Mather, W.B. and K.S. Tam. University of Queensland, Brisbane, Australia. Inversions from Chiang Mai, Thailand. 3rd Report.

TABLE

Inversion	Chromosome	Simple	Complex	Het. Freq. %
A_5	II L	X		30.2
E	II L	X		22.2
C_5	II R	X		11.1
B_5	III	X		12.7
C_1	III	X		3.2
D_5	II L		X	1.6

In July 1983 sixty-three isolines of **D.s.albostrigata** and six isolines of **D.albomicans** were established from Chiang Mai, Thailand. Inversions in these species were last reported on from Chiang Mai in November 1982 (Mather & Pope, DIS 60:141).

(a) D.s.albostrigata. Five simple and one complex inversion were detected. All inversions had previously been detected in Southeast Asia but D_5 was new to Chiang Mai. Heterozygosity frequency of all inversions detected is given in the Table.

(b) D.albomicans. One simple and one complex inversion were detected. E' (simple) had previously been detected from Chiang Mai but J_8 (complex) although recorded from South East Asia was new to Chiang Mai.

The material was collected and the isolines established by W.B.M. The laboratory work was carried out by K.S.T.

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Table 1.

Inversion	Chromosome	Simple	Complex	Het.Freq.%
A_3	I	X		3.6
G	I	X		7.1
A_5	II L	X		71.4
E	II L	X		28.6
D_5	II L		X	17.9
C_5	II R	X		7.1
D_3	II R	X		3.6
B_5	III	X		25.0
C_1	III	X		3.6

In November 1983 twenty-eight isolines of **D.s.albostrigata** and one isoline of **D.albomicans** were established from Chiang Mai, Thailand. Inversions in these species were last reported on from Chiang Mai in July 1983 (Mather & Tam, DIS 61: this issue).

(a) **D.s.albostrigata.** Eight simple and one complex inversion were detected. All inversions had previously been detected in Southeast Asia, but D_3, G, and A_5 were new to Chiang Mai. The heterozygosity frequency of all inversions detected is given in the Table.

(b) **D.albomicans.** One simple inversion was detected (inversion E^1). This had previously been detected from Chiang Mai.

The material was collected and the isoline established by W.B.M. The laboratory work was carried out by K.S.T.

Matyunina, L.V. and T.I. Gerasimova. Inst. of General Genetics, USSR Academy of Sciences, 117908 Moscow, USSR. Study of spontaneous sex-linked lethal mutations in the unstable line ct^{MR2} of **Drosophila melanogaster.**

Earlier an unstable ct^{MR2} mutation was obtained at the locus cut (20.0; 7B 3-4) under conditions of P-M hybrid dysgenesis (Gerasimova 1981). This mutation is induced by a mobile dispersed gene MDG4 (Gerasimova 1984a). This line homozygous for X-chromosome is characterized by a high reversion frequency, segregation of novel ct mutations with a new phenotype and superunstable ct mutations, as well as by the formation of new visible mutations for other loci of the X-chromosome (Gerasimova 1981).

Phenotypical changes arising in the $ct^{MR}2$ line are the markers of so-called transpositional bursts, i.e., mass simultaneous transpositions of different mobile elements in the same germ cell (Gerasimova 1984b). The frequency of transpositional bursts in the ct^{MR2} line was about $1x10^{-3}$. Since transpositional bursts result in active insertion mutagenesis, the true frequency of "bursts" may be significantly higher because in this case lethal mutations should occur. Therefore the objective of the present work is the estimation of the lethal mutation frequency in the X-chromosome of the ct^{MR2} line and characterization of the distribution of the lethals.

Table 1. Location of lethal mutations in the 6C12-8A5 region of X-chromosome.

