Table 2. Frequency of transmissibility of y mosaics.

	Transmitted	Sterile	Non-transmitted mosaics	Total yellow mosaics
EMS run #1	0	1	2	3
EMS run #2	3	4	1	8
EMS run #3	2**	1	0	3
EMS run #4	1	0	1	2
total	6	6	4	16

**Both lethal--no (y) males.

$\frac{\text{Total transmitted}}{\text{Total (y) mosaics}}$ = (6/16)(100) = 37.5% frequency of transmission.

$\frac{\text{Total transmitted}}{\text{Total fertile (y) mosaics}}$ = (6/10)(100) = 60% frequency of transmission.

the y w f / y^{ems} females do not produce (y) sons, indicating the presence of an independent lethal to the right of white. Of 4 surviving stocks carrying the EMS-induced yellow, one shows a (y^2) phenotype (dark bristles). The others have typical yellow-brown bristle color. Unlike X-rays, which frequently involve the 1J1$^+$, ac$^+$, or sc$^+$ regions, none of the transmitted mutants in this series shows evidence of minute structural rearrangements or multiple involvement of these neighboring genes.

This work was supported by USPHS Biomedical Research Support Grant RF431-H114G.

Mukhina, L.I., V.A. Kulitchkov and I.F. Zhimulev. Institute of Cytology and Genetics, Novosibirsk, 630090, USSR. Distribution of chromosome rearrangement breaks along the polytene chromosomes of D. melanogaster.

Hannah (1951) described two main characteristics of intercalary heterochromatin in D. melanogaster: ectopic pairing and a high frequency of chromosome rearrangement break points. Since then new peculiarities if intercalary heterochromatin have been described: late replication (Arcos-Teran 1972; Zhimulev and Kulitchkov 1977), "weak points" (Zhimulev and Kulitchkov 1977), and strong homologous synapsis (Kulitchkov and Belyaeva 1975; Polyanskaya 1975). After in situ hybridization, bands, having the characteristics of intercalary heterochromatin, preferentially bind labelled nucleic acids, i.e., c-DNA (Rudkin and Tartof 1974), c-RNA (Gvozdev et al. 1980), poly (A^+) RNA (Spredling et al. 1975; Gvozdev et al. 1980) and also some cloned D. melanogaster sequences (Ilyin et al. 1977; Finnegan et al. 1977). In addition, more precise data on the location of the regions of ectopic pairing in the polytene chromosomes (Kaufman and Iddles 1963; Kulitchkov and Zhimulev 1976) and numerous chromosome rearrangements have been published in recent years.

Distribution of chromosome rearrangement breaks along the polytene chromosomes will be described here.

We have chosen to exclude mutations selectively induced in a specific region by investigator and have included only those rearrangements which either were induced in Drosophila genome at random or those found in populations (we classify these provisionally as "spontaneous"). Table 1 lists the origin of the rearrangements analyzed.

Data on the localization of break points in F_1 larvae after mating females with irradiated males (Prokofyeva-Belgovskaya and Khvostova 1939; Kaufman 1946) were also used.

The distribution of breaks is shown in Figs. 1-6. Data on translocations and inversions both naturally occurring and induced are presented separately. In the summary histogram as well as the inversions and translocations, all the remaining aberrations listed in Table 1 are included. For the regions adjacent to the centromere: 20A-F, 40A-41F, 80D-81F, the total number of breaks was divided by the number of letter subdivisions of these regions and mean data are shown in Figs. 1, 2 and 4.

Distribution of the breaks in the X chromosome (Fig. 1F) is clearly non-random. In addition to the centromeric region 20A-F such regions as 1B, 2B, 3C, 7B, 11A, 12E, 16F, 19E show marked peaks as well. All these regions are considered to be intercalary heterochromatin re-

Table 1. List of chromosome rearrangements studied.

