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The relationship between heterochromatic homology and meiotic segregation of compound second autosomes during spermatogenesis in *Drosophila melanogaster*

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SUMMARY

In this report we examine the meiotic segregation of compound second autosomes sharing varying extents of heterochromatic and euchromatic homology. The second chromosome heterochromatin does not appear to influence the random meiotic segregation of compound second autosomes during spermatogenesis; however, the proximal euchromatin is implicated in male meiotic pairing. We conclude that male autosomal meiotic pairing sites are specific euchromatic chromosomal regions.

1. INTRODUCTION

During male meiosis in *Drosophila melanogaster* the reductional segregation of homologous chromosomal bivalents occurs in the absence of meiotic recombination (Morgan, 1912). The sex chromosomes regularly segregate during male meiosis (Bridges, 1916) and the *X* chromosome pairing sites responsible for its segregation from the heterochromatic *Y* chromosome lie in the *X* chromosome heterochromatin (Xh) (Muller & Painter, 1932; Gershenson, 1940; Sandler & Braver, 1954; Cooper, 1964; Peacock, 1965).

Analysis of second chromosome (and second chromosome derivative) bivalents heterozygous for heterochromatic deletions has failed to provide evidence for male meiotic pairing sites within the second chromosome heterochromatin. Hilliker (1975) analysed non-disjunction in males heterozygous for *In(2LR)SM1,Cy* and *Df(2R)M-S2*^{10}, a chromosome deficient for the 2R heterochromatic block. No non-disjunction between *Df(2R)M-S2*^{10} and *In(2LR)SM1* was observed and a 95 % upper confidence limit of non-disjunction of 0·068 % was established. Yamamoto (1979) examined cytologically the meiotic pairing during spermatogenesis of a *F(2R)* chromosome (a chromosome-2 with most of the left arm deleted – hence a 'free 2R') and *Df(2R)M-S2*^{10}. In 19 cells examined at metaphase I *F(2R)* paired

with *Df(2R)M-2*[10]. However, although Yamamoto diagrammatically represents *F(2R)* as having no heterochromatic homology with *Df(2R)M-S2*[10] (his figure 8*a*), its method of synthesis (Grell, 1970) indicates that *F(2R)* must have substantial 2L heterochromatin, and this has been documented cytologically (C. B. Sharp, personal communication; see also Holm, Fitz-Earle & Sharp, 1980). Further, the cytological assay is not sufficiently sensitive to reliably assay low levels of non-disjunction. Nevertheless, *F(2R)* has less heterochromatic homology with *Df(2R)M-S2*[10] than *In(2LR)SM1*, yet pairs effectively with *Df(2R)M-S2*[10] during spermatogenesis.

The results of the foregoing experiments must be interpreted with caution, for if male meiotic pairing sites are distributed throughout the autosomal heterochromatin and euchromatin, deleting or rearranging some or even all heterochromatic sites may not be sufficient to cause non-disjunction (see for example Appels & Hilliker, 1982).

Yamamoto (1979) made the important observation that *Dp(2;f)1*, a free duplication carrying a small euchromatic portion of *2R* and some of the proximal heterochromatin of chromosome 2 (Lindsley & Grell, 1968), failed to pair with an homologous *Dp(2;f)1* (or a normal second chromosome) at metaphase 1 of spermatogenesis in *Dp(2;f)1/Dp(2;f)1* males. Thus, the most proximal heterochromatin of chromosome 2 would appear not to contain male meiotic pairing sites.

We considered that the analysis of the meiotic segregation of compound-2 autosomes would allow us to further probe for autosomal male meiotic pairing sites in heterochromatin. Compound autosomes generally share no euchromatic homology, but, since they arise by a translocation event involving heterochromatic breakpoints, a pair of complementary compound autosomes shares heterochromatic homology. This could lead to nonrandom segregation if there are male meiotic pairing sites in autosomal heterochromatin. Indeed, a number of compound-2 autosomes do not exhibit completely random male meiotic segregation on the basis of egg hatch studies (reviewed in Holm, 1976). Data from a number of cases presented in this paper document that heterochromatic homology is insufficient to support meiotic pairing of compound-2 autosomes in males. We have obtained preliminary evidence that male specific pairing sites may reside in the proximal euchromatin of chromosome 2.

