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 .

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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).

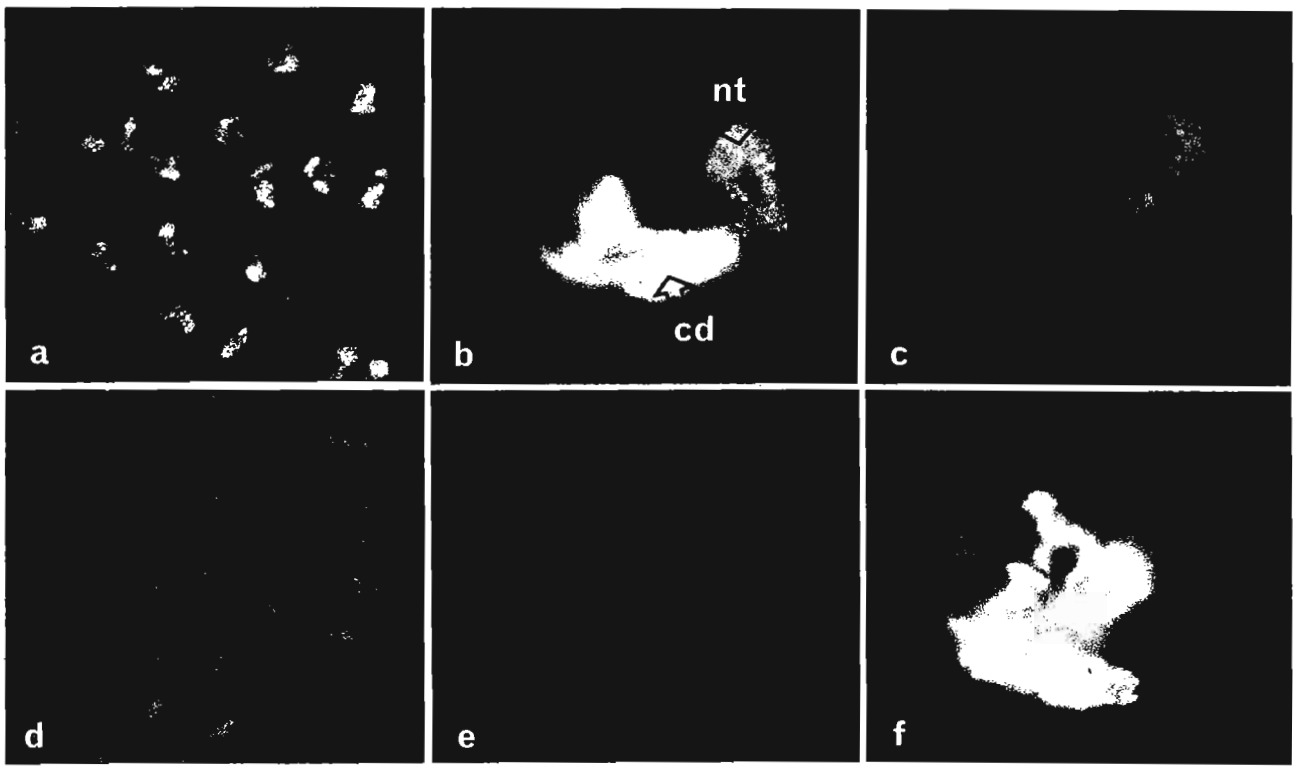

Figure 1. Localization of antigen S5 by indirect immunofluorescence. Staining pattern of: (a) X·$Y^{KL\cdot ks\text{-}1}$ V8-genotype; (b) X/Y-genotype; (c) X·Y^{KS} F12-genotype; (d,e) X0-genotype; (f) X/$Y^{kl\text{-}5}$ V24-genotype. **cd**: "clods"; **nt**: "net". Magnifications: a,d) x325; b,c)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

From Fig. 1b it is evident that two Y chromosomal structures are decorated by S5 (and X4) antibody. It appears that the antigenic determinants of both antibodies are located on the same RNP structures. One structure is best described as coarse and lumpy; the other Y chromosomal formation consists of a fine granular network. Following the tradition of naming the Y chromatin in primary spermatocytes in accordance with its morphological characteristics, we propose to call them "clods" (cd) and "net" (nt), respectively. This is all the more justifiable as the "clods" correspond to the fertility factor kl-5 and the "net" to the functional unit ks-1. Neither structure, however, has an equivalent in the phase contrast microscope (Meyer et al. 1961).

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Miglani, G.S. and V. Mohindra. Punjab Agricultural University, Ludhiana, India. Detection of chromosomal aberrations in the progenies of EMS-induced recombinants in **D.melanogaster** males.

Table 1. Chromosomal aberrations observed in the larvae produced in the TC2 progenies of various TC1 recombinants induced with 0.75% EMS. Aberrations (1) and (2) were from two different larvae selected from TC2 progeny of the same TC1 recombinant; (i) and (ii) were detected in the same chromosome complement; (a) and (b) were detected in two different chromosome complements of larva (2).

Phenotype of TC1 recombinant		Nature of aberration	Chromosome	Breakage-union Points
+ b cn		Inversion	2R	57F-60C
+ b cn		Inversion	2L	25B-28A
+ b cn	(i)	Inversion	2L	24B-27C
	(ii)	Deficiency	2R	52E-52F
(1)+ b cn	(i)	Inversion	3R	94A-95F
	(ii)	Deficiency	2R	52E-52F
(2)+ b cn	(a)	Inversion	3L	70F-73C
	(b)	Inversion	2L	25B-28A

F_1 (Oregon-K) +/dumpy (dp) black (b) cinnabar (cn) **D.melanogaster** males were treated with 0.75% ethyl methanesulphonate (EMS), through feeding in the second one-third part of 96 hr larval life, at 25±1°C. Control experiments were also performed simultaneously. The untreated and EMS-treated F_1 males were crossed with dp b cn females and recombinants were recovered in the first test cross progeny (TC1). In EMS experiments, flies of phenotype + b cn appeared predominantly over their complementary class dp + +, suggesting induction of non-reciprocal recombination in dp-b region. The recombinant flies were again test crossed to obtain second test cross generation (TC2). Late third instar TC2 larvae were randomly selected and sacrificed for study of salivary chromosomes to determine whether any chromosomal aberration induced with EMS was associated with induction of a particular recombinant.

From each of 20 different TC2 progenies of TC1 male recombinants recovered in EMS experiments, salivary chromosomes of 4 to 6 larvae were examined. Chromosomes of all the 83 larvae studied from the control experiments were found to be free of chromosomal abnormalities. Out of 118 larvae studied from 28 progenies of TC1 male recombinants induced in EMS experiments, only four were found to carry a total of 8 chromosomal aberrations. Phenotype of the TC1 recombinants, nature of aberrations detected and the breakage-union points of aberration detected in a particular chromosome are given in Table 1. The salivary chromosomes revealed 4 different and 2 identical inversions and two identical deletions. Out of the 6 inversions detected, three overlap dp-p region. The remaining 3 inversions and 2 deletions are located outside the dp-b-cn region.

In the present study, progenies of only a few + b cn TC1 male recombinants were found to carry chromosomal abnormalities; no such abnormality was detected in the progenies of many other + b cn recombinant males. Majority of the + b cn TC1 recombinants produced recombinant type flies in TC2 below 15% and in certain cases it was as low as 0.38%. The probability of recombinant-type larva being sampled in TC2 progeny was thus very low. The examination of salivary chromosomes of a very large number of TC2 larvae per TC1 recombinant is desirable to have a better understanding about the possible association of chromosomal aberrations with induction of male recombination.