The results are shown in Table 1. There is little consistent allele frequency change nor any evidence of selection in any of the cages. In particular the starch cages do not show any more change than the maltose cages. No evidence of selection has been detected using an experimental system designed to maximize the chance that selection will occur. Results from other experiments have been contradictory. Some have also failed to detect selection involving amylase (Yardley et al. 1977, Powell & Amato 1984) while others have seen selection (Anderson et al. 1979, Powell & Andjelkovic 1983). Taken together, these experiments emphasize how complex selection can be and show that the question of whether variation is selected or neutral may have no easy answers.

References: Anderson, Salceda & Turner 1979, Genetics 91:s2; Powell & Amato 1984, Genetics 106:625-629; Powell & Andjelković 1983, Genetics 103:675-689; Yardley, Anderson & Schaffer 1977, Genetics 87:357-369.

Semeshin, V.F.\* and J. Szidonya.\* \*Inst. of Cytology & Genetics, Siberian Branch, USSR Acad. of Sciences, Novosibirsk 630090 USSR; \*Inst. of Genetics, Biological Res. Ctr., Hung. Acad. of Sciences, H-6701, P.O.B. 521, Szeged, Hungary. EM mapping of rearrangements in the 24-25 sections of D.melanogaster 2L chromosomes.

Three series of chromosome rearrangements designated as dphx, sc 19-x and clhx were induced by X-ray irradiation (4000 R) of Oregon R and T(1;2)sc 19 males and then isolated by means of Dp(2;2)B3 de dp cl duplication; the first two series as well as Df(2L)M11 and Df(2L)M-z<sup>B</sup> were balanced over Dp(2;1)B19 (Reuter & Szidonya 1983; a detailed description rearrangements and induction and their genetic analysis will be given elsewhere). Heterozygotes for

Batumi L wild stock were used in electron microscopic (EM) mapping of deficiencies. Techniques of squashed chromosome preparations for EM have been described earlier (Semeshin et al. 1979). Revised Bridges' maps (Lindsley & Grell 1968) and EM data obtained on Batumi L were used for determination of rearrangement limits.

In the 24AF region Bridges described 30 bands including 8 doublets (Figure 1a). Some of the doublets, such as 24A1-2, D1-2, D5-6, F1-2, F6-7 and the 24C9, D3 and F3 thin bands were described by Saura (1980). However, in our EM studies all the doublets are visible as single bands; the 24B3, C6, C9, D3, F3, F5 and F8 thin bands were not detected (Figure 1b-e). Contradicting to Saura's data we could not visualize 4 bands in the 24F3-8 interval. In this region there are 2 zones with the diffused, slightly puffing material which are adjacent to the 24F1-2 and 25A1-4 thick bands (Figure 1f) and actively incorporates <sup>3</sup>H-uridine (Zhimulev & Belyaeva 1974). We observed the 24E2' thin band which was not described earlier (Figure 1e,f,g). Altogether, 24 bands were found in the 24 section of 2L chromosome.

The 25AC region was mapped earlier (Semeshin et al. 1985). As for the EM map of the 25DF region, it coincides with that of Bridges as well as with Saura's data, if Bridges' doublets are considered as single bands; the 25D1-2 "doublet" consisting of two separate bands is an exception (Figure 1f). The complete EM map of the 24 and 25 sections of the 2L chromosome is presented in Figure 6.

EM map of the 24 and 25 sections of the 2L chromosome is presented in Figure 6.

Df(2L)dp<sup>h19</sup>. As is seen from Figure 1g the deficiency presumably deletes the 24F1-2 to 24F6-7 region. We also can not exclude the possibility that the proximal and distal breakpoints are located in the left edge of 25A1-4 complex and in the 24F1-2 band, respectively, and the remaining parts of these bands are fused.

Df(2L)M-zB. According to Bridges' data this deficiency deletes the 24E2-F1 to 25A1-2 region (Lindsley & Grell 1968). However, Duttagupta & Dutta Roy (1984) consider that the 25A1-2 band remains intact and is not affected by the deficiency. EM data clearly show that the Df(2L)M-zB deletes the 24E1-2 to 24F6-7 region (Figure 1h), but its proximal breakpoint can not be determined accurately. Because of the underreplication and the break in normal chromosomes the appearance of the 25A1-4 complex is rather variable (Figure 1b-f; see also Zhimulev et al. 1982) which often looks like a wedge. Similar pictures are observed in the chromosomes with deficiency (Figure 1h). However, we can not exclude that the left part of the 25A1-4 complex is removed and therefore attribute it to the uncertainty of mapping.