2. MATERIALS AND METHODS

(i) *Compound autosomes.* Compound-2 strains employed include (1) *C(2L)SH3,+*; *C(2R)SH3,+* (2) *C(2L)P,b*; *C(2R)P,px* (3) *C(2L)SH1,+*; *C(2R)SH1,+* (4) *C(2L)SH1,+*; *C(2R)SH3,+* (5) *C(2L)SH3,+*; *C(2R)VK1,bw* (6) *C(2L)VH1,lt*; *C(2R)VK1,bw* (7) *C(2L)SH3,+*; *C(2R)SH3,+*; *In(3LR)TM3,y*[+] *ri p*[p] *sep bx*[34e] *s*[s] *Sb Ser/*[+] (8) *B*[s]*Y*; *C(2L)P,b*; *C(2R)P,px* (9) *C(1)RM,+/B*[s]*Y*; *C(2L)P,b*; *C(2R)P,px* (10) *C(2L)V12,SD72/+*; *C(2R)V43,SD72/cn bw*. Each independent *C(2L)* or *C(2R)* chromosome is assigned a code in which the first letter identifies the origin (S = Storrs, P = Pasadena, V = Vancouver). For more information on these

compound autosomes see Holm (1969), Hilliker & Holm (1975), Holm (1976), Hilliker (1976) and the Results Section. Information regarding the gene mutations associated with the compound-2 autosomes and the other chromosomal rearrangements employed may be found in Lindsley & Grell (1968).

(ii) *Cytological analysis.* Polytene chromosomes from larval salivary glands and mitotic chromosomes from larval ganglia were prepared by fixation, staining and

Table 1. *Recovery of non-segregational progeny from* C(2L); C(2R) *females mated to* C(2L); C(2R) *males*

Experiment	Parents		Total progeny	Non-segregational progeny	
	Female	Male		Number	%
1	SH3,+;SH3,+	P,b;P,px	18766	333	1·77
2	P,b;P,px	SH3,+;SH3,+	10017	67	0·67
3	SH3,+;SH3,+ In(3LR)TM3/+	P,b;P,px	11245	1411	12·55
4	P,b;P,px	SH3,+;SH3,+	1771	715	40·37

squashing in aceto-lacto-orcein (2 % orcein). Salivary glands were dissected in 45 % acetic acid, transferred to stain and immediately squashed. Ganglia were dissected in Ringers (0·75 % NaCl) pretreated in 1 % sodium citrate for 1 min, transferred to stain for 5 min, and then squashed.

(iii) In situ *hybridization. In situ* hybridization of [^{3}H]cRNA to mitotic chromosomes was performed as described in Steffensen, Appels & Peacock (1981) (see also Appels, Steffensen & Craig, 1979).

(iv) *General features of the meiotic segregation of compound-2 autosomes.* Crosses involving compound-2 parents yield two classes of progeny. Those receiving either *C(2L)* or *C(2R)* from the mother and the complementary compound from the father are termed 'segregational' and those receiving both compounds from the mother or from the father are termed 'non-segregational'.

Crosses between compound-2 parents in which the male has an otherwise normal karyotype yield a low frequency of non-segregational progeny (Expts 1 and 2 of Table 1). Accordingly, either in the male or in the female compound-2 bearing parent, or in both, *C(2L)* regularly segregates from *C(2R)*. To distinguish among these possibilities we examine Expts 3 and 4 of Table 1 in which the compound-2 bearing female also carries a supernumerary *Y* chromosome (B^sY) or inversion heterozygosity for the third chromosome. The yield of non-segregational progeny in these crosses increases dramatically, despite the fact that the males are the same as those used in the previous crosses. Clearly, *C(2L)* does not regularly segregate from *C(2R)* in the male; whereas, in otherwise structurally normal females, *C(2L)* and *C(2R)* regularly segregate, consistent with earlier observations (Holm, 1969; Grell, 1970).