Nature of rearrangements	Deficiencies	Duplications	Inversions	Translocations and transpositions	Rearrangements with non-precise locations of 1 break	Breaks in the first generation	References
Spontaneous	28	6	34	1	17		Lindsley, Grell 1968
			52	8			Yamaguchi et al. 1976
			43		8		Stalker 1976
			13		15		Pipkin et al. 1976
			6		2		Dubinen et al. 1940
			6				Zacharopoulou 1974a
			4				Yang et al. 1971
			3				Zacharoupoulou 1947b
			2				Paik et al. 1969
			2				Mukai et al. 1970
	1						Koliantz 1971
			21		4		Ashburner,Lemeunier 1976
			4				Choi 1977
			1				Mettler et al. 1977
			19				Yutaka et al. 1979
			103	4			Yamaguchi et al. 1974
			2				Alahiotis et al. 1977
Totally spontaneous	29	6	315	13	46		
Induced	9		11	309	61		Lindsley, Grell 1968
			8	269	6		Lindsley et al. 1972
			25	60	16		Roberts 1970
				53	1		Stewart, Meriam 1973
	5		7	25	17		Valencia 1970
	1		1	48	1		Mukhina, Zhimulev 1980
				7	17		Mamon et al. 1977
	43				1		Lefevre 1974
			14	4	1		Ashburner 1972
				12	3		Denell et al. 1978
				50			Roberts 1972
	1		20	5	6		Woodruff,Ashburner 1978
						170	Prokofyeva-Belovskaya, Khvostova 1939
						1389	Kaufman 1976
Totally induced	59		86	842	130	1529	
Totally	88	6	401	855	176	1529	

gions. Table 2 shows the correlation coefficients between frequencies of breaks in the regions and the other cytological characteristics of polytene chromosomes. All the coefficients with the exception of the underlined ones are statistically significant ($P \geq 0.05$). It is unlikely that break point frequencies follow the DNA concentration in the region. Although precise estimatations of DNA quantities in the lettered subdivisions have not been done, it can be seen that in the regions 9A, 10A and 10B where very large bands are located there are no peaks of break points. This suggests that higher break frequencies in different regions may have structural significance.

Table 2. Coefficients of correlation between the frequencies of breakage in weak points (wp), ectopic pairing (ep), late replication (lr), strong homologous synapse (ss), preferential binding of c-RNA (cR), poly(A^+) RNA (pR), and chromosomal rearrangements (b).

	ep	lr	cR	pR	b	ss
Chromosome X						
wp	0.77	0.61	0.49	0.48	0.47	0.49
ep		0.65	0.39	0.43	0.55	0.50
lr			0.55	0.58	0.34	0.59
cR				0.77	0.40	0.56
pR					0.36	0.64
b						0.50
Chromosome 2L						
wp	0.72	0.54	--	--	0.21	--
ep		0.62	--	--	0.27	--
lr			--	--	0.24	--
Chromosome 2R						
wp	0.54	0.40	0.33	0.27	0.08	--
ec		0.67	0.53	0.41	0.17	--
lr			0.68	0.67	0.32	--
cR				0.80	0.22	--
pR					0.30	--
Chromosome 3L						
wp	0.83	0.62	--	--	0.24	--
ep		0.59	--	--	0.35	--
lr			--	--	0.20	--
Chromosome 3R						
wp	0.78	0.58	--	--	0.06	--
ep		0.60	--	--	0.16	--
lr			--	--	0.25	--

Data were taken: for weak points, ectopic pairing, late replication from Zhimulev and Kulitchkov (1977), for strong homologous synapsis from Kulitchkov and Belyaeva (1975), for binding of c-RNA and poly(A^+) RNA from Gvozdev et al. (1980).

-- = No data available.

Some differences are rather clear between the distributions in Figs. 1D and 1E. For example, among rearrangements which are maintained in the stocks (Fig. 1D) the highest peak is located in 3C, and there are also high peaks in 11A, 12E and 20AF. As for the F_1 rearrangements (Fig. 1E), the highest peaks are seen in 20AF, 12E, 11A and 2B.

In the other chromosomes the "peaks" are located in the centromeric regions as well as in the regions 26A, 30B, 34A (Fig. 2), 50A, 56F, 59D (Fig. 3), 61F, 64C, 75C (Fig. 4), 101F (Fig. 6) which with the exception of 26A and 34A are considered to be intercalary heterochromatin (Zhimulev and Kulitchkov 1977).

The data examined show once more that chromosome rearrangement break points are distributed non-randomly among the chromosomes and are predominantly located in the regions of intercalary heterochromatin.