Df(2L)dph28 deletes the 24D8 to 24F6-7 region (Figure 2a). We are not sure whether the 24D7 thin band is retained and the part of the 25A1-4 band is deleted; this material is attributed to the uncertainty of mapping.

Df(2L)M11 clearly deletes a part of the 25A1-4 complex because of the different thickness of the band in normal and deleted homologues (Figure 2b). The distal breakpoint of the deficiency is located just to the right of the 24D5-6 band (Figure 2c). Thus, the 24D8 to 24F6-7 region and the left part of the 25A1-4 band are included in the deleted material.

Df(2L)ed dp<sup>h1</sup>. This deficiency also removes the left part of the 25A1-4 band (Figure 2d). Figure 2e shows clearly that the 24C3 band is preserved. Therefore, the deficiency deletes the region from the 24C5 band to the left part of the 25A1-4 complex. The 24C4 thin band is attributed to the uncertainty of mapping.

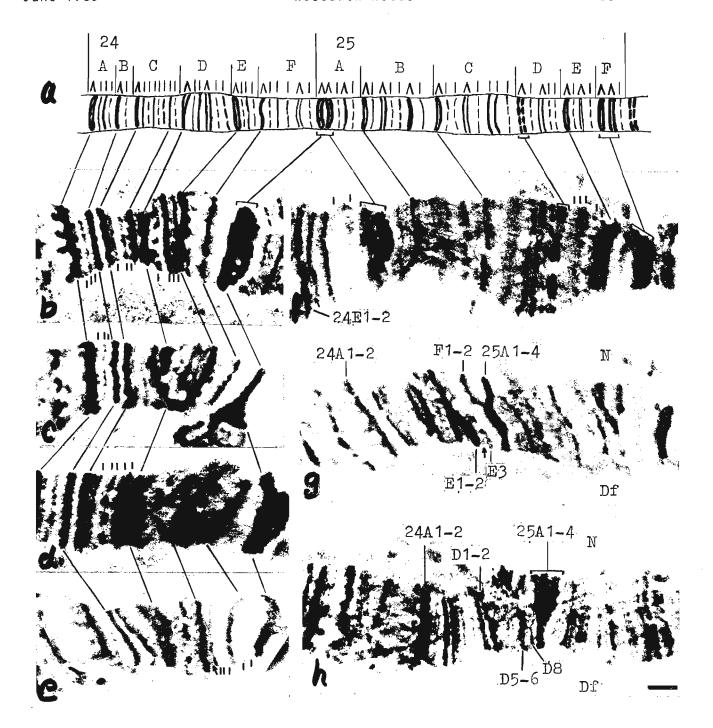


Figure 1. Banding pattern in the 24 (b-e) and 25 (f) sections of the 2L chromosome in the Batumi L stock and in heterozygotes for  $Df(2L)dp^{h19}/+$  (g) and  $Df(2L)M-z^B/+$  (h) deficiencies; (a) Bridges' map; (b,c,d,e) thin bands in the 24D, 24A, 24BC, 24EF subdivisions, respectively; the arrow (e,f,g) points to the new band 24E2'. The bar corresponds to  $1\mu$ .