We believe that the elevated recovery of non-segregational progeny in Expts 3 and 4 of Table 1 is due to non-homologous pairing, in the oocyte, i.e. between the

non-crossover third chromosomes and compound-2 autosomes in Expt 3, and between B^sY and the compound second autosomes, in Expt 4, as has been previously documented and discussed by Grell (1970). Indeed the low frequency of non-segregational progeny arising from otherwise structurally normal compound-2 bearing females (e.g. Expts 1 and 2 of Table 1) arises largely from non-homologous pairing with non-crossover X chromosomes (Harger & Holm, 1980).

Table 2. *Summary of studies on the percent hatch of eggs recovered from the indicated compound-2 crosses*

Experiment	Parents: Female	Parents: Male	Total eggs	Mean % hatch	95 % confidence interval
1	SH3, + ;SH3, +	P,b ;P,px	3447	26·7	24·5–28·9
2	P,b ;P,px	SH3. + ;SH3, +	4062	29·9	28·4–31·5
3	SH3, + ;SH3, + In(3LR)TM3/ +	SH3. + ;SH3, +	4007	18·8	17·1–20·5
4	SH3, + ;SH3, +	SH3. + ;SH3, + In(3LR)TM3/ +	4919	27·3	26·1–28·6
5	SH3, + ;SH3, +	SH3. + ;SH3, +	4000	26·9	25·0–28·8

Since $C(2L)$ and $C(2R)$ segregate regularly in the female, our prediction is that if they segregate randomly in the male, then only 25 % of the zygotes will give rise to balanced, diploid individuals (see Holm & Chovnick, 1975 for detailed discussion). The remaining 75 % will be aneuploid for the compound autosomes and, as shown by Scriba (1967), these will die during embryological development. For each test, 25 to 30 females were crossed individually to three males in half pint creamers inverted over 60 mm plastic Petri plates for a total of 5–6 24 h broods – depending on the number of eggs collected. Scoring of eggs was initiated on the day following the first observed hatch. Total eggs were counted when the parents were transferred, and the hatched eggs (or unhatched eggs) were counted 36 h later. All parents (males as well as females) were collected as virgins and aged for 3 days prior to mating. The reported mean hatch and the 95 % confidence intervals (Table 2) were determined by using arcsin transformation values of individual results. In all cases, the weighted means were almost identical. The results presented in Table 2 are in general agreement with our model save for Expt 3 (analogous to Expt 3 of Table 1). In Expt 3 of Table 1 and 2, the compound-2 bearing female is heterozygous for *In(3LR)TM3* and a normal third chromosome and we believe that the increased frequency of progeny non-segregational for the compound-2 autosomes is due to non-homologous pairing with non-crossover third chromosomes. Although increasing the frequency of non-segregation of compound-2 autosomes in the female should not change the frequency of diplo-2 zygotes from 25 % (Holm & Chovnick, 1975: Holm, 1976), it should reduce the viability of this class owing to non-disjunction of the third chromosomes. This non-disjunction results in haplo- and triplo-3 zygotes among the diplo-2 class which will reduce the overall hatch

from 25%. It is evident from the results presented in Table 3 that only zygotes which usually give rise to viable adults hatch in compound-2 crosses.

3. RESULTS

(i) *The meiotic segregation of compound second autosomes bearing heterochromatic duplications*

A number of compound autosomes carrying heterochromatic duplications of a portion of the complementary arm have been constructed. For example, *C(2L)SH1,+* carries a duplication of *2R* extending from the centromere to a point

Table 3. *Progeny recovered from hatched eggs in five single day broods involving the cross* C(2L)SH1,+ ; C(2R)SH1,+ *mated to* C(2L)SH1,+ ; C(2R)SH1,+

