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Studies on male recombination in a Southern Greek *Drosophila melanogaster* population

(*c*) Chromosomal abnormalities at male meiosis
(*d*) Cytoplasmic factor responsible for the reciprocal cross effect

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SUMMARY

A combined cytological and genetic analysis has been carried out to determine whether the spontaneous male recombination associated with a line of *Drosophila melanogaster* derived from Southern Greece (31.1 MRF) involves chromosome breakage and random reunion. In all crosses showing male recombination, extensive abnormalities involving anaphase bridges and fragments were found at first and second meiotic divisions. This confirms that the low level of recombination associated with male recombination lines is not produced by normal, controlled crossing-over of the type found in females, but by chance reunions, following more erratic breakage events at first and second anaphase. In addition the occurrence of some limited premeiotic recombination cannot be excluded. The *31.1 MRF* system shows higher recombination levels at elevated temperatures (29 °C) and chromosome abnormality frequency is also greatly increased. The *31.1 MRF* induces both male recombination and chromosomal abnormalities at meiosis when it is inherited from females deriving their cytoplasm from stock other than CyL^4/Pm. It is concluded that this factor acts independently of sex and that the reciprocal cross effect is caused by a cytoplasmic factor which the $31.1/CyL^4$ strain inherited from the CyL^4/Pm stock.

INTRODUCTION

A number of species belonging to the genus *Drosophila* regularly show male recombination, including *D. ananassae* (for a review see Moriwaki & Tobari, 1975), *D. simulans* (Woodruff & Bortolozi, 1976), *D. subobscura* (Philip, 1944), *D. virilis* (Kikkawa, 1935) and *D. willistoni* (Franca, DaCunha & Garrido, 1968), but in *D. melanogaster* the frequency is usually close to zero. However, Hiraizumi (1971) first reported that a low, but significant, level of spontaneous male recombination could also occur in *D. melanogaster*. Since then its occurrence has been confirmed and studied by many investigators (Voelker, 1974; Waddle & Oster, 1974; Yamaguchi & Mukai, 1974; Broadwater *et al.* 1973; Cardellino & Mukai, 1975; Kidwell & Kidwell, 1975*a*, *b*; Sved, 1974; Matthews & Hiraizumi, 1976;

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Yamaguchi, 1976; Woodruff & Thompson, 1977; Yannopoulos & Pelecanos, 1977; Green, 1977).

As it would appear that a limited amount of male recombination may occur in natural populations of *D. melanogaster*, it is of interest to identify the mechanism(s) involved and the genetic basis of this process. It is also important to determine what effect(s) these factors may have in natural populations.

Although Philip (1944) observed bridges and bridges + fragments associated with male recombination within heterozygous inversions in spermatocytes of *D. subobscura*, Hinton & Downs (1975) failed to find the same phenomenon in *D. ananassae* males showing male recombination. It has been postulated, on genetic grounds (Slatko & Hiraizumi, 1973; Voelker, 1974; Yamaguchi, 1976) that chromosome breakage is associated with male recombination in *D. melanogaster*. Recently, Henderson, Woodruff & Thomson (1977) found extensive chromosome breakage at first and second meiotic anaphase in crosses involving a male recombination line from Oklahoma, designated OK1.

It has been found (Yannopoulos & Pelecanos, 1977) that a second chromosome (symbol 31.1 MRF) isolated from a wild *D. melanogaster* population in North-Western Peloponnesus (at a distance of 8 km from the city of Patras, Greece) induces male recombination both in chromosomes 2 and 3, and that the phenomenon is temperature-sensitive in larval stages. In addition, male recombination induced by this chromosome was suppressed by a cytoplasmic factor from the CyL^4/Pm stock.

