff could be observed in our experiments.--Being conscious of a great deal of responsibility for the conclusions made, the author has carried out one more experiment, as follows. The laboratory 'normal, 112' stock with the mutation rate 0.173% was brought from Leningrad to Sukhumi in the summer of 1938. Here the flies were let out of the vials in an orchard, where they fed on putrefying fruit, and were isolated so that any mixing with the local D. melano-gaster was prevented. During the development of the generation in the experiment the temperature in the fruit substratum varied from 20° to 30°C, while the relative humidity varied from 66 to 100 per cent. On account of a high mortality of the developing flies 620 gametes only could be studied with the ClB method. However, 17 of them were found to contain a lethal or semi-lethal mutation in the X-chromosome tested in F<sub>3</sub>. This makes 2.74%, or sixteen times the percentage observed in the same stock in the control laboratory conditions, the difference in percentage being about 10 times its probable error. This rate of mutation was higher than that of the local race in the same natural conditions, whereas in laboratory conditions the relations are reversed.--The effect of humidity on mutation frequency as a factor closely related to temperature for all the land-dwelling organisms is the object of the author's studies at the present time.