Brood	Hatched eggs	Progeny recovered as adults	Recovered (%)
2	367	365	99·5
3	391	386	98·7
4	398	367	92·2
5	390	390	100·0
6	425	401	94·4
Total	1971	1909	96·9

distal to the rl^+ locus within the *2R* heterochromatin. Our previous analysis of the second chromosome proximal heterochromatin (Hilliker & Holm, 1975; Hilliker, 1976) sets limits on the extent of the duplications. *C(2L)SH1,+* and *C(2L)VH1,1t*, which bears rl^+ duplications of *2R*, are duplicated for at least half of the 2R heterochromatin and *C(2R)VK1,bw* is duplicated for most of the *2L* heterochromatin. Segregation was assayed by crossing males from selected compound-2 autosome-bearing strains to differentially marked compound-2 autosome-bearing females possessing a marked *Y* chromosome. These B^sY; *C(2L)P,b*; *C(2R)P,px* females give a high frequency of compound-2 autosome non-segregation (Table 1). Female gametes non-segregational for the compound-2 autosomes will result in a viable zygote only if fertilized by a sperm non-segregational for the paternal compound-2 autosomes. Thus, a strain in which compound-2 autosomes partially segregate in males when crossed to B^sY; *C(2L)P,b*; *C(2R)P,px* females will give a lower frequency of progeny completely matroclinous or patroclinous for the two compound-2 autosomes than will a strain in which *C(2L)* and *C(2R)* segregate at random in the male.

Therefore, males of the *C(2L)SH3,+ ; C(2R)SH3,+* strain, in which nearly equal frequencies of *C(2L)*; *C(2R)*; diplo-2; and nullo-2 sperm are produced, and of several other strains, in which one or both compound autosomes have heterochromatic duplications of the other arm, were crossed, singly, to B^sY; *(2L)P,b*; *C(2R)P,px* virgin females. The results are presented in Table 4.

Since no significant reduction in the frequency of non-segregational progeny is

Table 4. *Progeny of* B^sY; C(2L)P,b; C(2R)P,px *females and various compound-2 autosome-bearing males and the frequency of progeny non-segregational for compound-2*

	Chromosomes from mother									
	B^sY			B^sY		B^sY				
Male genotype	C(2L)P,b; C(2R)P,px	C(2L)P,b; C(2R)P,px	B^sY C(2R)P,px	C(2R)P,px	C(2L)P,b	C(2L)P,b		O	Total	Progeny non-segregational (%)
C(2L)SH3,+ C(2R)SH3,+	1	462	300	273	204	279	247	5	1771	40·4
C(2L)SH1,Dp(2R)rl$^+$ C(2R)SH1,+	1	567	395	365	286	383	447	4	2448	41·6
C(2L)SH1,Dp(2R)rl$^+$ C(2R)SH1,+	1	197	159	176	112	197	239	5	1086	40·7
C(2L)SH1,Dp(2R)rl$^+$ C(2R)SH3,+	0	57	39	46	31	47	44	0	264	38·3
C(2L)SH3,+ C(2R)VK1,Dp(2L)lt$^+$,bw	0	127	153	146	122	174	215	3	940	36·7
C(2L)VH1,Dp(2R)rl$^+$,lt C(2R)VK1,Dp(2L)lt$^+$,bw	1	122	127	102	80	146	163	1	742	38·7

shown by those crosses involving parental males carrying *C(2L)* and *C(2R)* chromosomes with extensive heterochromatic homology, we deduce that *heterochromatic* homology *per se* is not a major factor in the meiotic segregation of second chromosomes in male *Drosophila melanogaster*.

The *2L* and *2R* heterochromatin *per se* share significant DNA sequence homology, being major sites of localization of the 1·705 g/cc satellite sequence (Steffensen, Appels & Peacock, 1981). If we examine *C(2L)SH3,+* and *C(2R)SH3,+* for the 1·705 g/cc satellite by *in situ* hybridization (Fig. 1) we observe that, as expected, the heterochromatin of both compound autosomes contains the 1·705 g/cc satellite. Despite this homology, *C(2L)SH3,+* and *C(2R)SH3,+* exhibit random meiotic segregation during spermatogenesis.