The aim of the present study was to investigate whether male recombination induced by the 31.1 chromosome occurs by classical exchange as in females, or by chromosome breakage and random reunion. If male recombination occurs at meiosis by chromosome breakage and by chance reunion, the breaks could occur at different positions within the two exchange chromatids. This might result in both normal recombinants and recombinants with deficiencies and/or duplications. Furthermore, chromosomal abnormalities such as bridges and fragments may be visible in meiotic anaphases. Were chromosome breakage to occur premeiotically then chromosome abnormalities might be visible during premeiotic mitosis or at meiotic metaphase I and early anaphase I.

MATERIAL AND METHODS

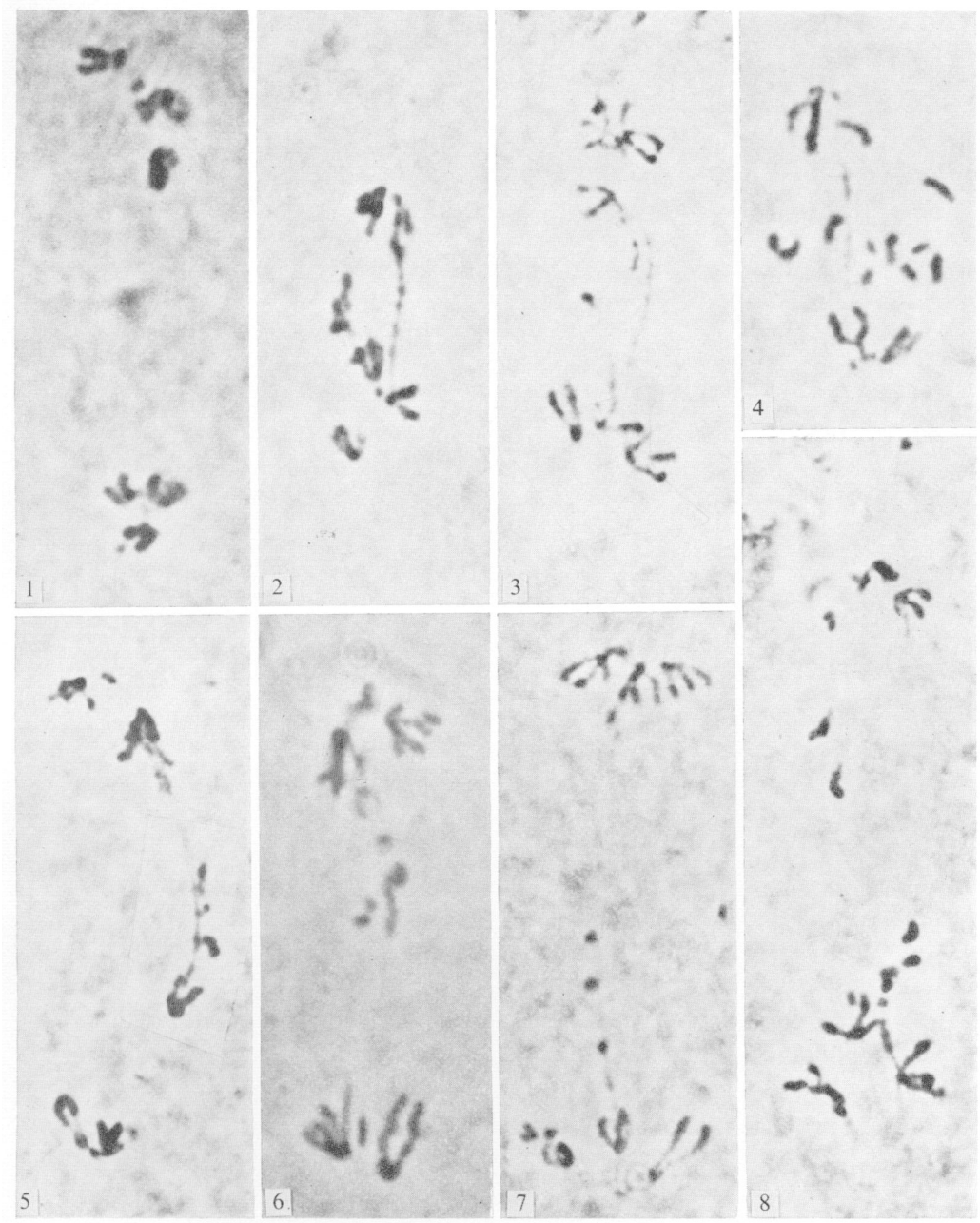
The following strains of *Drosophila melanogaster*, maintained at $25 \pm \frac{1}{2}$ °C were used:

