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Comparative studies of two male recombination factors (MRF) isolated from a Southern Greek *Drosophila melanogaster* population

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SUMMARY

A comparative study of two male recombination factors (31 . 1 MRF and 23 . 5 MRF) isolated from the same Southern Greek natural population, revealed specific differences in their activities. 23 . 5 MRF induces female sterility due to atrophic ovaries at a wide range of temperatures while 31 . 1 MRF does so only at high temperatures. The gross morphology of the atrophic ovaries was the same and unilaterally affected pairs were found in the F_1 of crosses with both 23 . 5 and 31 . 1 MRF. Furthermore, 23 . 5 MRF induces: (*a*) lower frequencies of abnormal anaphases I and II than 31 . 1 MRF, (*b*) higher frequencies of 'double crossovers' resulting from deficiences or duplications, (*c*) large clusters of recombinants, suggesting premeiotic origin and (*d*) cases where one of the non-recombinant phenotypes was not produced. Such cases have never been observed with 31 . 1 MRF. Moreover, the cytoplasm of the *Cy* L^4/Pm strain that suppresses 31 . 1 MRF does not affect the activities of 23 . 5 MRF. Hypotheses to explain the different behaviour of the two factors are presented and discussed.

1. INTRODUCTION

Studies on male recombination factors (MRF) all over the world seem to suggest that, up to now all these factors isolated from natural populations possess more or less similar properties. However, we still do not know whether the factors studied are identical. Of course, some differences have been reported but these could result from either differences in the genetic background (e.g. suppressors), or from different experimental conditions; however, specific differences in the structure of male recombination factors cannot be excluded.

In 1971, a second chromosome (31 . 1) was isolated from a large natural population of Southern Greece (Yannopoulos & Pelecanos, 1977). The chromosome was found to bear an MRF factor (31 . 1 MRF). Among other properties 31 . 1 MRF was able to induce: (1) male recombination in both the second and third chromosomes; the phenomenon is temperature sensitive in larval stages (Yannopoulos & Pelecanos, 1977), (2) chromosome breakage during male meiosis (Yanno-

poulos 1978*a*), (3) female and male sterility due to the inability of the ovaries and testes to complete their normal development (Yannopolos, 1978*c*) and (4) high frequencies of chromosome rearrangements in both males and females (Yannopoulos & Zacharopoulou, 1980). Furthermore, it was shown that the cytoplasmic factor responsible for the reciprocal cross effect could be progressively acquired when 31 . 1 MRF is introduced by outcrossing into the cytoplasm of a laboratory strain (Yannopoulos, 1978*b*). 31 . 1 MRF also possesses the ability to be transposed to another chromosome (Yannopoulos, 1979).

Wild chromosomes isolated recently from the same natural population in Southern Greece have shown the ability to induce male recombination (Stamatis, 1979). One lethal second chromosome (23 . 5) bears another MRF factor (23 . 5 MRF) (see Materials and Methods) which has shown specific differences in its activities from those of 31 . 1 MRF.

In the present study we have compared 31 . 1 MRF and 23 . 5 MRF mainly as regards: (*a*) female sterility due to atrophic ovaries, (*b*) male recombination and (*c*) the induction of abnormal meiotic divisions.

2. MATERIALS AND METHODS

The following strains of *Drosophila melanogaster* maintained at 25 ± 0·5 °C were used. (For a description of mutants and balanced strains, see Lindsley & Grell, 1968.)

(1) A second and a third chromosomal line homozygous, respectively, for *dp b cn bw* and *ve*.

(2) Canton-S (Canton-Special) a wild type strain.

(3) A multibalanced strain *M-5*; *Cy/Pm*; *Ubx/Sb* = | In (1) $sc^{S1L}sc^{8R} + S\ sc^{S1}\ sc^{8}\ w^{a}\ B$; In(2LR)SM1, $al^{2}\ Cy\ cn^{2}\ sp^{2}$; In(3LR)$Ubx^{130}$, $Ubx^{130}\ e^{s}$|.