(ii) *The influence of proximal euchromatin on second chromosome segregation during spermatogenesis*

At least one compound second autosome, *C(2R)cn*, exhibits appreciable non-random segregation relative to its complementary *C(2L)* during male meiosis (Sandler *et al.* 1968; Evans, 1971; Gethmann, 1976); this chromosome has a duplication of *2L* proximal euchromatin and thus may be duplicated for a *2L* euchromatic male meiotic pairing site. Yamamoto's (1979) cytological analysis of the meiotic segregation of *C(2R)cn* from a complementary *C(2L)* during male meiosis led him to conclude that segregation was random; however, the data in support of his conclusion are not presented in sufficient detail to allow critical evaluation.

We wish to report here additional observations which implicate chromosome-2 proximal euchromatin in male meiotic pairing. Compound-2 autosomes were generated in oocytes heterozygous for *In(2LR)SD72* and a structurally normal second chromosome. The synthesis of *C(2L)* and *C(2R)* chromosomes heterozygous for the left and right halves, relative to the centromere, of *In(2LR)SD72* is outlined in Fig. 2. The breakpoints of *In(2LR)SD72* lie in the *2L* proximal euchromatin and *2R* proximal euchromatin (Lewis, 1962). Hence, *C(2L)V12,SD72/+* is heterozygous for a proximal deficiency in *2L* and carries a duplication for *2R* proximal euchromatin. *C(2R)V43,SD72/cn bw* is heterozygous for deficiency in proximal *2R*, but carries a duplication of *2L* proximal euchromatin. Cytological analysis of *C(2L)V12,SD72/+*; *C(2R)V43,SD72/cn bw* demonstrates that this is the case and, indeed, further refines the cytological description of *In(2LR)SD72* reported by Ganetsky (1977) (Fig. 2). *C(2R)V43,SD72/cn bw* carries a duplication of *2L* proximal euchromatin extending to 39D3-4 of the *2L* polytene chromosome map (Bridges, 1943) and *C(2L)V12,SD72/+* has a duplication of *2R* proximal euchromatin extending to 42A of the *2R* polytene chromosome map (Bridges & Bridges, 1939). Moreover, analysis of somatic chromosomes demonstrated that *C(2R)V43,SD72/cn bw* is duplicated for the prominent secondary constriction at the *2L* heterochromatic–euchromatic junction.

The experiments documented in Table 5 indicate that *C(2L)V12,SD72/+* and

C(2R)V43,SD72/cn bw exhibit a high degree of meiotic segregation in males. In these experiments, *C(2L)V12,SD72/+*; *C(2R)V43,SD72/cn bw* males were crossed to compound-2 autosome-bearing females possessing, in addition, a supernumerary *Y* chromosome or an attached *X* chromosome in addition to the *Y*. *C(2L)SH3,+*; *C(2R)SH3,+* males when crossed to *C(1)RM/B^{s}Y; C(2L)P,b; C(2R)P,px* and to *B^{s}Y;C(2L)P,b; C(2R)P,px* females yield, respectively, an F_1 of 29·9 and 40·4% progeny that are non-segregational for compound-2. However, when *C(2L)V12,SD72/+*; *C(2R)V43,SD72/cn bw* males are crossed to these same females, the non-segregational progeny are reduced to 5·1 and 7·3% for *C(1)RM/B^{s}Y; C(2L)P,b; C(2R)P,px* and *B^{s}Y; C(2L)P,b; C(2R)P,px* parental females, respectively. The sixfold reduction in non-segregational progeny is consistent with a reduction in diplo-2 plus nullo-2 sperm from 50% to approximately 8% (see Holm, 1981). This is the consequence of 20% of male meioses failing to result in effective pairing between *C(2L)V12,SD72/+* and *C(2R)V43,SD72/cn bw*. Accordingly, we estimate that *C(2L)V12,SD72/+* and *C(2R)V43, SD72/cn bw* pair with 80% fidelity during male meiosis.

4. DISCUSSION

Unlike the *X* chromosome heterochromatin, the second chromosome heterochromatin does not influence male meiotic chromosomal segregation. The proximal euchromatin of chromosome-2 is implicated in male meiotic pairing. We thus infer that specific euchromatic regions are the sites of second chromosome male meiotic pairing.