(1) *dp b cn bw*; *ve*. A standard stock marked with five recessive mutants, *dp* (dumpy wings, 2L-13), *b* (black body colour 2L-48.5), *cn* (cinnabar eye colour 2R-57.5), *bw* (brown eyes 2R-104.5) and *ve* (veinlet wings 3L-0.2).

(2) *dp b cn bw*. As (1) but lacking veinlet.

(3) A balanced stock Sp/SM5, al^2 *Cy* lt^v sp^2 (for description see Lindsley & Grell, 1968).

(4) Canton-S (Canton-Special) a wild type stock.



Anaphase I. (1) Normal, (2) bridge, (3) bridge + fragment, (4) bridge + fragments, (5) bridge + fragments stuck on the bridge, (6) broken bridge + fragments, (7) and (8) fragments. (1) and (6) × 2000; 2, 3, 4, 5, 6 and 7 × 1600.

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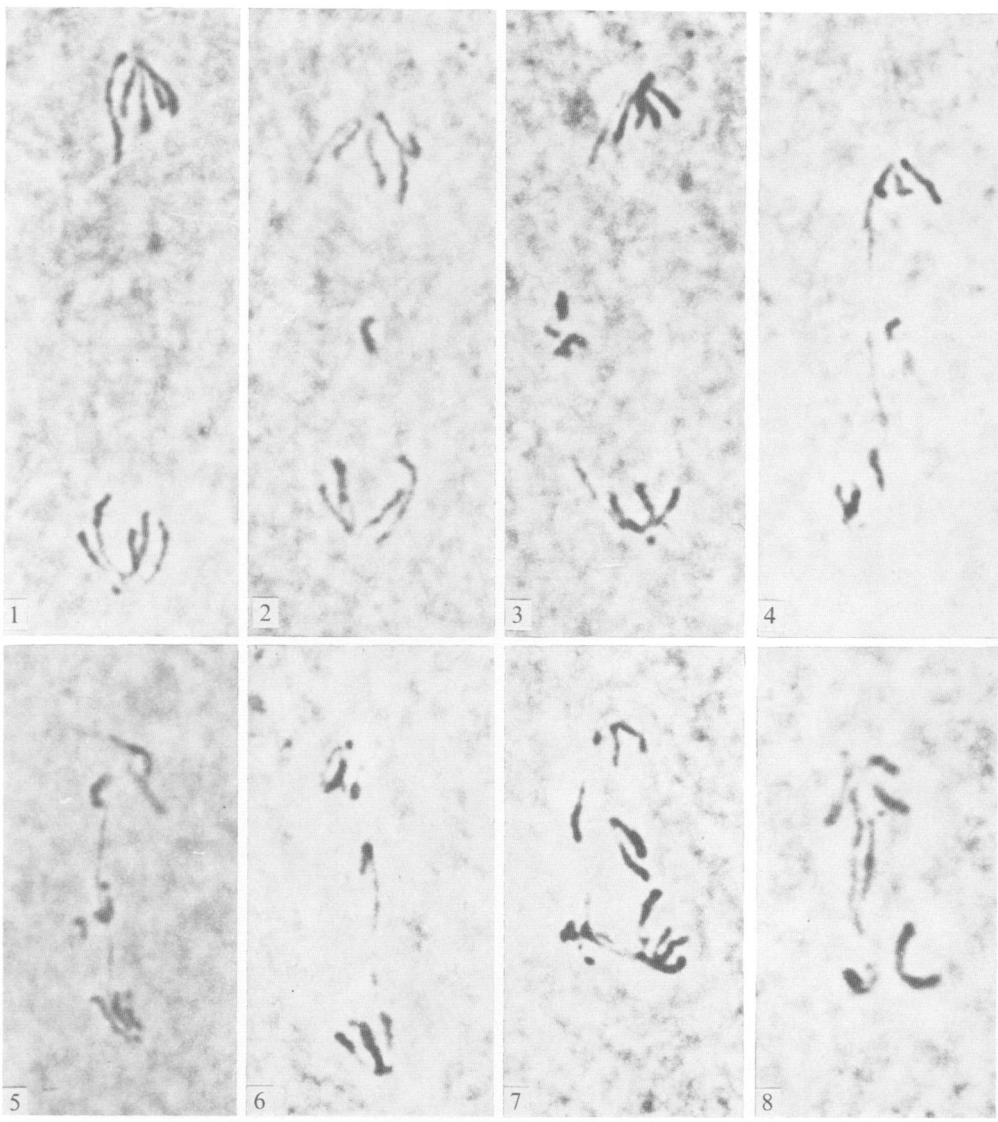