(4) A balanced strain for the 1st and 3rd chromosome M-5; *dp b cn bw*; Ubx^{130}/Sb.

(5) A balanced strain CyL^{4}/Pm = In(2L + 2R) *Cy*, $L^{4}\ sp^{2}$

(6) 31 . 1/*Cy* L^{4}, a second chromosome line (symbol 31 . 1) bearing the 31 . 1 MRF in both the 2nd and 3rd chromosomes (Yannopoulos, 1978*a*; Yannopoulos, 1979).

(7) 23 . 5/CyL^{4}, a second chromosome line (symbol 23 . 5) bearing the 23 . 5 MRF and established as follows: Sons of a wild caught female (no. 23) were mated with virgin *dp b cn bw*; *ve* males. Heterozygous 23 . 5/*dp b cn bw*; 23 . 5/*ve* (F_1) male progeny were then individually mated to virgin *dp b cn bw*; *ve* females. One F_1 male (no. 5) yielded recombinants among its progeny. Heterozygous 23 . 5/*dp b cn bw*; *ve* males derived from this male were individually backcrossed to virgin *dp b cn bw*; *ve* females for five generations. In all five backcrosses, recombinants were found among the progeny. In order to have the 23 . 5 chromosome II in a similar genetic background to that of 31 . 1, we have isolated the 23 . 5 chromosome by the CyL^{4}/Pm method (the CyL^{4}/Pm strain used is the original one with 31 . 1 chromosome II isolated from one wild male (Yannopoulos, 1978*a*). The absence of + / + flies among the F_3 offspring showed that the 23 . 5 chromosome carries a recessive lethal gene(s). F_3 males and females 23 . 5/CyL^{4} were then intercrossed to establish the 23 . 5/CyL^{4} strain used in this study.

A standard cornmeal food was used throughout the present experiments. Parents were 2–3 days old when crosses were set up. In the male recombination tests, progeny were scored until the 17th day after commencement of matings. The cultures were kept in 18, 25 and 28 °C incubators, according to the experiments (see results).

The process of the collection of female progeny and the examination of their ovaries is described elsewhere (Yannopoulos, 1978*c*). Ovaries without any egg chambers were classified as atrophic (A), and those with at least one egg chamber, including those up to one third of the normal size, as reduced (R), all the others were considered to be normal (N). In Table 1 the females with only one ovary atrophic are designated as A- while those having both ovaries atrophic as AA. The reduced ovaries are included with the normal. Whenever we mention 'female sterility' below we refer to females having both ovaries atrophic (AA).

For the examination of meiotic anaphases I and II, testes from pupae prior to eye pigment formation were dissected in insect saline; squash preparations were stained with propionic carmine orcein (PCO) (for more details see Yannopoulos, 1978*a*). All observations were made on fresh preparations. Meiotic anaphases I and II were scored separately according to whether they were cytologically normal or showed bridges and/or fragments.

3. RESULTS

(i) *Female sterility*

In order to analyse the specific differences between 31 . 1 and 23 . 5 MRF with respect to female sterility we crossed 31 . 1/CyL^4 and 23 . 5/CyL^4 males with virgin females from several laboratory strains. A set of each of these matings was placed at 18, 25 and 28 °C and parental flies were discarded 4 days before progeny eclosion. The results for various crosses are presented in Table 1.

The heterozygous 23 . 5/*dp b cn bw*; +/*ve* daughters of cross 1a showed their highest frequency of sterility (66 %) when raised at 25 °C, and their lowest (12 %) at 28 °C. The females grown at 18 °C had both their ovaries atrophic at a frequency of 30 %. In contrast the 31 . 1/*dp b cn bw*; 31 . 1/*ve* female progeny derived from cross 1b displayed their highest sterility when they were cultured at 28 °C (93 %). At 25 °C there was 54 % sterility and none at 18 °C. In the reciprocal crosses (lines 2 and 3) almost all of both types of daughters possessed normal ovaries in all three culture temperatures.