References: Alahiotis, S. et al. 1977, DIS 52:106; Arcos-Teran, L. 1972, Chromosoma 37:233; Ashburner, M. 1972, DIS 49:34; Ashburner, M. and F. Lemeunier 1976, Proc. Roy. Soc. Lond. B193: 137; Choi, Y. 1977, DIS 52: 89; Denell, R.E. et al. 1978, Mutat. Res. 49:219; Dubinin et al. 1940, Zhurn. Obsch. Biol. 1:543; Finnegan et al. 1977, Cold Spring Harb. Symp. Quant. Biol. 42:1053. Gvozdev et al. 1980, Chromosoma, in press; Hannah, A. 1951, in: Adv. in Genet. (M. Demerec, ed.); Ilyin, Y.V. et al. 1977, Cold Spring Harb. Symp. Quant. Biol. 42:959; Kaufman, B.P. 1946, J. Exp. Zool. 102:293; Kaufman, B.P. and M.K. Iddles 1963, Portugal Acta Biol. 7:225; Koliantz, G. 1971, DIS 46: 52; Kulitchkov, V.A., and E.S. Belyaeva 1975, Dokl. Acad. Nauk SSSR 221:463; Kulitchkov, V.A. and I.F. Zhimulev 1976, Genetica (USSR) 12(5):81-89.

Lefevre, G. Jr., Cold Spring Harb. Symp. Quant. Biol. 38:591; Lindsley, D.L. and E.H. Grell 1968, Carn. Inst. Wash. Publ. N627; Lindsley, D.L. et al. 1972, Genetics 71:157; Mamon, L.A. et al. 1977, Genetica (USSR) 13:1378; Mettler, L.E. et al. 1977, Genetics 87: 169; Mukai, T. et al. 1970, DIS 45:77; Mukhina, L.I. and I.F. Zhimulev 1980, DIS 55:209; Paik, Y.K. et al. 1969, DIS 44:67; Pipkin, S.B. et al. 1976, J. Hered. 67:258; Polyanskaya, G.G. 1975, Cytology (USSR) 18:693; Prokofyeva-Belgovskaya, A.A. and V.V. Khvostova 1939, Dokl. Acad. Nauk SSSR 23:269; Roberts, P.A. 1970, Genetics 65:429; Roberts, P.A. 1972, Genetics 71:401; Rudkin, G.T. and K.D. Tartof 1974, Cold Spring. Harb. Symp. Quant. Biol. 38:397; Spradling, A. et al. 1975, Cell 4:395; Stalker, H.D. 1976, Genetics 82:323;

Stewart, B. and J.R. Meriam 1973, DIS 50:167; Valencia, R. 1970, DIS 45:37; Woodruff, R.C. and M. Ashburner 1978, Amer. Naturalist 112:456; Yamaguchi, O. et al. 1974, Genetics 78:1209; Yamaguchi, O. et al. 1976, Genetics 83:409; Yang, H. et al. 1971, DIS 47:71; Yutaka, I. et al. 1979, Japan J. Genet. 54:69; Zacharopoulou, A. 1974a, DIS 51:110; Zacharopoulou, A. 1974b, DIS 51:52; Zhimulev, I.F. and V.A. Kulitchkov 1977, Genetica (USSR) 13(1):85.

Fig. 1-6: Distribution of chromosome rearrangement breaks along the X chromosome (Fig. 1), 2L chromosome (Fig. 2), 2R chromosome (Fig. 3), 3L chromosome (Fig. 4), 3R chromosome (Fig. 5) and fourth chromosome (Fig. 6).
Abscissa: chromosome regions according to Bridges' revised maps.
Ordinate: number of breaks in the letter subdivision of the map.

