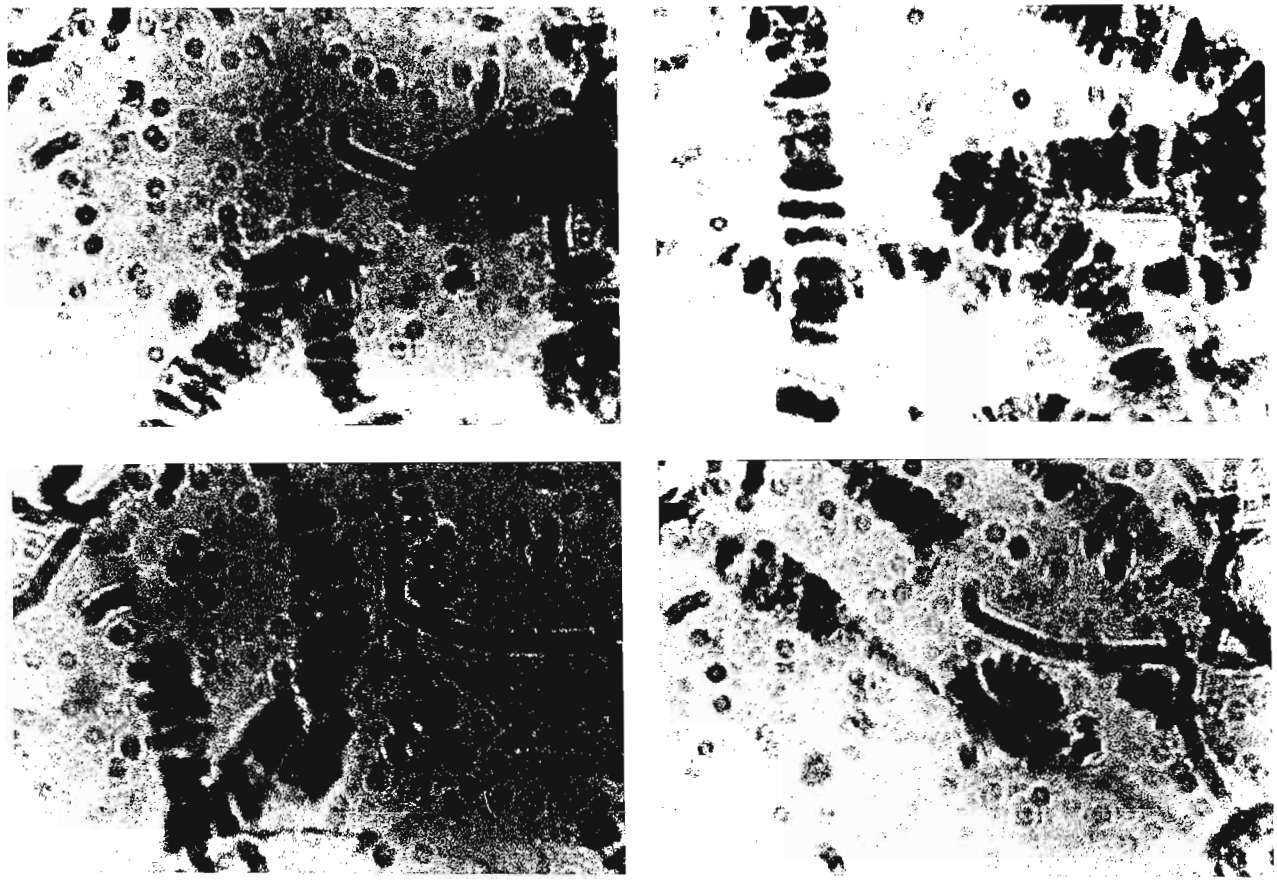
Chenevix Trench, G. Trinity College, Dublin, Ireland. An endemic inversion in the X-chromosome of D. melanogaster.