The results of cross 4a appear to be different from those of 1a. The 23 . 5/Canton female progeny had equal sterility frequencies, when cultured at 25 and 28 °C (68 and 69 %, respecitively). The sterility found at 18 °C was 45 %. The results obtained with 31 . 1 MRF (cross 4b), appeared to be similar to those of cross 1b with the highest sterility at 28 °C and none at 18 °C.

In order to determine whether different second chromosome homologues or third chromosome combinations would interact differently with 23 . 5 and 31 . 1 MRF, four types of female progeny derived from each of crosses 5a, and 5b and two types derived from 6a and 6b were separately examined. As no differences in the number

Table 1. *Female sterility due to atrophic ovaries caused by 23.5 and 31.1 MRF*

	Type of mating ♀×♂	Genotype of the ♀♀ examined	18 °C			25 °C			28 °C		
			No. of ♀♀ examined	A−‡ (%)	AA‡ (%)	No. of ♀♀ examined	A− (%)	AA (%)	No. of ♀♀ examined	A− (%)	AA (%)
(1*a*)	**dp*; *ve* × 23.5/*CyL*^{4}	23.5/*dp*	243	25	30	176	17	66	224	21	12
(1*b*)	*dp*; *ve* × 31.1/*CyL*^{4}	31.1/*dp*	71	0	0	195	16	54	94	5	93
(2)	23.5/*CyL*^{4} × *dp*; *ve*	23.5/*dp*	116	1	2	167	0	1·2	132	0	0
(3)	31.1/*CyL*^{4} × *dp*; *ve*	31.1/*dp*	146	0	0	123	0	0	118	0	0
(4*a*)	Canton × 23.5/*CyL*^{4}	23.5/Canton	212	24	45	106	25	68	120	13	69
(4*b*)	Canton × 31.1/*CyL*^{4}	31.1/Canton	174	0·6	0·6	195	14	71	149	2	98
(5*a*)	M-5; *Cy*/*Pm*; *Ubx*/*Sb* × 23.5/*CyL*^{4}	23.5/...†	423	10	86	456	19	77	472	21	72
(5*b*)	M-5; *Cy*/*Pm*; *Ubx*/*Sb* × 31.1/*CyL*^{4}	31.1/...†	438	0·2	0	565	14	4	445	5	95
(6*a*)	M-5; *dp*; *Ubx*/*Sb* × 23.5/*CyL*^{4}	23.5/*dp*†	317	24	53	267	24	68	312	32	52
(6*b*)	M-5; *dp*; *Ubx*/*Sb* × 31.1/*CyL*^{4}	31.1/dp†	267	6	4	145	9	91	191	3	97
(7*a*)	*CyL*^{4}/*Pm* × 23.5/*CyL*^{4}	23.5/*CyL*^{4}	220	27	41	208	24	56	200	29	23
		23.5/*Pm*	195	28	33	233	32	45	217	28	33
(7*b*)	*CyL*^{4}/*Pm* × 31.1/*CyL*^{4}	31.1/*CyL*^{4}	132	0·8	0	128	0·8	0	101	1	0
		31.1/*Pm*	121	0	0	142	0·7	0	109	1	0
(8)	31.1/*CyL*^{4} × 23.5/*CyL*^{4}	31.1/23.5	103	1	2	91	2·2	2·2	102	1	2
(9)	23.5/*CyL*^{4} × 31.1/*CyL*^{4}	23.5/31.1	109	1	1	98	2	1	140	0	0·7

* *dp*: stands for the hole *dp b cn bw* chromosome. † See text.
‡ See Materials and Methods.

of sterile females were found among the various genotypes, the results were pooled together in Table 1.