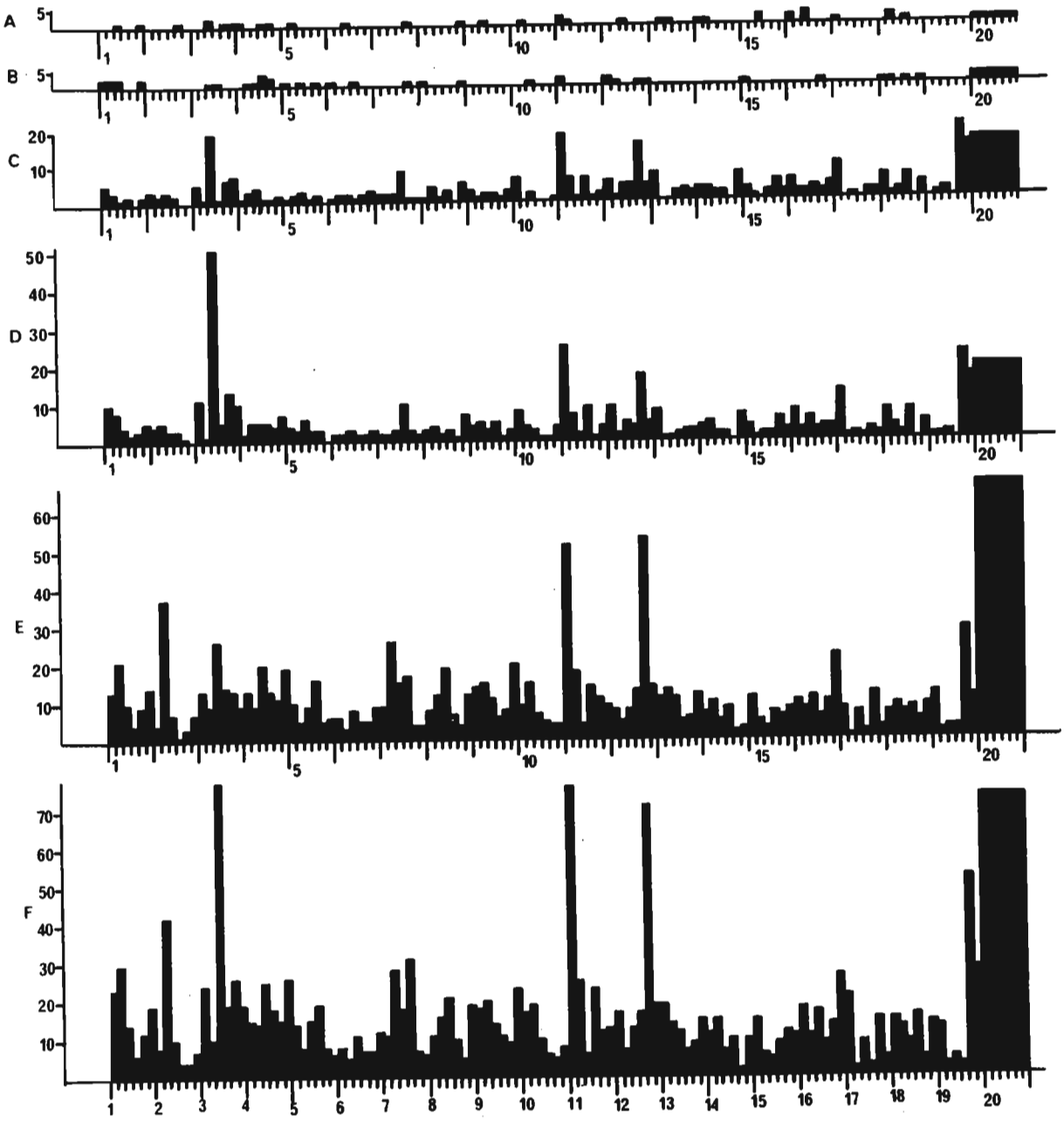

Fig. 1. A=inversions induced, B=inversions spontaneous, C=translocations induced, D=sum of A-C plus deficiencies, duplications and translocations indicated in Table 1. E=sum of the data of Kaufman and Prokofyeva-Belgovskaya and Khvostova, F=sum of D and E.