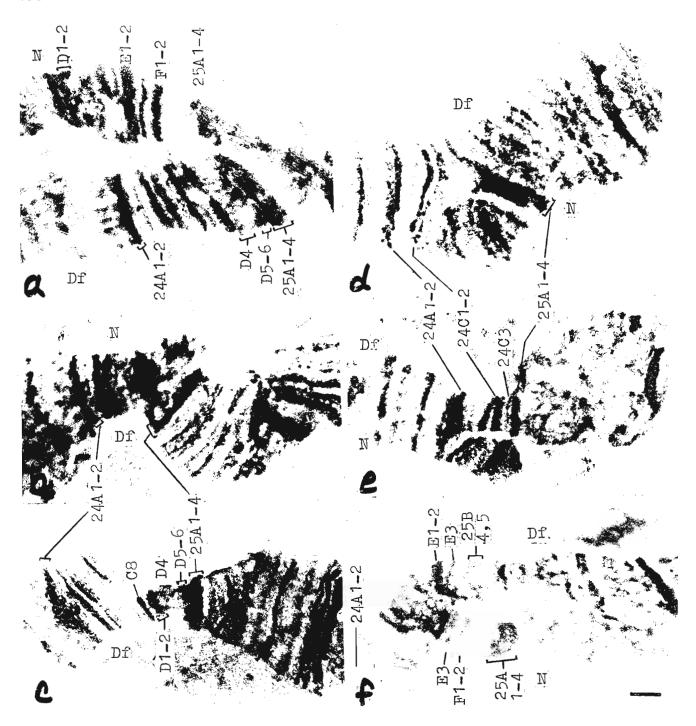


Figure 2. Deficiencies in the 24-25 regions of the 2L chromosome: (a)  $Df(2L)dp^{h28}/+$ ; (b,c) Df(2L)M11/+; (d,e)  $Df(2L)dp^{h28}/+$ ; (f)  $Df(2L)dp^{h28}/+$ .

Df(2L)dph25. The EM pictures may be interpreted in two ways: the deficiency deletes either the 24E4-25B2 region or 24F1-2 to 25B3' one (Figure 2f). The 24E4,5 and 25B3,3' bands are very thin and morphologically similar. So, it is impossible to determine exactly which pair of the bands is preserved.

Df(2L)sc19-3. The deficiency deletes the 24E3 to 25A6-7 region. It is observed both in the

Df(2L)sc<sup>19-3</sup>. The deficiency deletes the 24E3 to 25A6-7 region. It is observed both in the heterozygote (Figure 3a) and in homozygote balanced over a duplication of deleted region (Figure 3b). The 24E2' and 25A8,8' thin bands are not identified on the EM photographs and so attributed to the uncertainty of mapping.

Df



Figure 4 [previous page]. Deficiencies (a-c) and duplication (f,g) of the 24-25 region: (a)  $Df(2L)sc^{19-5}/+$ ; (b) homologue with the same deficiency as (a) is slightly stretched; (c)  $Df(2L)c1^{h2}/+$ ; (d)  $Df(2L)c1^{h1}/+$ ; (e)  $Df(2L)c1^{h4}/+$ ; (f) and (g) heteroand homozygote for duplication Dp(2;1)B19, respectively.

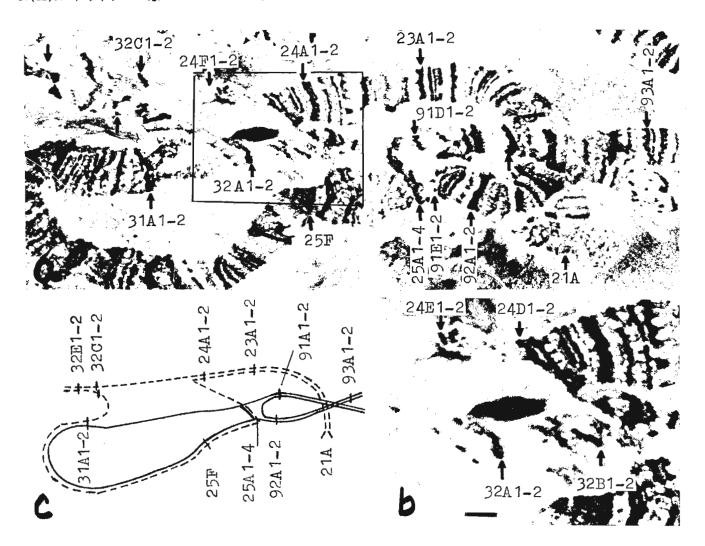


Figure 5. Translocation T(2;3)dph27/+ (a); (b) magnified fragment from (a) showing breakpoints in the 2L chromosome; (c) scheme of translocation: Dotted line - 2L; solid line - 3R chromosome.

Df(2L)sc<sup>19-1</sup>. The deficiency deletes either the 24D5-6 to 25C8 or the 24D8-25C9 region (Figure 3c). The 24D5-6 and 25C9 bands are morphologically very similar large singlets, so it is impossible to determine which of them is deleted.

Df(2L)sc 19-6. Three interpretations of the EM pictures of this deficiency (Figure 3d) are possible:

(1) either the 24F1-2 to 25B10 region is deleted; (2) the 24F4 to 25C1-2 region is removed; or (3) the deletion covers the region from the middle part of the 24F1-2 band to middle part of the 25C1-2 band with remaining parts of both of the band fused. These variants can not be distinguished cytologically.

Df(2L)sc 19-4 deletes either the 25A5 to 25E4-5 region or the 25A6-25E6 one (Figure 3e). The 25A5

and 25E6 bands are very thin and were not distinguished in EM picutres.

