relationship suggested that the increased body weight at low temperatures may be due to the slower development of the individual. This view was supported by highly significant differences ($p < 0.001$) in mean body weight among three developmental stages (third instar larvae > pre-emergency pupae > adult) raised at 18°C and 28°C. It was also observed that the rate of development at 18°C was approximately one half the rate at 28°C. The above relationship suggested a possible cline between the body weight and altitude in Mexican populations of D. melanogaster.

Reference: Pipkin et al. 1976, J. Hered. 67:258-266.

Miglani, G.S. and F.R. Ampy. Howard University, Washington, D.C. ADH denaturation depends on native ADH activity levels in D. melanogaster.

Native ADH activity (nM of NADH produced/ml/min/mg of live weight) was determined spectrophotometrically for 12 Adh I/Adh I and 40 Adh II/Adh II isochromosomal lines extracted from 16 Mexican populations of D. melanogaster (Pipkin et al. 1976) raised at 25°C. The native ADH activity was compared with the activity of ADH for each line after treatment with 0.7M guanidine hydrochloride (GuHCl) or 1.0M urea (UR) for 40 seconds or with heat for 15 minutes at 45°C. The relationship between native ADH activity and ADH activity after treatments with denaturants was investigated. A significant correlation ($r = 0.63$; $p < 0.05$) was observed between native ADH activity for the 12 Adh I/Adh I lines and ADH activity after treatment with UR. Significant correlations were observed between native ADH activity for the 40 Adh II/Adh II lines and ADH activity after treatment with GuHCl ($r = 0.59$; $p < 0.01$) and UR ($r = 0.71$; $p < 0.01$). These relationships suggested that the degree of ADH denaturation was possibly dependent on the native ADH activity levels of the strain.

Reference: Pipkin et al. 1976, J. Hered. 67:258-266.

Moss, L.J. and E.A. Carlson. State University of New York, Stony Brook. EMS induced yellow mosaics in D. melanogaster.

Wild type Ore-R males were fed EMS (ethyl methane sulfonate) using an 0.0125M concentration for 24 hours. These males were mated to virgin y w f females and the F_1 flies were observed for mutations of yellow body, white eyes, or forked bristles. Altogether 21 yellow, 5 forked (all mosaic), and 5 white (all mosaic, one of which was an apricot) mutations were found among 9638 F_1 progeny.

The yellow mutations were classified as mosaic or complete in phenotype and then mated to y w f males for a test of transmissibility. Of the 21 yellow mutations, 7 were transmitted, 4 were probably gynandromorphs involving (y f) F_1 mosaic phenotypes with non-white head areas. Of these 4 gynandromorphs, 2 were sterile, and the 2 which were fertile segregated the y w f and Ore R wild type X (along with some f crossovers). Of the 17 yellow phenotypes not due to chromosome loss, 6 were sterile. Of the 11 fertile yellow mutants, 10 were mosaic and 1 was complete. The complete transmitted as did 6 of these 10 mosaics. The transmissibility data for the yellows are shown in Table 1.

Table 1. Frequency of mutation chart.

	yellow phenotypes	F_1 females
EMS run #1	3	1235
EMS run #2	11	5162
EMS run #3	3	854
EMS run #4	4*	2387
total	21	9638

*Includes one yellow complete; all others in runs 1-4 are mosaic.

Total yellow phenotypes = (21/9638)(100) = 0.217% frequency of yellow phenotypes.

Total F_1 females = (17/9638)(100) = 0.176% frequency of yellow mutations.

These results show that EMS induces chromosome loss as well as gene mutations affecting yellow (none of the transmitted viable yellows showed achaete or scute mutations in association with the yellow). One of the two lethals is not associated with an lJl lethal because

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-