Female progeny heterozygous for the 23.5 chromosome derived from cross 5a exhibited their highest frequency of sterility at 18 °C (86%). The corresponding frequencies at 25 and 28 °C were 77 and 72% respectively. In contrast, the female progeny heterozygous for the 31.1 chromosome in cross 5b displayed, as before, their highest sterility at 28 °C.

The results of cross 6a also show that 23.5 MRF induces high female sterility in all three temperatures studied. As before sterility induced by 31.1 MRF (cross 6b) was high when the female progeny were cultured at both 25 and 28 °C (91 and 97% respectively) but very low at 18 °C (4%).

The homogeneity χ^2 test for 2df showed statistically significant differences among the AA females derived from the same cross at 18, 25 and 28 °C. This holds for both 23.5 and 31.1 MRF (crosses: 1a. $\chi^2 = 142$ $P < 0{\cdot}001$, 1b. $\chi^2 = 140$ $P < 0{\cdot}001$, 4a. $\chi^2 = 25$ $P < 0{\cdot}001$, 4b. $\chi^2 = 344$ $P < 0{\cdot}001$, 5a. $\chi^2 = 27$ $P < 0{\cdot}001$, 5b. $\chi^2 = 1244$ $P < 0{\cdot}001$, 6a. $\chi^2 = 19$ $P < 0{\cdot}001$ and 6b. $\chi^2 = 537$ $P < 0{\cdot}001$). Similar results were also obtained when both A− and AA females were taken together in all the above cases.

It has been found that the cytoplasm of the CyL^4/Pm strain (utilized to isolate the 31.1 chromosome from a single captured male) suppresses male recombination induced by 31.1 MRF (Yannopoulos & Pelecanos, 1977). However, the CyL^4/Pm strain induces neither sterility (Yannopoulos, 1978*c*) nor male recombination (Yannopoulos, 1979). The data provided from crosses 7a and 7b (Table 1) indicate that the cytoplasm of this strain does not suppress the ability of 23.5 MRF to induce atrophic ovaries (the cross was carried out twice with similar results); however, it does suppress that of 31.1 MRF. The observation that the cytoplasm of the CyL^4/Pm stock suppresses the 31.1 MRF led Yannopoulos & Pelecanos (1977) to suggest that the cytoplasmic suppressors carried by the CyL^4/Pm and $31.1/CyL^4$ strains are identical. However, the results of cross 8 show that the $31.1/CyL^4$ strain suppresses 23.5 MRF. Thus the results of the 7a and 8 crosses favour the view that the cytoplasmic factors (suppressors) possessed by these two strains are different. It is known (Yannopoulos, 1978*b*) that when the 31.1 MRF is introduced by outcrossing into the cytoplasm of a normal strain, the latter acquires progressively a cytoplasmic resistance against the activities of the factor. Consequently, we may say that when 31.1 MRF is introduced into the cytoplasm of the CyL^4/Pm strain, the established $31.1/CyL^4$ stock develops its own resistance. The results of cross 9 show that the $23.5/CyL^4$ strain suppresses 31.1 MRF. However, it remains obscure whether this suppression is due either to the cytoplasmic suppressor(s) carried by CyL^4/Pm strain (the $23.5/CyL^4$ strain bears the cytoplasm of the CyL^4/Pm strain) or to a new kind of cytoplasmic resistance acquired by $23.5/CyL^4$ strain after its establishment.

Very low frequencies of sterility ranging from 0.0 to $\simeq 1\%$ due to atrophic ovaries were found in the utilized laboratory strains as well as in the strains $23.5/CyL^4$ and $31.1/CyL^4$ when they were cultured at 18, 25 and 28 °C. In each case more than 100 females were examined.