	Character of complementation					
	Dp(1;3) sn^{13a}	Df(1) ct^{J4}	Dp(1;2)$^{+72d}$	Df(1)^{4b1}	ct^{JA124}	Number of
Region	6C12-7C9	7A1-7C1	7A8-8A5	7B3-7C4	7B3-4	lethals
6C12-7A13	-	+	+	+	+	13
7A2-7A8	-	-	+	+	+	-
7A8-7B2	-	-	-	+	+	2
7B3-4	-	-	-	-	-	33
7B5-7C1	-	-	-	-	+	4
7C2-7C8	+	-	+	-	+	-
haplolethal region						
7C9-8A5	-	+	-	+	+	29
						81

"+" = a lethal complementary to Df or Dp; i.e., 1/Df females survive and +/Dp; 1/Y males do not. "-" = a lethal noncomplementary to Df or Dp; i.e., 1/Df females do not survive and +/Dp; 1/Y males do.

To estimate the frequency of lethal mutations in the X-chromosome, the following matings were made: ♀ct^{MR2}/ct^{MR2} x ♂FM4/Y → (F_1) ♀ct^{MR2}/FM4 x ♂FM4/Y → (F_2) ct^{MR2}/FM4, ct^{MR2}/Y, FM4/Y. 31742 ct^{MR2} chromosomes were examined in F_2 and 353 lethal mutations; 50 reversions and several visible mutations were discovered. The frequency of ct^{MR2} → ct^+ reversion in this experiment was 1.9×10^{-3}. The frequency of lethal sex-linked mutations was 1.1×10^{-2}. The same scheme was used in the control experiments in which females of the wild line Oregon R were used instead of ♀ct^{MR2}/ct^{MR2} ones. 5120 chromosomes were examined and not a single lethal or visible mutation was found. This means that the formation of lethal, visible mutations and reversions is induced by the unstable line ct^{MR2}.

Since in the "ct^{MR2} system" visible mutations occur most frequently at genes cm(6E), ct (7B) and sn (7D), it was of interest to study this region in detail with respect to lethal mutations. Therefore the lethal mutations obtained were analyzed for the presence in the 6C12-8A5 region of the X-chromosome. To this end, females carrying the lethal mutation 1/FM4 were mated to males containing various deficiencies and duplications of this region: Df(1)4b1, ct^{4b1}oc ptg/In(1)dl-49, y sc lz^5B; Dp(1;3)sn^{13a} [Df(1)4b1 deficiency involves the 7B3-C4 region, duplication Dp(1;3)sn^{13a} involves the 6C12-7C9 region]; ct^{JA124} ; Dp(1;2)sn^{+72d}/In(2LR)Gla [the lethal mutation ct^{JA124} at the ct locus induced by X-rays and pertaining to complementation group III of the locus cut]; Dp(1;2)sn^{+72d} involves the 7A8-8A5 region; Df(1)ct^{J4} deficiency involves the 7A1-7C1 region. The results of the complementation analysis are presented in Table 1.

As is seen from Table 1, 81 of the mutations analyzed are located in the region 6C12-8A5. However, this region comprises about 8% of the whole X-chromosome. Thus, a disproportionately large number of lethal mutations are located in the 6C12-8A5 region. 33 of them were located at the cut locus. The rest of the lethals were tested for allelism with mutations at the loci carmine and singed. However, all the lethals tested were complementary to cm and sn mutations. Thus, besides the locus cut the remaining mutations pertain to unknown, vitally important loci and are located at different sites of the 6C12-8A5 region. It is of interest that 7C2-7C8 bands contain a haplo-lethal region (Lefevre & Johnson 1973) in which it is impossible to detect lethal mutations with the help of X-rays. In the present work, no lethal mutations have been found in the haplo-lethal region 7C2-7C8, too.