Anaphase II. (1) Normal, (2) fragment, (3) fragments, (4) bridge + fragment, (5) bridge + fragments, (6) bridge, (7) broken bridge + bridge between chromosomes moved to the same pole, (8) two bridges. × 1600.

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(5) A second-chromosome line (symbol *31.1* MRF) isolated from a Southern Greek *D. melanogaster* population in the autumn of 1971 by the CyL^4/Pm method. This chromosome is inversion-free and is kept balanced with the In (2L+2R) *Cy*, CyL^4sp^2 chromosome as it is homozygous lethal.

An 'Instant' *Drosophila* medium (Philip & Harris) was used throughout the present experiments. Parents were 2 or 3 days old when the crosses were set up and progeny were scored until the 17th day after setting up matings. The cultures were maintained in vials kept at $25 \pm \frac{1}{2}$ °C or $29 \pm \frac{1}{2}$ °C according to the experiments (see Results). Pupae prior to eye pigment formation were used for testes preparations since in this stage many meiotic divisions occur (see Cooper, 1956). Testes were dissected in insect saline and squash preparations were stained with propionic carmine orcein (PCO). All observations were made on fresh preparations. Meiotic anaphase I and II were scored separately according to whether they were cytologically normal or showed bridges, fragments or bridges + fragments.

As high clusters of recombinants were not observed throughout the present experiments two estimates of male recombination were calculated. (1) Male recombination frequency; total number of recombinants × 100/total number of progeny. (2) Number of males that produced recombinants; number of males with at least one recombinant progeny.

RESULTS

Extensive cytological abnormalities were observed during meiosis of males showing male recombination. These abnormalities included bridges and fragments at both the first and second meiotic divisions. They are illustrated in Plates I and II.

Parallel genetic and cytological tests were made on males of various genotypes to determine the association between the *31.1* MRF chromosome and both male recombination and abnormal chromosome behaviour during meiosis (Tables 1 and 2). In cross 1 virgin *dp b cn bw*; *ve* females were mated to $31.1/CyL^4$ males (G1) (mass culture) and, after allowing egg laying to occur for 3 days, the parents were discarded. Then five vials from this cross (G1) were used to collect male pupae for cytological analysis, while the other five were used to collect *31.1/dp b cn bw*; *ve*/+ males. These males were then individually mated to *dp b cn bw*; *ve* virgins (G2) and their progeny were scored for recombinants. The male recombination frequency was found to be 2·90 % (cross 1, Table 1) and the frequency of chromosomal abnormalities in anaphase I and II was 22·1 and 25·6 % respectively (cross 1, Table 2). In order to test whether the G1 *31.1/dp b cn bw*; *ve*/+ sons induce the same frequency of abnormalities in anaphase I and II as their parents, five G2 vials were used to collect pupae for testis preparations. From the G2 adults, which were scored for recombinants, *31.1/dp b cn bw* males were also collected at random and were backcrossed to *dp b cn bw*; *ve* virgins (G3). The individuals of this cross were also scored for recombinants. The results (cross 5,