Df(2L)sc<sup>19-5</sup>. The deficiency probably deletes the 25A5-25D6 region (Figure 4a); the 25D7 thin band is preserved (Figure 4b); there are no signs of the presence of the 25A5 and D6 thin bands.

Df(2L)cl<sup>F2</sup> deletes the 25D6 to 25E4-5 region (Figure 4c). We attribute the 25D6,7 and 25E6 thin bands to the uncertainty of mapping.

Df(2L)cl<sup>h1</sup> deletes bands in the 25D4-5 to 25F1-2 interval (Figure 4d).

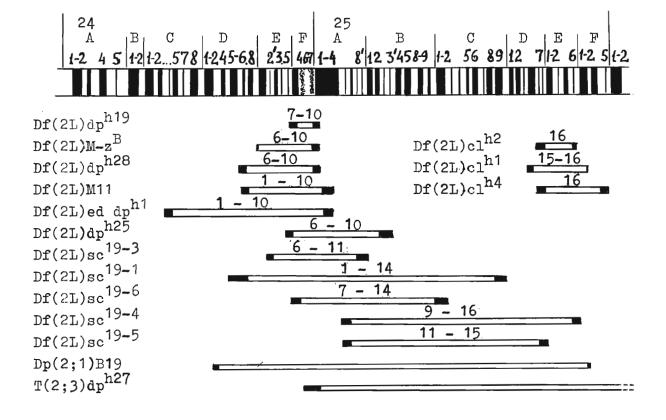


Figure 6. EM map of the 24 and 25 sections of the 2L chromosome and cytological limits of the chromosome rearrangements analyzed. Shaded areas represent the uncertainty of mapping. Numbers indicate mutations (complementation groups) localized in the deleted regions according to Szidonya's data (unpubl.): 1 - G-ft complex; 2 - Sz5-1; 3 - Sz49-1; 4 - ed; 5 - M(2)LS2; 6 - dw-24F; 7 - Sz3-1; 8 - h26-1; 9 - dp-complex; 10 - M(2)z; 11 - Sz64-1; 12 - Sz59-1; 13 - DTS; 14 - M(2)S1; 15 - tkv; 16 - c1.

Df(2L)cl<sup>h4</sup> deletes the 25D6 to 25F3-4 region (Figure 4e). The 25D6,7 and 25F5 thin bands we attributed to the uncertainty of mapping.

**Dp(2;1)B19.** The duplication limits are 9B14-15/25F1-2 to 24D4/9C1-2 (Figure 4g). Figure 4f shows that the 9C1-2 band is located to the right of the duplication.

T(2;3)dp<sup>h27</sup> was induced together with the series of deficiencies dp<sup>hx</sup>. According to the EM data (Figure 5a-c), the breakpoints of translocation are: 91D4-5/24F4-7 to 32B1-2/91E1-2. We also asssume that the break may be located in the left part of the 25A1-4 band.

Figure 6 summarizes the data of EM mapping. The genetic analysis of the deficiences described permitted to find 10 complementation groups to the left of the 25A1-4 complex (Szidonya & Reuter, unpubl.) which are included in the Df(2L)M11 (the 24D8 to 25A1-4 region). The comparison of these data with cytological ones leads to the conclusion that 4 complementation groups, Sz3-1, h261, dp and M(2)z, are located in the 24F1-2 to 25A1-4 interval; the remaining 6 groups are situated in the 24D7-24E5 interval. In the 25 section the tkv mutation can be localized in the 25D4-5 band and cl in the 25E1-5 region.

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References: Duttagupta, A. & A. Dutta Roy 1984, DIS 60:92; Lindsley, D.L. & E.H. Grell 1968, in:Genetic variations of Drosophila melanogaster; Reuter, G. & J. Szidonya 1983, Chromosoma 88:277-285; Saura, A.O. 1980, Hereditas 93:295-309; Semeshin, V.F., E.M. Baricheva, E.S. Belyaeva & I.F. Zhimulev 1985, Chromosoma, in press; Semeshin, V.F., I.F. Zhimulev & E.S. Belyaeva 1979, Genetics (USSR) 15:1784-1792; Zhimulev, I.F. & E.S. Belyaeva 1974, Genetics (USSR) 10:71-78; Zhimulev, I.F., V.F. Semeshin, V.A. Kulichkov & E.S. Belyaeva 1982, Chromosoma 87:197-228.