

**Furman, D.P., and T.A. Bukharina.** Institute of Cytology and Genetics, Siberian Department of the Russian Academy of Sciences, Novosibirsk 630090, Russia. Transposable elements reveal crossing-over.

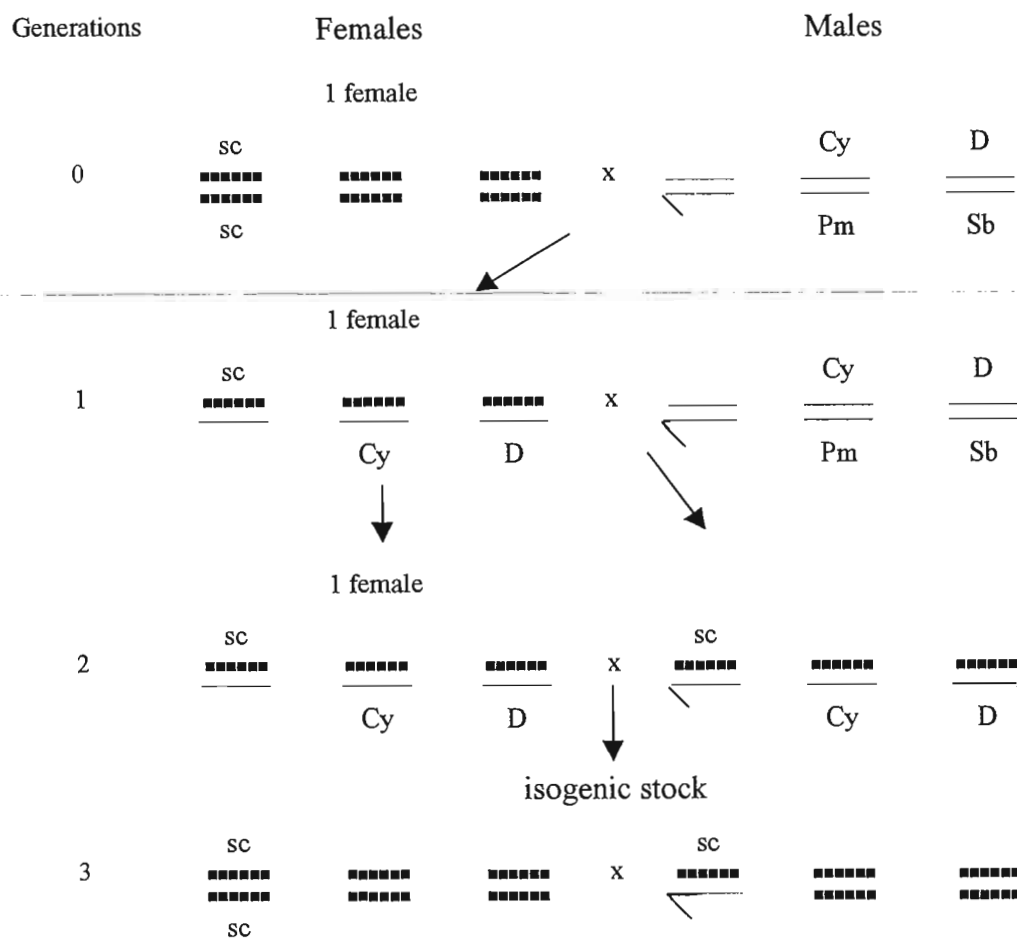
Balancing chromosomes are widely used in the genetic experiments which require preservation of the genetic constitution of homologous chromosomes, in particular, in developing isogenic lines. When coupled with the standard sequence homologues, inverted balancers are believed to suppress recombination

effectively; the homologues should therefore remain intact (MacIntyre and Wright, 1966).

Meanwhile, events of exchange in such heterozygotes as these take place and viable crossover gametes occur either by single exchange in inversion-free regions or by double exchange in inverted regions. Visible mutations may not help identify these events, but transposable genetic elements (TEs), which appear to be more sensitive indicators, perhaps can do that. By comparing the location of transposable elements in the balanced, balancing, and derived lines, it is possible to answer the question as to whether there is an exchange between the balancer chromosomes and their normal homologues. The interchanged chromosomal regions will show up clearly: the labeling sites either appear on or disappear from the chromosomes of the derivative, which is free from inverted balancing chromosomes.

*Cy/Pm;D/Sb*, a balancer line from the collection of the SD RAS Institute of Cytology and Genetics, contains multiple inversions, lethal and visible mutations (Lindsley and Zimm, 1990). Our interest was to see whether recombination is absolutely suppressed in between-line crosses with *Cy/Pm;D/Sb*.

Isogenic lines are perhaps the best we can use in this crossing-over study, because no within-line polymorphism for the location of transposable elements has been observed in them as yet (Furman *et al.*, 1993). *Mdg1*, *Dm412*, *copia*, and *B104* served as markers. Their location was determined by *in situ* hybridization on salivary gland chromosomes; not less than 4-5 preparations with not less than 10 nuclei on each were assayed. The mating scheme was a routine (Scheme 1). Analysis covered the balancing, balanced, and derived isogenic lines.