Table 1. *Second chromosome male recombination in* 31.1/dp b cn bw; ve/+ (*crosses 1, 2, 3, 5, 7*), 31.1/dp b cn bw (*crosses 4, 6*) *and* C-S/dp b cn bw; ve/C-S (*cross 8*) *males. Crossover regions* 1 dp-b, 2 b-cn, 3 cn-bw

Sex of parent	Temperature (°C)	No. of males tested	No. of progeny	No. of recombinants by region						Male recombination frequency (%)	No. of males that produced recombinant (%)	Average k†
				1	2	3	1,2	1,3	2,3			
(1) 31.1/CyL^4 ♂	25	31	6490	21	52	112	0	0	3	2·90	31 (100)	0·45
(2) 31.1/CyL^4 ♀	25	30	5277	0	1	2	0	0	0	0·06	3 (10)	0·57
(3) 31.1/Cy ♀ Cytopl. Cy/Sp	25	20	3805	19	40	62	1	0	0	3·21	19 (95)	0·45
(4) 31.1/CyL^4 ♂	25	23	3671	9	24	74	0	0	0	2·91	23 (100)	0·42
(5) 31.1/dp b cn bw; ve/+ ♂ from cross 1	25	21	2979	19	21	39	0	0	2	2·72	19 (90)	0·45
(6) 31.1/dp b cn bw ♂ from cross 4	25	22	3889	18	31	61	0	0	5	2·96	21 (95)	0·42
(7) 31.1/CyL^4 ♂	29 → 25*	29	2488	11	40	43	0	0	2	3·86	27 (93)	0·45
(8) C-S ♂	25	35	10450	0	0	2	0	0	0	0·02	2 (5·7)	0·57

* Until puparium formation at first temperature and until eclosion at second.

† k, The proportion of wild-type amongst total progeny (excluding crossovers).