An overwhelming majority of lethal mutations occurred at the background of the ct^{MR2} allele. Only 6 of 353 lethals were accompanied by reversals of ct^{MR2} to the wild type ct^+. As well as visible mutations, many lethal mutations appeared to be unstable. Most reversions involved only the lethal mutation and sometimes in double revertants of the 1^+ct^+ type. In some cases, both types of revertants ($ct^{MR3}1$ and ct^+1^+) also carried new visible mutations at different loci of the X-chromosome (y, w, cm, ct, sn, g, pn, m, v, r, B) and the processes of reversion and mutagenesis involved 2-5 loci simultaneously. Besides, mutational transitions accompanied by the reversion of some lethal mutations and formation of lethals at other loci were observed. Thus, the lethal apparently changed its location on the chromosome. This type of mutational transition could be determined only after repeated localization of lethal mutations. Repeated localization of lethals in the 6C12-8A5 region was carried out after a 1-year period. It appeared that 14 out of 81 lethal mutations had changed their location. 4 lethals "left" this region; i.e., the transition l(6C12-8A5) ct^{MR2} → $1^+ct^{MR2}1$ occurred [1 being out of the 6C12-8A5 region]; one lethal "transited" from the locus to the neighbouring region 7B5-7C1, i.e., ct^{13} → $ct^{MR2}1^3$; 9 lethals earlier located in the 6C12-7A1 region occurred in 7B3-4, i.e, in the region of the cut locus: 1 ct^{MR2} → 1^+ct^1.

The unstable and apparently insertional character of the lethal mutations obtained suggests that they, as well as visible mutations, must be the result of transpositional bursts. Individuals with lethal mutations will be eliminated in early ontogenesis, the result being a sharp reduction in the yield of viable mutants. This points out that the true frequency of transpositional bursts must be higher as compared to that of visible mutations at least by a magnitude of one order.

The authors are deeply indebted to Dr. T.K. Johnson for sending stocks Df(1)4b1, ct^{4b1} oc ptg/In(1)dl-49, y sc lz^{5}B; Dp(1;3)sn^{13a}; ct^{JA124} Dp(1;2)sn^{+72d}/In(2LR)Gla; and Df(1)ct^{J4}/ct^{+}.Y .

References: Gerasimova, T.I. 1981, Mol. Gen. Genet. 184:544-547; Gerasimova, T.I., Y.V. Ilyin, L.J. Mizrokhi, L.V. Semjonova & G.P. Georgiev 1984a, Mol. Gen. Genet. 193:488-492; Gerasimova, T.I., L.J. Mizrokhi & G.P. Georgiev 1984b, Nature 309:714-716; Lefevre, G. & T.K. Johnson 1973, Genetics 74:633-645.

Melzer, S. and K.H. Glätzer. Institut für Genetik, Düsseldorf, FR Germany. Localization of RNP antigens in primary spermatocytes of **Drosophila melanogaster** by indirect immunofluorescence and their correlation to fertility factors.

A subset of monoclonal antibodies raised against nuclear proteins of **D.melanogaster** cells are specific for ribonucleoprotein complexes (RNP) (Risau et al. 1983). It could be shown that some of these crossreact with polytene chromosomes of **D.hydei** (Saumweber et al. 1980). Surprisingly the respective antigens are concentrated on distinct Y chromosomal structures in primary spermatocytes of this species (Glaetzer 1984). Because the Y chromosome in Drosophila is indispensible for male fertility, similar functions may be reflected by a similar accumulation of RNP antigens on particular Y chromosomal formations. We therefore tested a number of monoclonal antibodies on cytological preparations of spermatocytes of **D.melanogaster**. In addition we mapped the labeled nuclear structures on the Y chromosome with the positively reacting antibodies. For this purpose we used translocation stocks of J.A. Kennison (1981) which he had kindly donated to Dr. U. Schaefer of our institute.

Out of six antibodies tested (P11, Q16, S5, T7, V4, X4), four (S5, X4, P11, Q16) showed a positive reaction with spermatocyte nuclei. The comparison between X0 cells (Fig. 1d, e) and cells carrying a Y chromosome (Fig. 1a, b) or a fragment of it (Fig. 1c,f) revealed that S5 and X4 antigens are concentrated on Y chromosomal chromatin (data with X4 antibody not shown). The other antibodies P11 and Q16 are associated with the presumed autosomes and the remaining nuclear compartment (data not shown).

