A. S. Mukherjee and Ashish Dutta Gupta.
University of Calcutta, India. The role of heterochromatin in the control of gene activity.

In a recent report from Dr. J. Schultz's laboratory, it has been shown that a block of heterochromatin when brought close to certain euchromatin bands inhibits puffing of those bands in the salivary gland chromosomes of Drosophila melanogaster (Rudkin 1964). It appears that heterochromatin may have a general inhibitory role on the genetic activity.

While studying the puffing pattern in Drosophila ananassae (a local population), in our laboratory, we have found certain features which do not permit us to generalize the inhibitory role of heterochromatin. Salivary gland chromosomes of D. ananassae contain a large number of chromosomal rearrangements (Jha, 1964). In addition, chromosome aberrations involving only one nucleus or two nuclei are found in high frequency in the $a^{6}+$ (Calcutta) stock currently under study. On the basis of observations so far made it appears that the reasons for such frequent "aberration mosaicism" may be due to the presence of heterochromatin of varying size over the length of the chromosomes. The details of the distribution of heterochromatin, degree of heterochromatinization of different bands and their relation to chromosomal aberrations will be published elsewhere. It is intended to present here a few cases of puffing which appear to be differentially modulated by the heterochromatin adjacent to them.

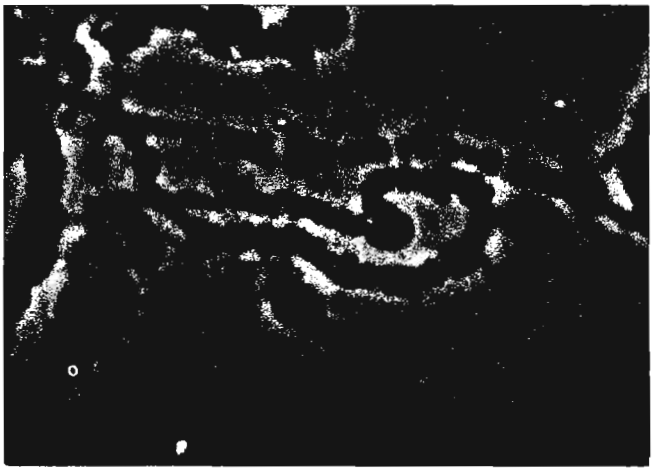

Fig. 1. Photograph of the subterminal inversion in the 3L. Arrow indicates the region of puff.

Figure 1 presents an inversion loop (the delta inversion of Jha, 1964) in which the two breaks are in 8A4 and 1A, respectively. The distal break is between the last dark band and the tip puff. Normally the tip remains puffed throughout the third instar. In this case, however, the tip of one and the same member of the two homologous chromosomes always shows puffing, while that of the other almost always ends in one or more dotted bands. A thin delicate thread-like connection is frequently observed under phase contrast, in the latter homolog, between the region preceding the dotted bands and any other non-homologous region. Such ectopic pairing indicates the presence of heterochromatin (intercalary?) in that region. This means that due to the inversion a piece of heterochromatin has been transposed into the region preceding the tip. It is clear, therefore, that this small piece, perhaps comparable to one single band, of heterochromatin is capable of inhibiting the tip puff. It is interesting to note that in cases, where the chromosomes concerned are completely unpaired, one homolog ends in a puff, the other shows two dark bands followed by one faintly stained dotted band.