Table 2. *Chromosomal abnormalities at meiosis induced by 31.1 MRF*

Type of mating*	No. of progeny†				Total freq. of abnormal (%)	Total no. examined	No. of pupae screened
	Normal	Br (%)	Fr (%)	Br+Fr (%)			
			Anaphase I				
(1) dp b cn bw; ve × 31.1/CyL^4 ♂	74	9 (9·5)	5 (5·3)	7 (7·4)	22·1	95	82
(2) 31.1/CyL^4 × dp b cn bw; ve ♂	107	0	0	0	0	107	45
(3) 31.1/Cy × dp b cn bw; ve ♂	74	6 (6·2)	8 (8·3)	8 (8·3)	22·9	96	36
(4) dp b cn bw × 31.1/CyL^4 ♂	86	8 (6·8)	9 (7·7)	14 (12·0)	26·5	117	48
(5) dp b cn bw; ve × 31.1/dp b cn bw; ve/+ ♂ (♂♂ from cross 1)	62	6 (7·7)	7 (9·05)	3 (3·8)	20·5	78	46
(6) dp b cn bw × 31.1/dp b cn bw ♂ (♂♂ from cross 4)	55	5 (7·0)	5 (7·0)	6 (8·4)	22·5	71	48
(7) dp b cn bw; ve × 31.1/CyL^4 ♂	48	12 (10·3)	38 (32·8)	18 (15·5)	58·6	116	49
(8) Stock CyL^4/31.1	98	0	0	0	0	98	45
(9) 31.1/Cy (♂♂ and ♀♀ from cross 10)	59	7 (7·5)	16 (17·2)	11 (11·8)	36·5	93	48
(10) Cy/Sp × 31.1/CyL^4 ♂	87	6 (6·1)	3 (3·0)	3 (3·0)	12·1	99	87
(11) Cy/Sp × 31.1/CyL^4 ♂	104	2 (1·8)	5 (4·3)	5 (4·3)	10·4	116	39
(12) dp b cn bw; ve × C-S ♂	107	0	0	0	0	107	35
(13) Cy/Sp × dp b cn bw; ve ♂	111	0	0	0	0	111	37
			Anaphase II				
(1) dp b cn bw; ve × 31.1/CyL^4 ♂	166	29 (13)	20 (9·0)	8 (3·6)	25·6	223	82
(2) 31.1/CyL^4 × dp b cn bw; ve ♂	126	0	0	0	0	126	45
(3) 31.1/Cy × dp b cn bw; ve ♂	125	22 (13·1)	8 (4·2)	10 (6·1)	24·2	165	36
(4) dp b cn bw × 31.1/CyL^4 ♂	156	16 (7·5)	25 (11·7)	16 (7·5)	(26·8)	213	48
(5) dp b cn bw; ve × 31·1/dp b cn bw; ve/+ ♂ (♂♂ from cross 1)	176	9 (4·5)	9 (4·5)	4 (2·0)	11·1	198	46
(6) dp b cn bw × 31.1/dp b cn bw ♂ (♂♂ from cross 4)	142	5 (3·0)	17 (10·4)	0	13·4	164	48
(7) dp b cn bw; ve × 31.1/CyL^4 ♂	96	48 (23·2)	31 (15·0)	32 (15·5)	53·6	207	49
(8) Stock CyL^4/31·1	127	0	0	0	0	127	45
(9) 31.1/Cy (♂♂ and ♀♀ from cross 10)	103	27 (17·6)	15 (9·8)	8 (5·2)	32·7	153	48
(10) Cy/Sp × 31.1/CyL^4 ♂	165	17 (8·1)	17 (8·1)	10 (4·8)	21·0	209	87
(11) Cy/Sp × 31.1/CyL^4 ♂	127	18 (11·2)	11 (6·9)	4 (2·5)	20·6	160	39
(12) dp b cn bw; ve × C-S ♂	198	0	0	0	0	198	35
(13) Cy/Sp × dp b cn bw; ve ♂	176	0	0	0	0	176	37

* All treatments were kept at 25 °C with exception of no. 7 (29 → 25 °C see text). † Br, Bridge; Fr, fragment(s).

Table 1) show that the frequency of male recombination (2·72 %) was slightly lower than that in cross 1, but, while the frequency of chromosomal abnormalities in anaphase I was almost the same (20·5 %) as in cross 1, the frequency of chromosomal abnormalities at anaphase II was about half (11·1 %).

Exactly the same procedure was used with another stock, *dp b cn bw*. As can be seen (cross 4 (G1) and cross 6 (G2), Tables 1 and 2) the results are similar to those with *dp b cn bw*; *ve* stock both with respect to male recombination frequencies and the frequency of chromosomal abnormalities at anaphase I and II.

No chromosomal abnormalities were found at meiosis in the *31.1/CyL*^{4} stock (cross 8, Table 2) nor when virgin *31.1/CyL*^{4} females from the stock were mated to *dp b cn bw*; *ve* males (cross 2, Table 2). The frequency of male recombination was also very low in the latter cross (cross 2, Table 2).