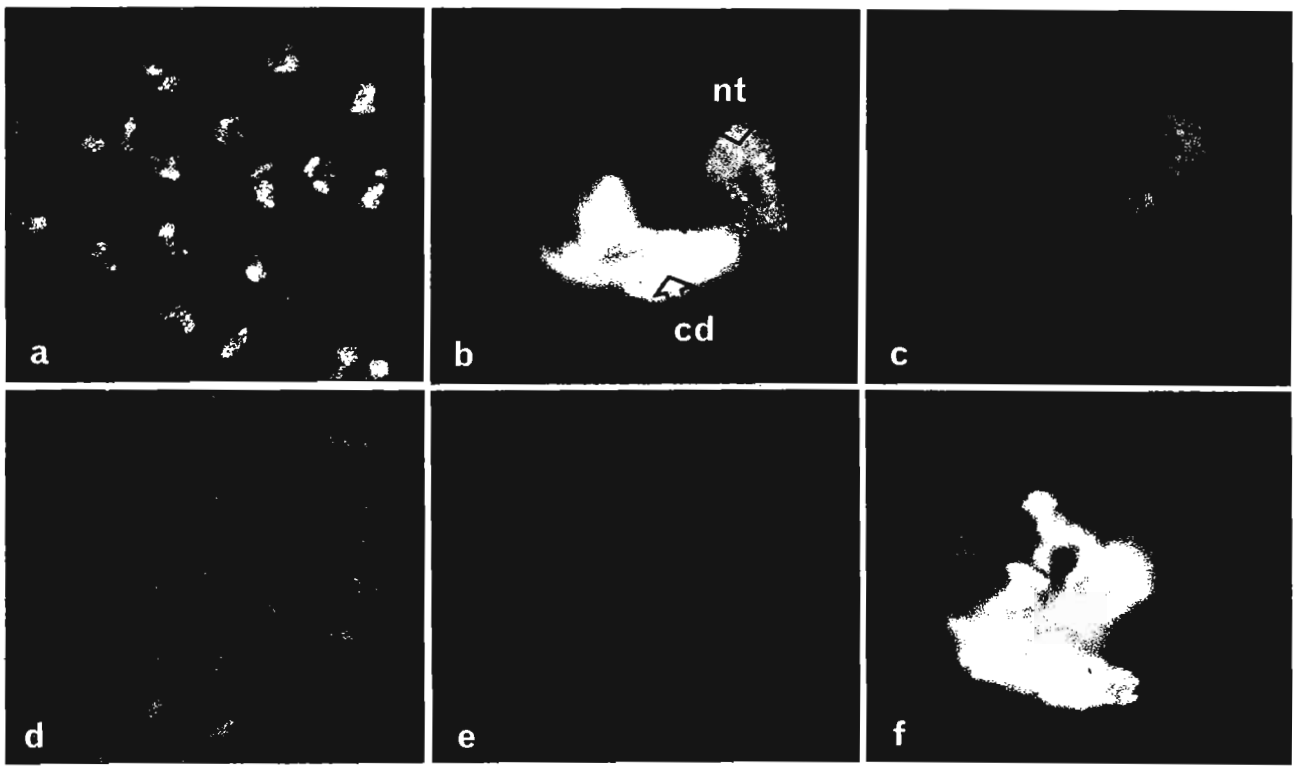

Figure 1. Localization of antigen S5 by indirect immunofluorescence. Staining pattern of: (a) X·$Y^{KL\cdot ks\text{-}1}$ V8-genotype; (b) X/Y-genotype; (c) X·Y^{KS} F12-genotype; (d,e) X0-genotype; (f) X/$Y^{kl\text{-}5}$ V24-genotype. **cd**: "clods"; **nt**: "net". Magnifications: a,d) x325; b,c) x1600; e) x1800; f) x2200.