Research Notes

Voss, R. Israel. The Hebrew University, Jerusalem, A common suppressor for a lethal mutation and a forked mutation.

The reversion of lethal mutations that were induced in the proximal segment of the X-chromosome is now being studied. Most revertants are sterile. One revertant (in an X-ray induction series) of  $1^{3DES}$ , a lethal induced by

diethyl sulfate (Lifschytz & Falk, 1968) was fertile. The reversion of the lethal effect was found to be due to a suppressor mutation on the X-chromosome. The mutant also suppresses the forked mutation and was found to be allelic to su-f. The new suppressor is recessive, the heterozygous female  $1^{3DES}$  su-fv/ $1^{3DES}$  being inviable. The hemizygous male  $1^{3DES}$  su-fv has outstretched wings and a shortened body. Homozygous females  $1^{3DES}$  su-fv/ $1^{3DES}$  su-fv are sterile and show the same abnormal phenotype as the hemizygous males; the pigmentation of the abdomen of these females resembles that of bobbed females, but on crossing su-fv to bb they were found to be non-allelic. Experiments to determine the range of other mutations suppressed by su-fv (especially other lethals at loci nearby in the proximal segment of the X-chromosome) are now under way. However, no interaction between  $v^a$  and su-fv was found.

The fact that mutations at the su-f locus affect more than one kind of mutant loci suggests the involvement of this gene in the synthesis of some basic protein being a common precursor to more than one metabolic pathway.

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replication pattern of the puffing sites
in the X-chromosome of Drosophila hydei.

DNA replication pattern in polytene chromosomes of Drosophila hydei confirms the earlier observations (Berendes 1966; Lakhotia and Mukherjee 1970; Mulder et al. 1968; and Rodman 1968) supporting the continuous to discontinuous pattern of replication. The pattern observed shows

that the sites replicating faster include most of the puffing sites.

Studies on the replication pattern of the X-chromosomal and autosomal puffs at a particular "puff stage" provides some information on the relation of replication sequence and transcribing activity and its bearing on dosage compensation in Drosophila. For this study, the excised glands were incubated in 0.05ml Ringer containing 5µCi of H³-TdR (Sp. Activity 5.27 Ci/mM) for 20 minutes. Ten days exposure and usual methods of autoradiography were followed. The presence of a minimum of 8 grains was considered as a criterion for designating a puff labeled.

The replication patterns of 14 autosomal and 18 X-chromosomal puffs arranged in a continuous-to-discontinuous sequence are presented in Table 1. It is clear that at the beginning of

Table 1. Replication pattern of 14 autosomal and 18 X-chromosomal puffs of Drosophila hydei

No. of labeled puffs in autosome	No. of labele	d puffs in X
(male and female)	female	male
14	18	13 - 18
9 - 12	15	10 - 12
5 - 8	12 - 13	2 - 10
3 - 4	9 - 12	1 - 2
1 - 2	0 - 9	0 - 1

replication cycle the pattern is same in both the sexes, but as the replication proceeds the puffing sites of the X in male show less and less incorporation when compared with the puffing sites in the female X's (Table 1). For example, when 9 autosomal puffs are labeled on the 4th chromosome, the number of labeled puffs on X in the male and the female are 10 and 15 respectively. Conversely, when same puffs on the X in the male and the female are labeled, the number of autosomal puffs labeled are more in the male than in the female.

These results reveal that the single-X of the male shows a differential replication pattern with respect to the pattern in the female X's and early completion of the process as well. In addition, individual puffs of the male-X present a faster rate of replication when compared with those in the female X. The data are compatible with hyperactive male X model of dosage compensation in Drosophila.

References: Berendes, H.D., 1966 Chromosoma 20: 32-43; Lakhotia, S.C. and A.S. Mukherjee, 1970 J. Cell. Biol. 47: 18-33; Mulder, M.P., P. van Duijin and H.J. Gloor, 1968 Genetica 39: 385-428; Rodman, T.C., 1968 Chromosoma 23: 271-287.