It has been found (Yannopoulos & Pelecanos, 1977) that the cytoplasm of a *CyL*^{4}*/Pm* stock suppressed recombination in heterozygous males that inherited the *31.1* chromosome from males. In order to test whether the *31.1* chromosome induces male recombination, as well as chromosomal abnormalities in meiosis, when it is inherited from females that derive their cytoplasm from a stock other than the *CyL*^{4}*/Pm* strain, virgin *Cy/Sp* females were mated to *31.1/CyL*^{4} males. Virgin *31.1/Cy* females from this cross were mated to *dp b cn bw*; *ve* males. Twenty of the vials from this cross were used to collect pupae for testis preparations and 20 to collect *31.1/dp b cn bw*; *ve*/+ males. These males were backcrossed to *dp b cn bw*; *ve* virgins and their progeny were scored for recombinants. The results are shown in cross 3 (Tables 1 and 2). The male recombination frequency was found to be slightly higher (3·21 %) than in crosses 1 and 4, while the frequency of chromosome abnormalities at both anaphase I and II were similar to that seen in cross 1 (22·9 and 24·2 %, respectively). These results clearly demonstrate that the action of *31.1* MRF is independent of the sex from which it is inherited. Furthermore, when virgin *31.1/CyL*^{4} females and males from cross 10 (Table 2) (*Cy/Sp* × *31.1/CyL*^{4}) were crossed together, the frequencies of chromosomal abnormalities at anaphase I and II in their male progeny were 36.6 and 32·7 %, respectively (cross 9, Table 2).

Testes from male pupae from cross 10 (Table 2) were also examined for chromosomal abnormalities at meiosis. In this case the frequency of chromosome abnormalities at anaphase II was about the same (21.1 %) as in crosses 1 and 3 (Table 2), but the frequency in anaphase I was nearly half that in anaphase II. Because of this marked difference in frequency of chromosome abnormalities at anaphase I and II, this experiment was repeated (cross 11, Table 2) with similar results. Unfortunately the genotype of this cross did not allow one to score for male recombination.

It has been shown that *31.1* MRF is temperature sensitive in larval stages (Yannopoulos & Pelecanos, 1977). In order to detect whether larvae kept at 29 °C would give higher frequencies of abnormalities at meiosis, *31.1/CyL*^{4} males were crossed to *dp b cn bw*; *ve* virgins (25♀ × 25♂) and the females were allowed to lay eggs at 25 °C for 4 h. After 20 h the vials were kept at 29 °C until the first

pupae formed. These pupae were discarded and the vials returned to 25 °C. Pupae from five vials were used for testis preparations while the *31.1/dp b cn bw*; *ve*/+ sons from the other five vials were individually mated to *dp b cn bw*; *ve* virgins and their progeny scored for recombinants. The results are given in cross 7 (Tables 1 and 2). The frequency of chromosomal abnormalities at anaphase I and II was 58·6 and 53·6 %, respectively, which is much higher than in cross 1. The male recombination frequency was also higher than in cross 1, ($\chi^2 = 5{\cdot}43$, DF = 1, $P < 0{\cdot}05$), although the increase in meiotic abnormalities and male recombination are not directly proportional.

Attempts to test whether the *31.1/CyL*^{4} males mated to C-S virgins could induce chromosomal abnormalities in spermatocytes failed as the pupal testes were atrophic. No chromosomal abnormalities were found at meiosis when virgin *dp b cn bw*; *ve* females were crossed to C-S males (cross 12, Table 2). Moreover, heterozygous *C-S/dp b cn bw*; *C-S/ve* from this cross individually backcrossed to *dp b cn bw*; *ve* virgins, showed very low male recombination frequency, i.e. 0·02 % (cross 8, Table 1). Chromosomal abnormalities were also not observed at meiosis *Cy/Sp* virgins were mated to *dp b cn bw*; *ve* males (cross 13, Table 2).

In addition to abnormalities during meiosis of males showing recombination as detected above, a very low frequency of abnormal premeiotic mitotic anaphases have also been observed, i.e. 1 % (4 abnormal mitosis amongst 389 screened).

DISCUSSION

The present study clearly shows that the *31.1* MRF second chromosome results in chromosomal abnormalities (bridges and fragments) both in first and second meiotic divisions. Whenever male recombination was observed, chromosome abnormalities were also found at meiosis. This implies that the male recombination induced by the *31.1* MRF system does not occur by the same mechanism as female recombination, that is, by classical crossing-over, but is the consequence of chance reunions following more erratic chromosome breakage during male meiosis. However, the occurrence of some limited premeiotic recombinants cannot be ignored. These results, being similar to those found with the OK1 male recombination system (Henderson *et al.* 1977), suggest that these two systems act in the same way.