Scheme 1. Mating scheme for developing the isogenic line. Crosses 1 and 2 with the same female.

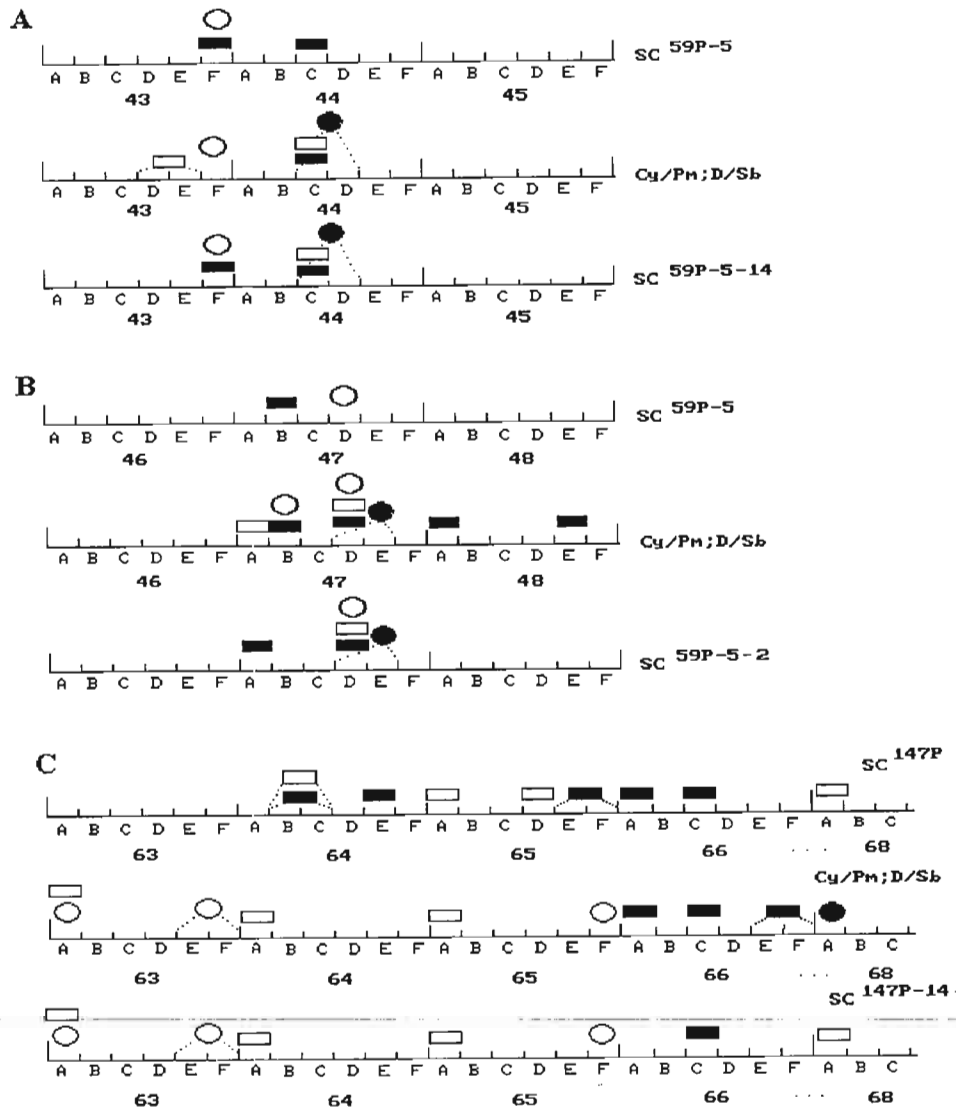


Figure 1 a-c. Examples of chromosomal exchange between *Cy/Pm;D/Sb* (balancing line) and homologues from *sc59-5* and *sc147*: black ovals - *Mdg1*, white ovals - *Dm412*, black rectangles - *copia*, white rectangles - *B104*.

Exchange between the balanced and balancing chromosomes was assumed if a set of transposable elements was found to occupy either the same site in an isogenic derivative and the balancing line or the same sites over a more or less extending region of the respective chromosomes in the isogenic derivative and the balancing line.

The presence/absence of a single transposable element at/from a separate site of the derivative line was not looked at as evidence for exchange. Phenomenologically, either looks like insertion/excision, and therefore may have been preceded by crossing-over, but not necessarily.

The most convincing examples of chromosome exchange are presented in Figure 1a-c. Figure 1a-b depicts fragments of chromosomes from *Cy/Pm;D/Sb*, *sc59-5*, and its two isogenic derivatives, *sc59-5-14* and *sc59-5-2*. Region 44C, labeled by three elements inserted in *sc59-5-14* (Figure 1a), and region 47DE with four inserts in *sc59-5-2* (Figure 1b), are from the balancer. Because no increase in the mortality rate was observed with the *sc59-5* × *Cy/Pm;D/Sb* offspring, double exchange can be assumed within the inverted regions.

