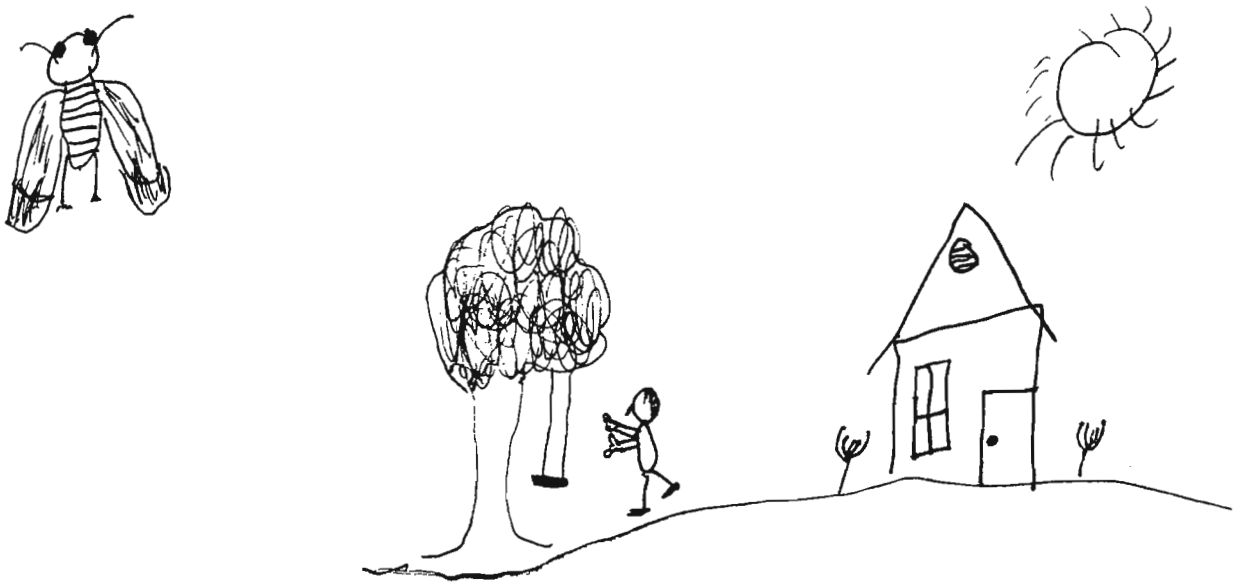
MDG2. It is of interest that the two other sites of MDG2 hybridization (2B, 3A) located in the X-chromosome of $y^{MR19}{}_w{}^{MR19}{}_{ct}{}^{MRpN19}$ line disappear in sn mutants in the course of reversion and appear again in the course of repeated mutagenesis in $y^{MR19a}{}_w{}^{MR19a}{}_{ct^+}{}_{sn}{}^{L2}$ (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.



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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 ^{3}H-Uridine autoradiography technique. The dissected glands were incubated in 500 µCi/ml of ^{3}H-Uridine (Specific activity 12,700 µCi/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^+.

Results revealed that thirteen out of thirty diferent 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.

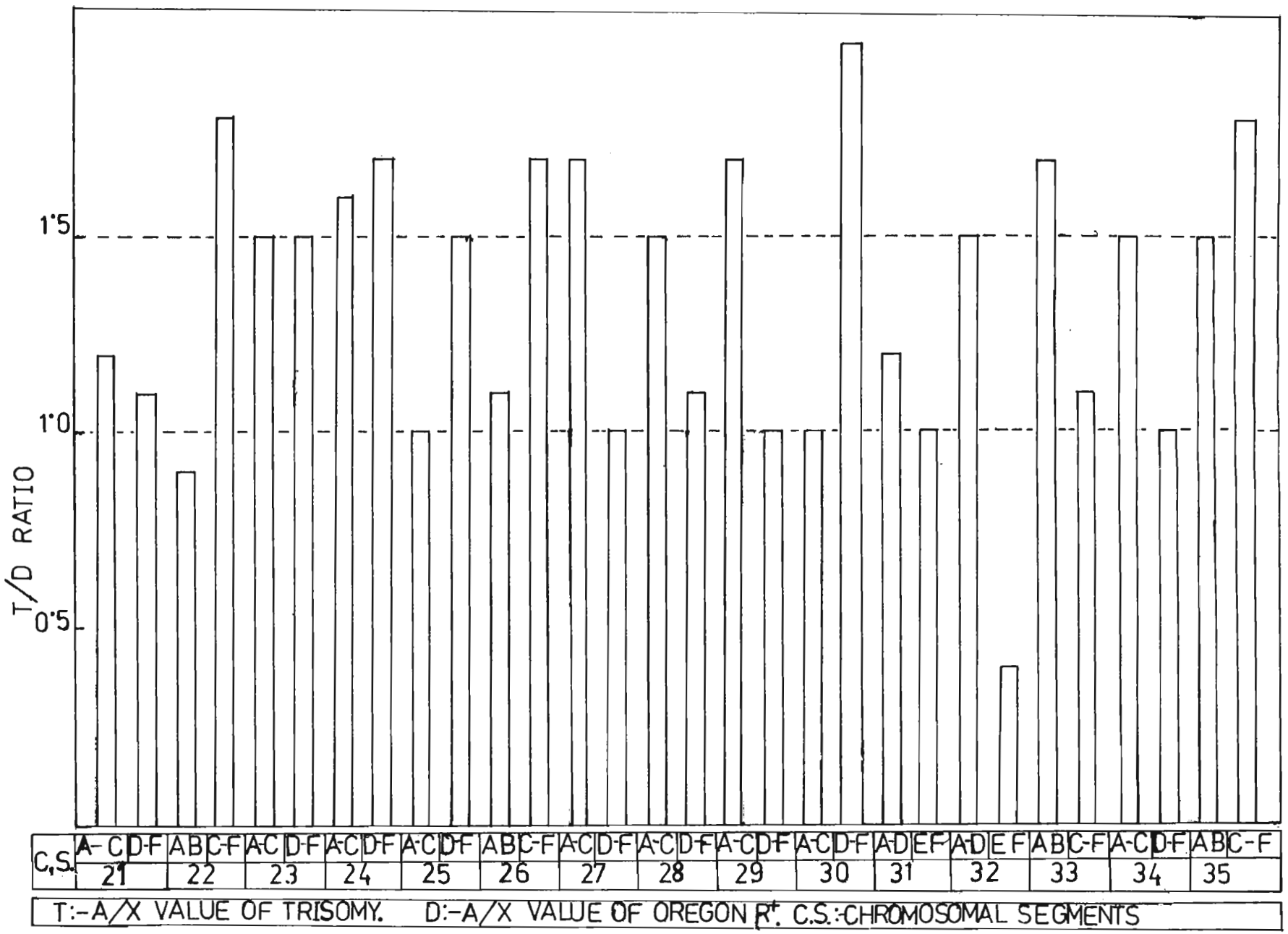

Figure 1. Histogram showing the level of compensation and level of dose-dependence of different sites of the left arm of second chromosome in trisomic condition. The segment 1A to 4F of X chromosome of the same nucleus has been used as reference.