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.