An endemic paracentric inversion has been found in the X-chromosome of D. melanogaster. 35 progeny from a multiple mating (12 virgin females from a wild population, Dahomey, mated with 5 males of the Oregon-K inbred strain) were examined cytologically by salivary gland preparation. In addition, 2 or 3 larvae from each of 5 single crosses were examined. This new inversion, which has been named In(1)A, was found in 20 of the 47 individuals examined. It is a small paracentric inversion with break-points at 16D and 18D, and was always observed to form an inversion loop in salivary gland cells of female larvae.

This discovery is particularly interesting in view of the rarity of X-chromosome inversions. Dahomey is a wild type stock, collected in West Africa in 1969 and maintained in large cage populations in Edinburgh, whence this population came. It is noted for the high level of genetic variation which it has retained in the laboratory.



Chernyshev, A.I. and B.A. Leibovitch. Institute of Molecular Genetics, USSR Academy of Sciences, Moscow, USSR. The effect of temperature during development on the amount of heterochromatin DNA fractions in D. melanogaster.

It has been shown earlier by cytophotometry that low temperature may cause underreplication of heterochromatin in a number of different organisms (Evans 1956). Heterochromatin DNA in Drosophila basically consists of ribosomal DNA and highly reiterative DNA. The latter contains two fractions: satellite and rapidly renaturing DNA. Satellite DNA consists of long blocks of simple repeats which differ from the main DNA fraction in buoyant density and are localized, with a few exceptions, in centromeric heterochromatin (Peacock et al. 1974). The rapidly renaturing DNA fraction consists of short blocks of simple repeats which do not differ from the main fraction in buoyant density (Hearst et al. 1974) and are localized in centromeric heterochromatin and some other loci, as follows from in situ hybridization (Fig. 1).

Fig. 1. Hybridization of ^{3}H RNA synthesized on rapidly renaturing DNA with D. melanogaster polytene chromosome. The intensive labeling of centromeric heterochromatin and some other loci is clearly visible. Note regions 39D-E where histone genes are localized.

To find out whether the amount of heterochromatin DNA depends on the conditions of the flies' growth, we determined the amount of ribosomal, rapidly renaturing and satellite DNA in larvae and flies of the wild Oregon-RC stock which had been kept at 16°C for two generations and in control flies permanently kept at 25°C. For this purpose RNA was hybridized, in the presence of excess RNA, with ^{3}H ribosomal RNA and with ^{3}H complementary RNA synthesized by E. coli RNA polymerase on the total satellite DNA (a mixture of satellites I, II and IV) and on the rapidly renaturing DNA. The satellite DNA was isolated in a CsCl gradient with actinomycin D (Peacock et al. 1974) and the rapidly renaturing DNA with Cot 10^{-1} was obtained from the main band DNA fraction by fractioning on hydroxyapatite (Hearst et al. 1974).

The low temperature during development was found not to cause any decrease in the amount of the above heterochromatin DNA fractions either in larvae or in adult males (Table 1). Moreover, the amount of rDNA and rapidly renaturing DNA was the same in adult females reared at 16°C and 25°C. A significant difference was observed only with respect to satellite DNA in adult females reared at 16°C as compared with the control females (25°C). If the amount of ribosomal DNA and rapidly renaturing DNA is invariable, it is unlikely that the increase in satellite DNA content should be due to a relative decrease of the main DNA fraction. Since a large part of female DNA is constituted by oocyte DNA and that of the polytene nurse cells, the underreplication of satellite DNA at 25°C may be due to a greater polyteny of the nurse cells at normal temperature. But there is a reverse situation in the case of Drosophila salivary glands (Hartmann-Goldstein and Goldstein 1979). Another possible explanation of the cold-induced increase in the proportion of satellite RNA may be an increase in the amount of unfertilized eggs at 16°C, the proportion of satellite DNA in them being as high as 80% of total DNA (Travaglini et al. 1972).

The above suppositions may explain the lack of excess satellite DNA in males unlike females. At the same time we cannot exclude the possibility that the highly heterochromatized Y chromosome in males affects the heterochromatin of the ausotomes. This seems to be suggested by the fact that the introduction of an additional Y chromosome into the Drosophila genome causes a smaller increase of satellite DNA than could be expected from the satellite DNA content in the Y chromosome (Wollenzien et al. 1977). However, all these are suppositions that must be tested.