It has been found (Yannopoulos & Pelecanos, 1977) that male recombination was suppressed by a cytoplasmic factor from the *CyL*^{4}*/Pm* stock. The results of cross 3 (Tables 1 and 3) demonstrate that *31.1* MRF in the absence of the cytoplasm of *CyL*^{4}*/Pm* stock, does induce both male recombination and chromosome abnormalities at meiosis when inherited maternally. This indicates that its action is independent of sex and that the reciprocal cross effect (cross 2, Table 2) is caused by a cytoplasmic factor that the *31.1/CyL*^{4} strain inherited from the *CyL*^{4}*/Pm* stock. This is also confirmed by the results of cross 9 (Table 2). The absence of chromosomal abnormalities in the *31.1/CyL*^{4} strain (cross 8, Table 2) must also be due to the cytoplasm of the *CyL*^{4}*/Pm* stock. These results are in

contrast to those of Kidwell, Kidwell & Ives (1977) who suggested that the cytoplasmic effect may more readily be explained in terms of maternally influenced inheritance rather than true extrachromosomal transmission. Moreover, Sved (1976) suggested that, for hybrid dysgenesis to occur (male recombination and female sterility), it is necessary for the wild type chromosomes to be contributed by the male parent. Furthermore, Woodruff & Thompson (1977) did not find cytoplasmic suppression of male recombination in the OK1 stock. Recently Kidwell, Kidwell & Sved (1977) suggested that the most compelling general hypothesis to explain non-reciprocality is that hybrid dysgenesis is dependent on cytoplasm–chromosome interaction between different strains.

The data of cross 7 (Tables 1 and 2) confirm previous findings (Yannopoulos & Pelecanos, 1977) that the *31.1* MRF system is temperature-sensitive in larval stages and shows a relationship, although not direct proportionality, between male recombination frequency and the percentage of chromosome abnormalities at meiosis. Since larval stages contain only premeiotic cells (Cooper, 1965) with meiosis beginning in the pupae, the *31.1* temperature-sensitive period in larval life does not affect meiosis directly. It must have an indirect action (e.g. multiplication of virus?) which later finds its expression in meiotic cells in the pupae.

The results provided in Table 1 show that, in all crosses kept at 25 °C, with the exception of cross 2, male recombination frequencies were similar. However, the frequencies of chromosome abnormalities at anaphase I and II were more variable (Table 2) and a linear relationship was not found. The lower transmission of the *31.1* chromosome observed (defined as k) may be due to the preferential breakage of the *31.1* chromosome 2 in the first and second meiotic divisions.

What is the cause of the chromosome breakage observed? It has been postulated that episomes (Voelker, 1974; Waddle & Oster, 1974) or viruses (Roberts, 1976; Green, 1977) may be responsible for the induction of male recombination. Yamaguchi (1976) has also suggested that flies showing male recombination may be mutant genotypes which are deficient in repair enzymes that can repair breaks which normally occur in all genomes. Experiments carried out with the OK1 system have shown that the property of causing male recombination is infective in the sense that it can be transmitted by the injection of cell-free extracts (Sochacka & Woodruff, 1976). The present results seen to be similar to the previous one reported as regards the induction of chromosome breakage by viruses of mycoplasm infections (for a review see Nickols, 1974). These findings combined with the results of other experiments (G. Yannopoulos, in preparation) which have shown that all properties of *31.1* MRF may be transmitted to other homologous chromosomes, constitute evidence favouring the explanation that the '*31.1* factor' is a virus or an episome. However, it is clear from both the present and previous results (Yannopoulos & Pelecanos, 1977) that more than one factor is involved in the ultimate expression of the effect. For besides the existence of the inducer factor *31.1*, which is linked to chromosome 2, there is another, cytoplasmic, factor which suppresses the activities of the inducer. It would be of interest to identify the nature and mode of action of this suppressor factor.

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