The six inserts running in succession on chromosome 3L of *sc147-14* (Figure 1c) might be resulting from crossing-over (site 66A in *Cy/Pm;D/Sb* is polymorphic and hence our explanation is still consistent). The region in question lies off In(3L)69D3-E1;70C13-D1.

Therefore, it would be good to know how the transposable elements are distributed in the balancing line and balanced lines. It would be then easy to foresee crossing-over effects on the polymorphism in derivatives and to estimate correctly the frequencies of TE transpositions in between-line crosses of that sort. This could be of especial importance when TEs alone are markers of genomic regions in experiments of populational genetics, for example, in studying correlations between the pattern of TE distributions and the profile of alteration of polygenic traits.

Our data, however, are insufficient to say by which exact mechanism crossing-over takes place here. For example, recombination frequency may be dependent on whether inserts, especially multiple ones, are present at the same sites of the homologous chromosomes of the lines being crossed. Constructions of that kind either make chromosomes easily breakable and repairable at the homologous regions of TEs or allow exchange at long terminal repeats of TEs (McGinnis and Beckendorf, 1983; Goldberg *et al.*, 1983; Davis *et al.*, 1987; Tsubota *et al.*, 1989).

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References: Davis, P.S., *et al.*, 1987, Proc. Nat. Acad. Sci. 84:174-178; Furman, D.P., *et al.*, 1993, Genetika (Russ.) 29:6; Goldberg, M.L., *et al.*, 1983, Proc. Nat. Acad. Sci. USA 80:16; Lindsley, D.L. and G.G. Zimm 1990, Dros. Inf. Serv. 68; MacIntyre, R.J., and T.R.F. Wright 1966, Dros. Inf. Serv. 41:141; McGinnis, W., and S.K. Beckendorf 1983, Nucl. Acids Res. 11:3; Tsubota, S.I., *et al.*, 1989, Genetics 122:881-890.

**Jarman, M.G., and J.I. Collett.** School of Biological Sciences, University of Sussex, Brighton, Sussex, BN1 9QG, U.K. Siting the genes of *Dipeptidase-B* (*Dip-B*), several 'recessive-lethal' complementation groups and the breakpoints of chromosomal deficiencies within the region 87F12-88B1 of *D. melanogaster*.

This analysis was undertaken to establish the chromosomal site of *Dip-B* and to assess the possibility of association of mutant alleles of the complementation groups of *l(3)88Aa* and *Ac*, isolated by Mortin *et al.* (1992), with the expression of the *Dip-B* gene. At the outset of this analysis the status of the mapping of *Dip-B* was as follows. Electrophoretic analysis of heterozygotes of electrophoretic variants of *Dip-B* in *D.*

*pseudobscura* (Collett, unpublished) had indicated a homotetrameric structure and Onishi and Voelker (1981) had shown a map position within the region 87F12-88C1 of Df(3R)red-31. Subsequent aneuploid mapping of about 80% of the genome by Hall (1983) confirmed the position of *Dip-B* at this site and failed to reveal any other affecting *Dip-B* expression. Following the screen for recessive lethal mutations in the region of Df(3R)P52 (88A3-88B3), Mortin *et al.* (1992) assigned mutations to a number of complementation groups throughout the region including those of *l(3)88Aa* and *Ac*. Several mutant alleles of these two genes had shown some sign of affecting *Dip-B* activity in heterozygotes. Here, following definition of deficiency breakpoints, *Dip-B*, three previously identified genes and at least one new gene are assigned to sites throughout the region, and the identity of one of these genes found by both mutation and transposon insertion is established.

Compiled in Table 1 are the chromosomal deficiencies used here, the methods and sources of identification of their breakpoints and the results of complementation analysis to confirm, to define and to refine the positions of their breakpoints. All deficiencies and mutant alleles were maintained in stocks with either the 'balancer' TM3 or TM6B. Included among these are two, M36 and M49, which were thought to be small deficiencies by Mortin *et al.* (1992) on the basis of the complementation pattern. This expectation was confirmed here, as indicated below, and thus their inclusion in Table 1. The alleles designated M and Z (Figure 1) were isolated by Mortin *et al.* (1992) and supplied by M. Mortin. The allele *k43* of *l(3)88Ab* was supplied by A. Shearn and the transposon induced allele of *l(3)03477*, *j1D1*, was supplied by the *Drosophila* Genome Center (Baltimore).

Complementation was deemed to have failed when no unmarked flies were found among at least 100 progeny from more than one cross, or, in several cases, when results in smaller progenies were consistent with the progenies of similar crosses of flies with other deficiencies or with other alleles within a complementation group (Jarman, 1997). Among the M and Z mutants, each was complementation-tested in pairwise matings. *k43* was tested with each M allele, and similarly, the allele of *l(3)03477* was tested with both M and Z mutants. These alleles are listed in Figure 1 in complementation groups according to the results of these tests. Those listed are consistent with Mortin *et al.*'s (1992) assessment with the exception of the mutant M30. This was found to be in the region of Df(3R)P1. But, in addition, these analyses revealed allelism between the M mutants of *l(3)88Aa* and the P-transposon induced allele *j1D1*. Thus the