

**Gerasimova, T.I. and L.V. Matyunina.** Inst. of General Genetics, USSR Academy of Sciences, 117908 Moscow, USSR. Unstable mutations at the locus yellow induced by the mobile element MDG2.

derivatives is characterized by high locus specificity (Gerasimova 1984b). The locus yellow (0.0; 1AB) is one of the most frequently mutating loci in the "ct<sup>MR2</sup> system".

15 independent yellow mutations have been obtained from the ct<sup>MR2</sup> line and its derivatives. X-chromosomes in 6 y mutants and 2 y<sup>+</sup> revertants were analysed by the method of in situ hybridization with <sup>3</sup>H-DNA of mobile elements MDG1, MDG2, MDG3, MDG4, and copia. The results of hybridization

The unstable line ct<sup>MR2</sup> (Gerasimova 1981) is characterized by "transpositional bursts": mass simultaneous transpositions of different mobile elements in the same germ cell resulting in insertion mutagenesis (Gerasimova et al. 1984a). Insertion mutagenesis spontaneously occurring in the ct<sup>MR2</sup> line and its derivatives

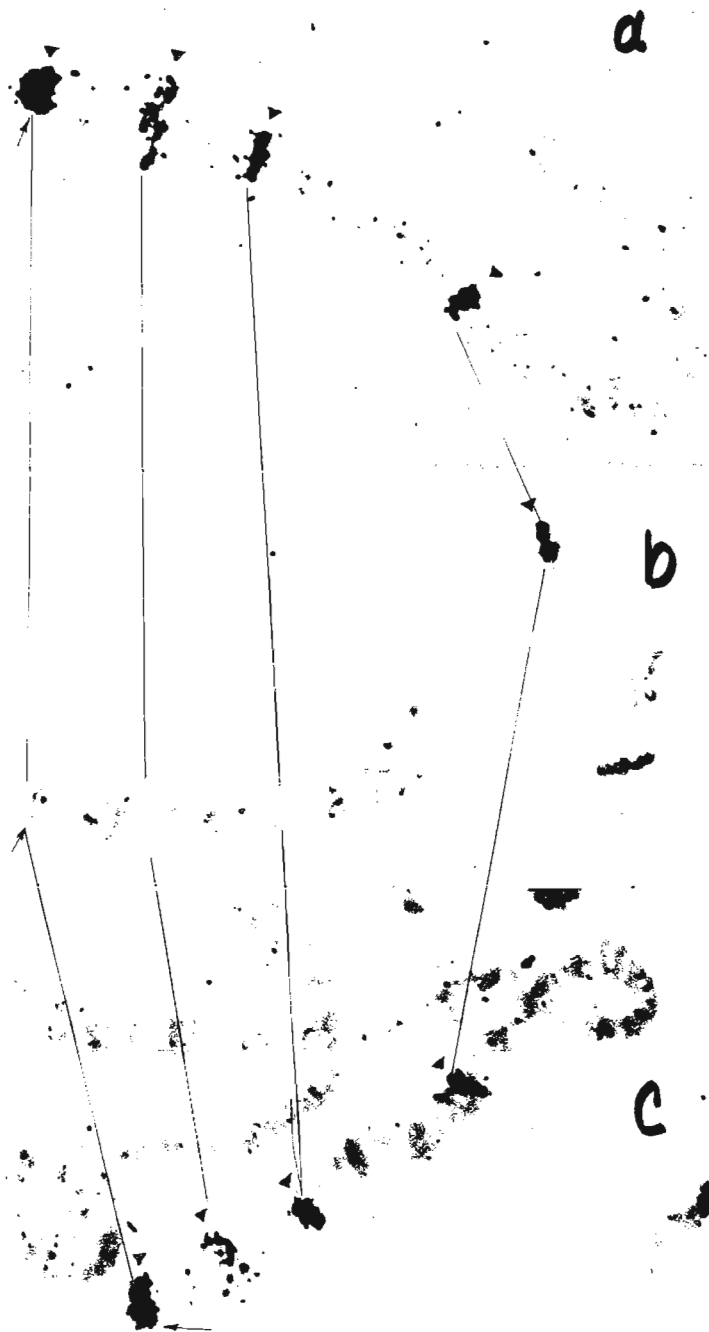
with MDG2 are given in Table 1. In all 6 independent yellow mutations, the site of hybridization for MDG2 was discovered in the 1AB region of the locus yellow (Figure 1). In the two y<sup>+</sup> revertants, no such hybridization site was found. The results obtained show with high probability that mutations at the locus yellow are induced by MDG2.

Among the yellow mutants and their revertants three lines successively obtained from each other were studied in detail: y<sup>MR19</sup><sub>w</sub>MR19<sub>ct</sub>MRpN19 → y<sup>+</sup>w<sup>+</sup>ct<sup>+</sup>snL2 → y<sup>MR19a</sup><sub>w</sub>MR19a<sub>ct</sub>snL2. The initial line y<sup>MR19</sup><sub>w</sub>MR19<sub>ct</sub>MRpN19 contains MDG2 in the 1AB region (two sites, Figure 1). The reversion to y<sup>+</sup>w<sup>+</sup>ct<sup>+</sup>snL2 is accompanied by MDG2 excision from the 1AB region of the locus yellow. In course of repeated mutagenesis in y<sup>MR19a</sup><sub>w</sub>MR19a<sub>ct</sub>snL2, MDG2 returns to this region: 1AB (both sites, Figure 1). Thus, repeated mutagenesis at the locus yellow is also associated with

Table 1. Distribution of MDG2 hybridization sites on X-chromosomes of yellow mutations and their revertants.