It is remarkable that adult females have twice as much satellite DNA as larvae, whereas the amount of ribosomal and rapidly renaturing DNA differs only slightly (Table 1). The excess of satellite DNA in flies corresponds to a smaller proportion of polytene tissues as compared with larvae. Meanwhile satellite DNA is more underreplicated than ribosomal DNA in larval salivary glands. The salivary glands of D. melanogaster contain 100 to 200 times less satellite DNA than the diploid tissues (Gall et al. 1971) but only 4 to 6 times less genes for rRNA than the diploid tissues (Spear and Gall 1973). The differences observed are probably due to this selective underreplication.

Lately it has been hypothesized that the amount of satellite DNA is unstable, for it may vary in closely related species (Gall et al. 1974) and in different tissues within one organism in the course of aging (Prashad and Culter 1976). Our data on the effect of temperature during development upon the satellite DNA content in Drosophila possibly support this hypothesis.

References: Evans, W.L. 1956, Cytologia 21:417-432; Gall, J.G., E.H. Cohen and M.L. Polan 1971, Chromosoma 33:319-331; Gall, J.G., E.H. Cohen and D.D. Atherton 1974, Cold Spring Harbor Symposia 38:417-421; Hartmann-Goldstein, I. and D.J. Goldstein 1979, Chromosoma 71:333-346; Hearst, J.E., T.R. Cech, K.A. Marx, A. Rosenfeld and J.R. Allen 1974, Cold Spring Harbor Symposia 38:329-340; Peacock, W.J., D. Brutlag, E. Goldring, R. Appels, C. Hinton and D.L. Lindsley 1974, Cold Spring Harbor Symposia 38:405-416; Prashad, N. and R.G. Culter 1976, Biochim.

Table 1. The amount of different fractions of heterochromatin DNA in larvae and flies reared at 25°C and 16°C.

DNA source	t°C during development	Ribosomal DNA: % of total DNA	Ribosomal DNA: 16°C in % of 25°C*	Ribosomal DNA: ♀ larvae & ♂ imago in % of ♀ imago	Ribosomal DNA: N	Rapidly renaturing DNA: % of total DNA	Rapidly renaturing DNA: 16°C in % of 25°C*	Rapidly renaturing DNA: ♀ larvae & ♂ imago in % of ♀ imago	Rapidly renaturing DNA: N	Total satellite DNA: % of total DNA	Total satellite DNA: 16°C in % of 25°C*	Total satellite DNA: ♀ larvae & ♂ imago in % of ♀ imago	Total satellite DNA: N
♀ larvae	25	0.254	(100)	82	2	5.87	(100)	95	2	3.6	(100)	62	2
	16	0.262	103±2	84	2	5.93	101±1.6	94	2	3.67	102±1.3	54	2
♀ imago	25	0.310	(100)	(100)	14	6.2	(100)	(100)	6	5.8	(100)	(100)	17
	16	0.313	101±1	(100)	14	6.32	102±3	(100)	6	6.84	118±1	(100)	17
♂ imago	25	0.350	(100)	113	3	6.95	(100)	112	5	7.43	(100)	128	6
	16	0.364	104±2.2	116	3	7.29	105±1.9	115	5	7.01	95±4	102	6

The hybridization of DNA immobilized on nitrocellulose filters was carried out with an excess of labeled RNA. 1 to 2 µg of DNA deposited on a 5mm HAWP filters was annealed in 25 µl of 4 SSC for 18 hours at 66°C in the presence of 0.1 µg of ^{3}H rRNA or 1-2 µg of ^{3}H RNA complementary to the satellite or rapidly renaturing RNA. After annealing the filters were treated with RNAase and counted by a scintillation counter.

N = the number of experiments.

* Results expressed as mean ± standard deviation.

Biophys. Acta 418:1-23; Spear, B.B. and J.G. Gall 1973, Proc. Natl. Acad. Sci. USA 70:1359-1363; Travaglini, E.C., J. Petrovic and J. Schultz 1972, Genetics 72:431-445; Wollenzien, P., P. Barsanti and J.E. Hearst 1977, Genetics 87:51-65.