The third chromosome may be similar in this regard. Holm (1969) and Holm & Chovnick (1975) found that all compound-3 autosomes analysed behaved as non-homologous chromosomes during male meiosis, exhibiting random segregation of *C(3L)* relative to *C(3R)*; whereas, in the sibling females, *C(3L)* regularly segregate from *C(3R)*. However, it is conceivable that pairing sites may lie within the third chromosome heterochromatin, but outside the limits of the duplications and deficiencies that can be tolerated by compound-3 bearing flies (see Holm, 1976). Yamamoto's (1979) analysis of meiotic pairing in males of the constitution *F(2L)/F(2L)*; *F(2R)/F(2R)*; *normal chromosome 3/3L^{D}2^{P}3R^D of T(2;3)108* was also instructive. The *3L^{D}2^{P}3R^D* component of *T(2;3)108* is a chromosome in which the centromere and proximal heterochromatin of chromosome 2 is substituted for the third chromosome centromere. [From its cytological and genetic description (Lindsley & Grell, 1968) it would appear highly probable that a portion of the third chromosome heterochromatin remains with the *3L^{D}2^{P}3R^D* element]. In 20 cells examined, neither *F(2L)* nor *F(2R)* paired with the *3L^{D}2^{P}3R^D* element, which paired with the normal third chromosome in these cells. Although the cytological assay may not detect low levels of non-disjunction, Yamamoto's results argue that neither the second nor the third chromosome heterochromatin can be solely determinative in the male meiotic pairing of these autosomes.

The involvement of heterochromatin in the meiotic pairing of the remaining

Table 5. *Recovery of compound-2 non-segregational progeny from crosses involving* C(2L)V12,SD72/+; C(2R)V43,SD72/cn bw *males and* C(1)RM/B^{s}Y; C(2L)P,b; C(2R)P,px *and* B^{s}Y; C(2L)P,b; C(2R)P,px *females and control crosses*

	Parents		Compound autosomes from mother					
			Segregational progeny		Non-segregational progeny			Non-segregational progeny (%)
Experiment	Female	Male	C(2L)P,b	C(2R)P,px	C(2L)P,b C(2R)P,px	O	Total progeny	
1	C(1)RM/B^{s}Y P,b;P,px	SH3,+;SH3,+	113	112	62	34	321	29·9
2	C(1)RM/B^{s}Y P,b;P,px	SH1,+;SH1,+	433	571	177	231	1412	28·9
3	C(1)RM/B^{s}Y P,b;P,px	V12,SD72/+; V43,SD72/cn bw	157	122	6	9	294	5·1
4	P,b;P,px B^{s}Y	SH3+;SH3,+	483	573	463	232	1771	40·4
5	P,b;P,px B^{s}Y	V12,SD72/+; V43,SD72/cn bw	115	101	4	13	233	7·3

autosome, the 'dot' or fourth chromosome, remains unclear. Yamamoto (1979) assayed cytologically meiotic pairing between $T(1;4)w^{m5}$ and a normal fourth chromosome during spermatogenesis. The IV^PX^D element of $T(1;4)w^{m5}$ failed to pair with the normal fourth chromosome, which about half the time paired with the X^PIV^D element. Yamamoto believed that all of the fourth chromosome heterochromatin remained with the IV^PX^D element (which is 'capped' with a small region of X chromosome distal euchromatin) and, hence, that its consistent failure to pair with the normal fourth chromosome argued strongly that the fourth chromosome heterochromatin contained no male meiotic pairing sites. However, we have cytologically examined $T(1;4)w^{m5}$ (Figure 3) and determined that the X^PIV^D element contains a large portion of the fourth chromosome heterochromatin, as indeed the variegation of the *ci* and *w* loci, associated with the rearrangement (Lindsley & Grell, 1968), would lead one to expect (see Spofford, 1976). In the light of these observations, Yamamoto's conclusion must be confined to the heterochromatin immediately flanking the fourth chromosome centromere; that is, the portion proximal to the fourth chromosome breakpoint associated with $T(1;4)w^{m5}$.