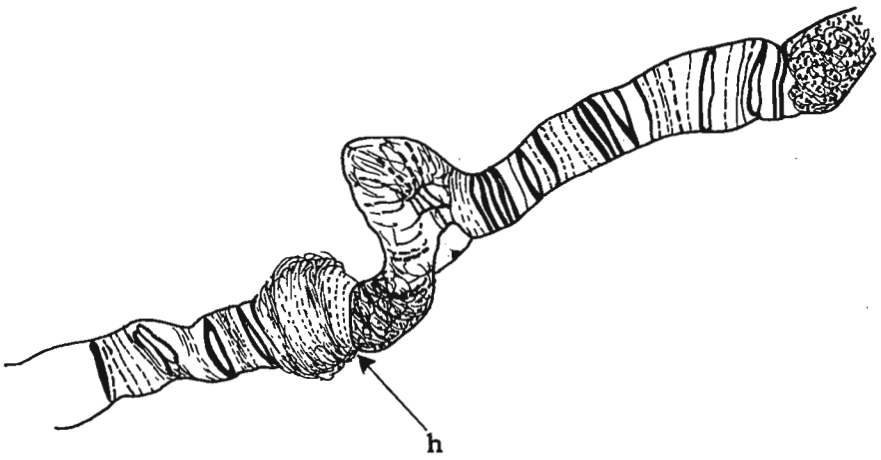


Fig. 2. Camera Lucida drawing of the puff and the adjacent heterochromatin in the XR. h=heterochromatic region.

The second set of cases on the other hand has larger segments of heterochromatin adjacent to puffs. One such case is presented in Figure 2. This represents the presence of a big puff close to a rather big block of heterochromatin. Clearly, the puff remains

unsuppressed even in presence of the heterochromatin closeby.

These preliminary findings show that there may be at least two functionally different kinds of heterochromatin. In normal gene sequence the bigger blocks of heterochromatin may have no or little effect on the control of genetic activity, while smaller intercalary heterochromatin bands may inhibit gene expression. Salivary gland chromosomes of D. ananassae proves to be a unique material for the study of the function of the heterochromatin in a normal genic complement. It remains for future investigation to examine the puffing in chromosomes homozygous for the inversion and also in homozygous normal chromosomes.

References

Jha, A.P. 1964. Ph.D. Thesis, B.H.U.; Rudkin, G. 1964. Proc, XI Int. Cong. of Genetics. Genetics Today, Vol. 2. 359-374.

Burnet, B. University of Sheffield, England. Allelism of tumour genes.

Hartung (1950, J. Hered.,41: 269) reports the location of a melanotic tumour gene at 2-83.9 in the $mt^A$ strain of D. melanogaster. This tu allele was used by Kanehisa (1956, Jap. J. Genet.,31: 144) for the synthesis of a number of tumour strains incorporating other mutants affecting eye pigmentation. Glass (1954, DIS 28: 74) reports that the tumour gene on the second chromosome of the su-er tu bw; st er su-tu strain is also an allele at 2-83.9. A detailed study of gene environment interactions involving this locus is given by Burnet and Sang (1964, Genetics 49: 223-235 and 599-610). The tumour gene on the second chromosome of the tu-B3 strain described by Barigozzi and De Pasquale (1956, Rend. Ist. Lomb. Sci. Lett., 90: 484) appears to be an allele at the same locus. The tumour penetrance (percentage of tumorous individuals) in crosses in all combinations between tu-B3, su-er tu bw; $+^{su-tu}$ and the $tu^{48a}$ vg bw strain described by Gélélovitch (1958, Biol. Méd., 47: 711) is shown in Table 1. The tumour gene $tu^{48a}$ is located at 2-29.5.

Table 1

| ♀♀ \ ♂♂ | tu bw; $+^{su-tu}$ | tu-B3 | $tu^{48a}$ vg bw |
|---|---|---|---|
| tu bw; $+^{su-tu}$ | 97.50 | 95.00 | 1.85 |
| tu-B3 | 85.20 | 90.40 | 2.14 |
| $tu^{48a}$ vg bw | 1.21 | 1.38 | 96.60 |

The allelism of the tumour genes in the su-er tu bw and tu-B3 second chromosomes is further supported by their interaction with the suppressor locus on the third chromosome st er su-tu. On a standardized first chromosome background both alleles are suppressed by su-tu, whereas $tu^{48a}$ does not appear to interact with the suppressor, as shown in Table 2.

Table 2

| | tu bw | tu-B3 | $tu^{48a}$ vg bw |
|---|---|---|---|
| $\frac{st\ su-tu}{+\quad +}$ | 93.60 | 93.40 | 43.1 |
| $\frac{st\ su-tu}{st\ su-tu}$ | 7.14 | 8.69 | 45.0 |

The reduction in penetrance observed in both these $tu^{48a}$ combinations is due to dispersion of the modifier background particularly in the first chromosome of the original strain. Further observations are necessary to decide whether the effects of the suppressor are restraictd to tu alleles at locus 2-83.9.