

a short deficiency,  $Df(1)N12$ , that extends from  $11D1-2$  to  $11F1-2$ .

By crossing over,  $N$  alleles, including  $fa^8$  and  $spl$ , were combined with  $g-1$ . Males carrying both  $fa^8$  and  $g-1$  show an exaggerated phenotype, have difficulty in eclosion, and usually survive only briefly. The combination with  $spl$  has a somewhat less drastic effect. Males with  $N^{264-40}$  (cytologically normal),  $g-1$ , and  $Dp\ w^{+51b7}$  exhibit simply the  $g-1$  phenotype. Two other euchromatically located  $N^+$  duplications interact with  $g-1$  exactly as does  $Dp\ w^{+51b7}$ ; however, three heterochromatically located  $N^+$  duplications, including  $w^+Y$ , produce a noticeably less effective suppression of the  $g-1$  phenotype.

Although we can rule out the origin of  $g-1$  as resulting from the transposition of part or all of the  $N^+$  locus from  $3C7$  to  $11D9-10$ , we can not yet decide whether the  $g-1$  locus is a persisting duplicate locus once identical with  $N$ , or is an unrelated locus whose altered product now interacts or competes with the product of the  $N$  locus. If regulatory genes in *Drosophila* can occur at a distance from their subject loci, in contradiction to the Crick (1971) model, then the  $g-1$  locus might be a regulator of the  $N$  locus, or vice versa.

To the best of our knowledge, this is the only case, except for *zeste* and *white*, in which the presence of an extra dose of one locus modifies the expression of a mutant at another, distantly located locus.

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This effect is usually described, following Bridges, as a decrease of recombination with increasing age during the first ten or so days of a female's egg laying (later on followed by an increase and another decrease). Deviations

from this pattern are of course known to all recombination workers. One such deviation, which has never been expressly described in the literature, concerns the X chromosome. One sometimes sees authors express surprise that maternal age does not influence recombination frequency in the region around vermilion ( $v$ : 1 - 33.0) which is in the middle of both genetical and cytological maps. However, maternal age effect in X consists of two components: an increase of recombination with increasing age distally and a decrease with increasing age proximally. The  $v$  region lies where these counteracting effects take out each other so that no maternal age effect is observed. This phenomenon is illustrated in Figure 1, where the linear regressions of recombination on maternal age for different X chromosome regions are shown.

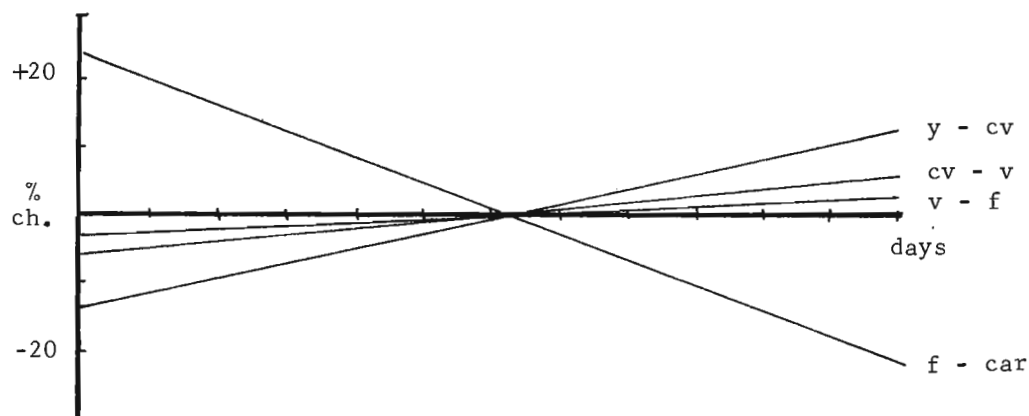


Figure 1. Linear regressions of the entity  $[(\text{observed rec. in one brood} - \text{mean rec.}) / \text{mean rec.}]$  on maternal age for different regions of X. Material from Bateman and Chandley (1965), Roberts (1962), Ting and Walker (1969) and Valentin (1969). Y axis  $100 [(\text{Obs.} - \bar{x}) / \bar{x}]$ ; X axis, maternal age in days.

Recombination is expressed as percentual difference from overall mean, i.e.,  $100[(\text{Obs.} - \bar{x}) / \bar{x}]$  in order to make results from different experiments compatible, and the correct calculation of variance for this unit would be difficult. If we however for purposes of demonstration only regard it as normally distributed, we obtain the following regressions and P-values for regressions being unreal:  $y - cv$ ,  $b = 2.20$ ,  $P = 0.06$ ;  $cv - v$ ,  $0.89$ ,  $8.06$ ;  $v - f$ ,  $0.34$ ,  $0.52$ ;  $f - car$ ,  $-3.72$ ,  $0.01$ .