Line	MDG2
ct <sup>MR2</sup>	2B, 3E, 4D, 20
y <sup>MR2</sup> ct <sup>+</sup>	1AB, 4D, 10C, 11A, 13F, 20
y <sup>+</sup> ct <sup>MR2</sup>	2B, 4A, 4D, 19E, 20
y <sup>MR7</sup> <sub>w</sub> MR7 <sub>ct</sub> <sup>+</sup>	1AB, 2B, 3A, 5A, 19E, 20
y <sup>MR14</sup> <sub>w</sub> MR10 <sub>ct</sub> <sup>+</sup>	1AB, 2B, 4A, 6F, 19E
y <sup>MR19</sup> <sub>w</sub> MR19 <sub>ct</sub> MRpN19	1AB, 2B, 3A, 5A, 19E, 20
y <sup>+</sup> w <sup>+</sup> ct <sup>+</sup> snL2	5A, 20
y <sup>MR19a</sup> <sub>w</sub> MR19a <sub>ct</sub> snL2	1AB, 2B, 3A, 5A, 20
y <sup>MR19b</sup> <sub>w</sub> MR19b <sub>ct</sub> MRpN19b	1AB, 2B, 3A, 5A, 20

**Figure 1.** The results of in situ hybridization of MDG2 with X-chromosomes of the mutant y<sup>MR19</sup><sub>w</sub>MR19<sub>ct</sub>MRpN19 (a); its revertant y<sup>+</sup>w<sup>+</sup>ct<sup>+</sup>snL2 (b); and back mutant y<sup>MR19a</sup><sub>w</sub>MR19a<sub>ct</sub>snL2 (c). The arrow points out the 1AB region of the locus yellow; the triangles designate the sites of hybridization 1AB, 2B, 3A, 5A.



MDG2. It is of interest that the two other sites of MDG2 hybridization (2B, 3A) located in the X-chromosome of  $y^{MR19_w}MR19_{ct}MRpN19$  line disappear in  $sn$  mutants in the course of reversion and appear again in the course of repeated mutagenesis in  $y^{MR19_w}MR19_{ct+sn}L2$  (Figure 1). A possible cause of such shuttle of transposons may be the existence of transpositional molecular memory, i.e., preservation of short transposon fragments (MDG2 in the given case) in the target site (locus).

Modolell with coworkers (1983) showed earlier that the collection mutation  $y^2$  was induced by MDG4. In our collection of  $y$  mutants no MDG4 transpositions to the locus yellow were found. This indicates that the high specificity of insertion mutations and transpositions of mobile elements strongly depends on the genotype.

**References:** Gerasimova, T.I. 1981, Mol. Gen. Genet. 184:544-547; Gerasimova, T.I., L.J. Mizrokhi & G.P. Georgiev 1984a, Nature 309:714-716; Gerasimova, T.I., L.V. Matyunina, Y.V. Ilyin & G.P. Georgiev 1984b, Mol. Gen. Genet. 194:517-522; Modolell, J., W. Bender & M. Meselson 1983, 80:1673-1682.



**Ghosh, A.K.** University of Calcutta, India.

Transcriptional activity of an autosomal arm (2L) in trisomic condition in *Drosophila melanogaster*.

The principal object of this present investigation is to measure the transcriptional activity of thirty different chromosomal segments of trisomy for the entire left arm of the second chromosome by using  $^3H$ -Uridine autoradiography technique. The dissected

glands were incubated in 500  $\mu Ci/ml$  of  $^3H$ -Uridine (Specific activity 12,700  $\mu Ci/mM$ , BARC, Trombay) and processed for autoradiography as described previously (Lakhotia & Mukherjee 1969). Trisomy for 2L were generated by crosses between stocks C(2L)dp; F(2R)bw and Oregon R<sup>+</sup>.

Results revealed that thirteen out of thirty different chromosomal segments (21A to 35F of 2L) synthesize equal amount of transcripts in both diploid and trisomy for 2L stock, whereas some segments behave in a dose-dependent manner (hyperploid/euploid - 1.5). The remaining segments of trisomy-2L(T2L) show increased or decreased transcriptional activity (see Figure 1).

Thus Devlin's enzymetic study of trisomy for 2L stock and the results of our transcriptional study suggest that at least some segments of the autosomal arm in the trisomic have the ability to maintain the transcriptional activity at the same level as in the disomic. This is in agreement with Devlin's (1982) proposition of autosomal dosage compensation. However, as this is true regardless of the sex, this phenomenon of compensation is different from the haplo-X compensation in *Drosophila* male.

This phenomenon is true also for other major autosomal arms, e.g., 3L.

**References:** Devlin, R.H. et al. 1982, PNAS 79:1200-1204; Lakhotia, S.C. & A.S. Mukherjee 1969, Genet. Res. Camb. 14:137.