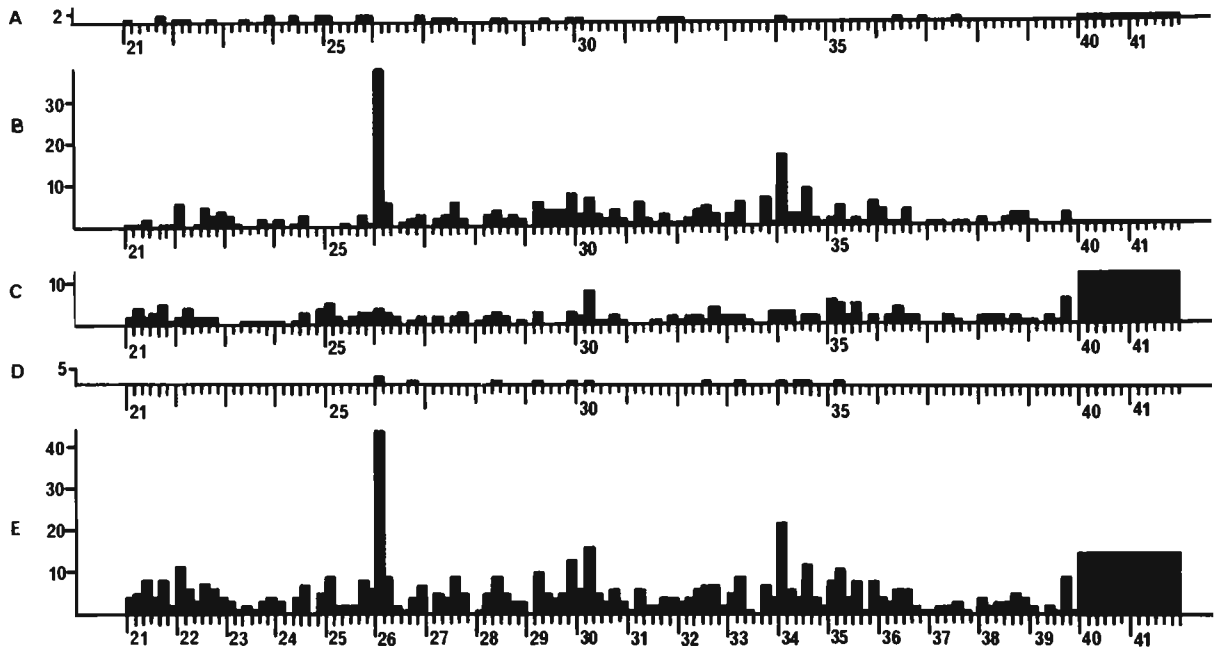

Fig. 2. A=inversions induced, B=inversions spontaneous, C=translocations induced, D=translocations spontaneous, E=sum of A-D plus deficiencies, duplications and transpositions, indicated in Table 1.

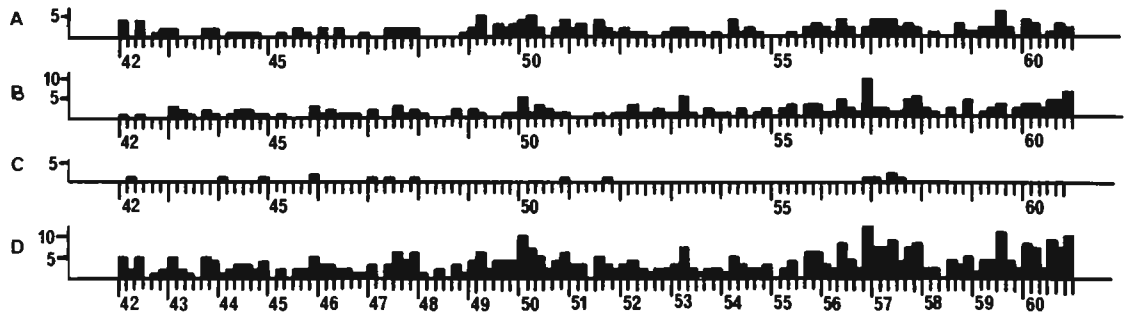

Fig. 3. A=inversions spontaneous, B=translocations induced, C=translocations spontaneous, D=sum of A-C plus deficiencies, duplications and transpositions (Table 1).

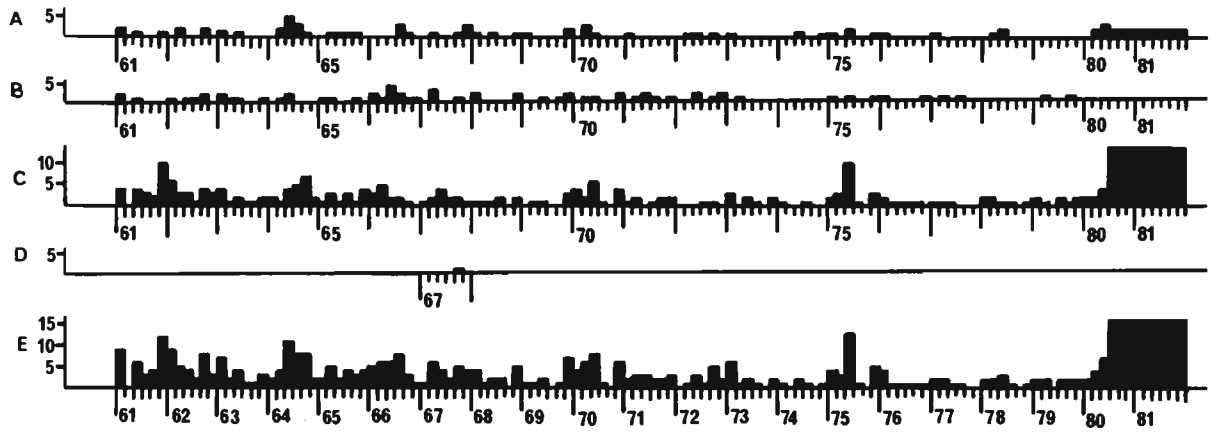

Fig. 4. A=inversions induced, B=inversions spontaneous, C=translocations induced, D=translocations spontaneous, E=sum of A-D plus deficiencies, duplications and transpositions, indicated in Table 1.