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EXPLANATION OF PLATES

PLATE 1

Fig. 1. *In situ* location of the 1·705 g/cc satellite in *C(2L)SH3,+* and *C(2R)SH3,+*. [^{3}H]cRNA synthesized from purified satellite DNA was hybridized to the mitotic chromosomes from *C(2L)SH3,+*; *C(2R)SH3,+* female larvae.

Fig. 3. Mitotic chromosomes of a $T(1;4)w^{m5}$ bearing male. The larger, $X^P IV^D$, element of the translocation has two heterochromatic regions. The larger of the two blocks is the undisturbed *X* chromosome heterochromatin and the smaller is derived from chromosome four.

Plate 2

Fig. 2. Synthesis of *C(2L)SD72/+* and *C(2R)SD72/+* and the cytological description of *In(2LR)SD72*. *C(2L)V12,SD72/+* and *C(2R)V43,SD72/cn bw* were synthesized by treating *In(2LR)SD72/cn bw* females with 2500 rads of gamma radiation. It should be noted that the *In(2LR)SD72* chromosome used in this study showed normal SD effects and normal disjunction in males (Sharp, 1975). Further, the combination of SD-bearing compound autosomes analysed does not show any SD effects; however, *C(2R)V43,SD72/cn bw* does show an SD effect in combination with some other *C(2L)* chromosomes (Holm, unpublished results).

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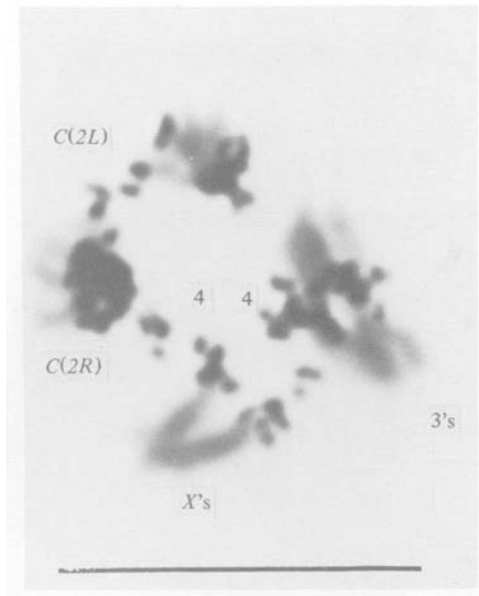