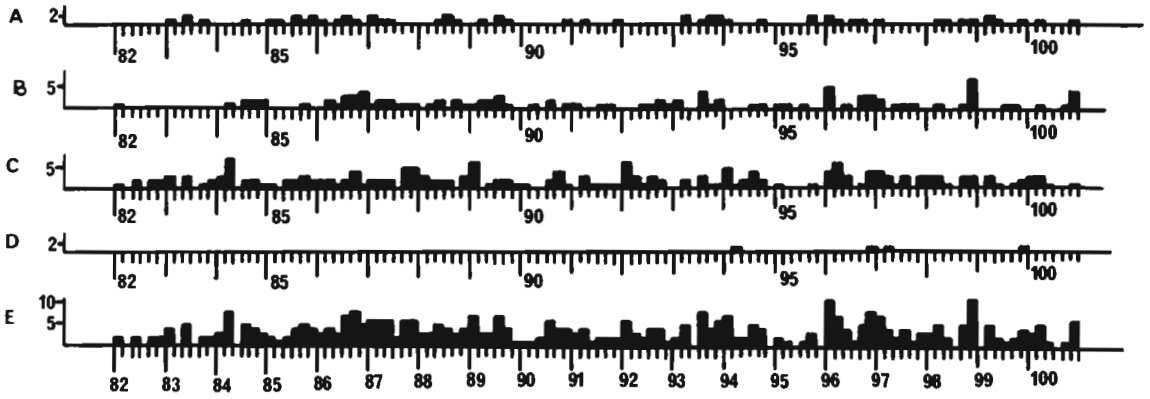

Fig. 5. A=inversions induced, B=inversions spontaneous, C=translocations induced, D=translocations spontaneous, E=sum of A-D plus deficiencies, duplications and transpositions, indicated in Table 1.

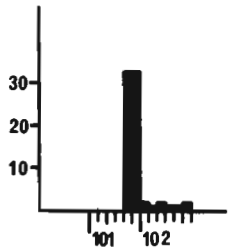

Fig. 6. Sum of all breaks.

Nikoshkov, A.B. and V.T. Kakpakov. Institute of General Genetics, Moscow, USSR.

Dosage compensation of sex-linked genes in established cell lines of D. melanogaster.

Established cultures of Drosophila cells which are homogeneous from caryological point of view and have different ratio between sex chromosomes and autosomes represent an advantageous model for the study of dosage compensation.

We measured the activity of two enzymes, 6-phosphogluconate dehydrogenase (Luccheci and Rawls, Jr. 1973) and fumarase (Pipkin et al. 1977), determined by sex-linked structural genes Pgd (1-0.64) and Fuh (1-19.9) and α-glycerophosphate dehydrogenase (Luccheci and Rawls, Jr. 1973), determined by autosomal structural gene α-Gpdh (2-20.5) in cell cultures with different ratio between sex chromosomes and autosomes (see Table 1).

Table 1. Ratio of fumarase and α-glycerophosphate dehydrogenase activity in cell lines of D. melanogaster.

Cell lines		Passage	Caryotype	X:A	Fumarase activity -glycerophosphate dehydrogenase activity
KcH	(3)	80-100	1X:2A	0.5	0.163 ± 0.008
KcI	(3)	1-10	2X:2A	1.0	0.175 ± 0.011
67jDBS	(4)	180-200	2X:2A	1.0	0.151 ± 0.002
67j25D	(4)	600-620	2X:2A	1.0	0.172 ± 0.006

Identification of the isozymes of all three enzymes was carried out by means of polyacril amide gel electrophoresis. All cultures showed heterozygosity in three enzymes except Kc cell line cells. We could find only two bands of fumarase in heterozygotes.

The ratio of fumarase and α-glycerophosphate activity remains approximate-