Fig. 1

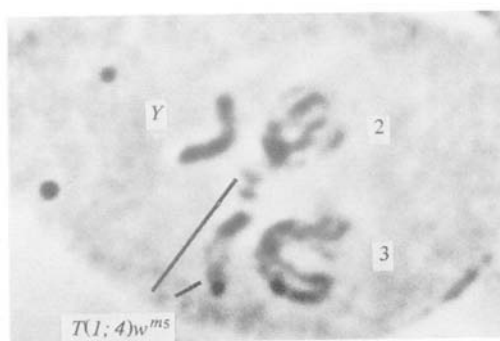

Fig. 3

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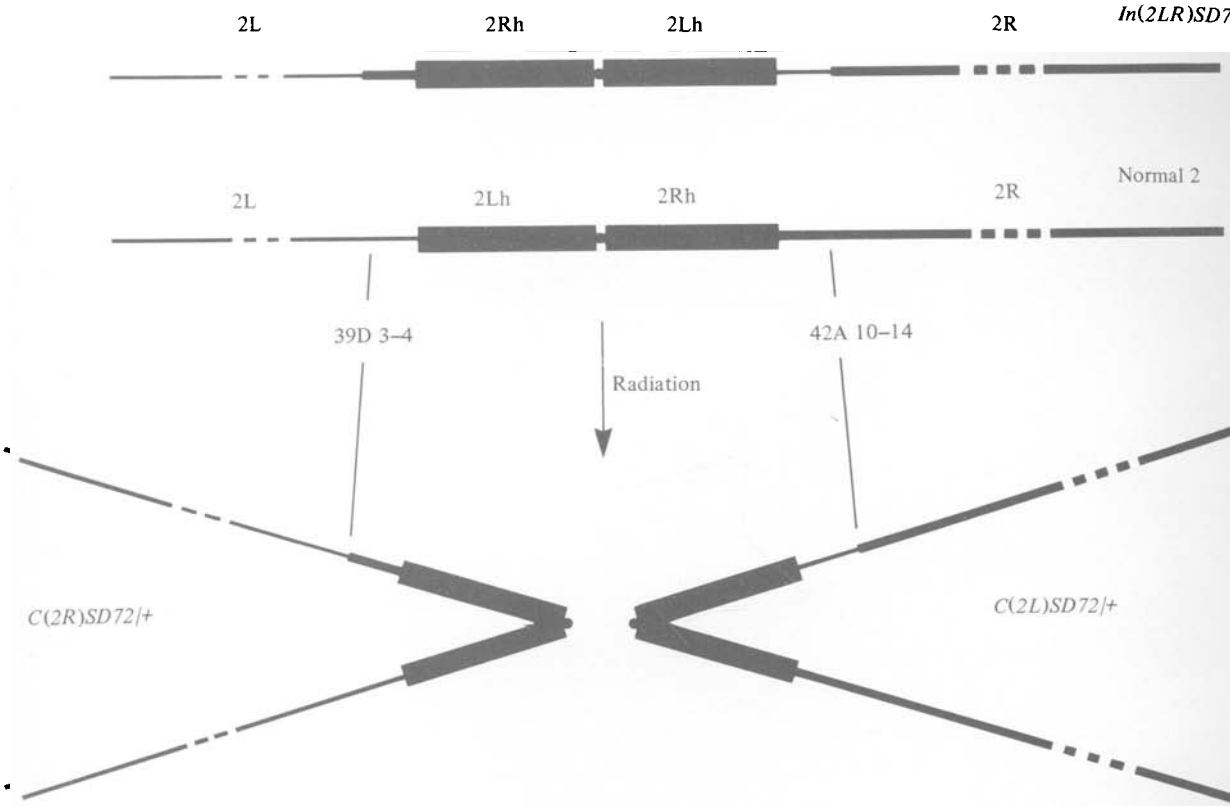

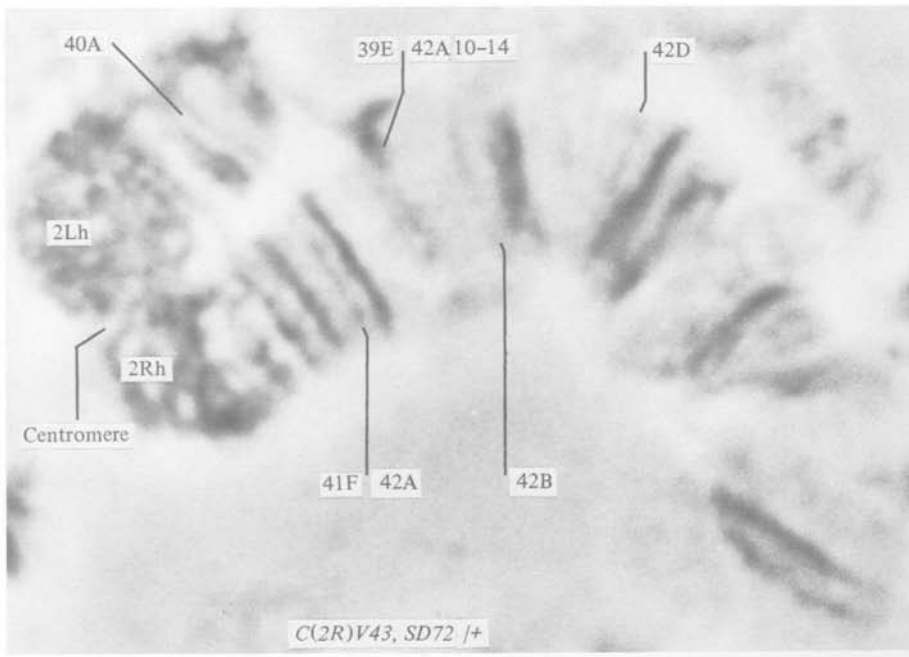

Fig. 2

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