Female progeny with one ovary normal or reduced and the other atrophic were also observed in all crosses of Table 1 in which female sterility was scored. This phenomenon was found in crosses with both 23 . 5 and 31 . 1 MRF. The morphology of the atrophic ovaries induced by the two factors was identical. These observations suggest that the mechanisms involved in the induction of ovarian atrophy are similar for both factors. The females possessing normal, reduced or atrophic ovaries, did not show any difference in their external morphology.

(ii) *Male recombination*

Heterozygous 23 . 5/*dp b cn bw*; +/*ve* and 31 . 1/*dp b cn bw*; 31 . 1/*ve* sons derived from crosses 1a and 1b respectively, which were cultured at 18, 25 and 28 °C, were separately collected and individually mated with *dp b cn bw*; *ve* virgin females (G_2). The G_2 crosses were kept at 25 °C. The data presented in Table 2 show that 23 . 5/*dp b cn bw*; +/*ve* males gave the highest number of recombinants when raised at 25 °C. On the contrary, those grown at 28 °C produced no recombinants among their progeny. The male recombination frequency at 18 °C was found to be 0·59 %.

Forty-three of the recombinants recovered at 25 °C were phenotypically *cn* (double recombinants). They were produced from five different 23 . 5/*dp b cn bw*; +/*ve* males with the following distribution: Three gave one *cn* each, one gave 11, and the fifth gave 28. The male which produced the cluster of 28 *cn* individuals did not yield the expected normal phenotype but only *dp b cn bw* (42 individuals). Clusters of the single recombinants are provided in Table 2. It is interesting to mention here that large clusters of single and double recombinants have been repeatedly observed in crosses with 23 . 5 MRF. From experiments which are not included in this paper (Yannopoulos *et al.* 1981) six such males (23 . 5/*dp b cn bw*; +/*ve*), when mated to *dp b cn bw*; *ve* virgin females, gave the following in their progney: (1) 12 *dp b cn bw* + 11 *cn*, (2) 65 *dp b cn bw* + 47 *cn*, (3) 72 *dp b cn bw* + 44 *bw*, (4) 16 *dp b cn bw* + 16 *bw*, (5) 32 *dp b cn bw* + 23 *dp b*, + 79 *cn bw* + 4*b*, and the sixth produced only 87 *cn bw* individuals. Such a case has never been observed with 31·1 MRF during 5 years of research.

It was found (Zacharopoulou, Yannopoulos & Stamatis, 1980; Yannopoulos *et al.* 1981) that the phenotypically 'double recombinants' (*b*, *dp cn bw*, *cn*, *dp b bw*) are in reality deletions or duplications. Furthermore, it was detected that the 'double recombinants' of the same cluster bear an identical deletion or duplication. Besides *cn* other single and double recombinants appear frequently in clusters with 23 . 5 MRF.

As regards the heterozygous 31 . 1/*dp b cn bw*; 31 . 1/*ve* F_1 males, they yielded recombinants in all three culture temperatures with higher values at higher temperatures. Large cluster of recombinants were not observed (see Table 2). Moreover, as far as we know, the 'double recombinants' produced by 31 . 1 MRF are never clustered, each one corresponding to one male.

Table 2 shows that the cytoplasm of the CyL^4/Pm stock suppresses male recombination induced by 31 . 1 MRF (crosses 2b and 3) in contrast to the absence of suppression in 23 . 5 MRF (cross 2a).

Table 2. *Second chromosome male recombination induced by 23.5 and 31.1 MRF*

	Type of mating ♀ × ♂	Temperature (°C)	No. of ♂♂ tested	No. of progeny	Total no. of recombinants (%)	Double recombinants (%)	Clusters of single recombinants	♂♂ which yielded recombinants
(1*a*)	**dp*; *ve* × 23.5/*CyL*^{4}	18	17	2195	13 (0·59)	1 (0·05) cn	12^{+} × 1 = 12	9
		25	47	5886	122 (2·07)	43 (0·73) cn	18 × 1 + 3 × 2 + 3 + 13 + 19 + 20 = 79	27
		28	21	2745	0	0	0	0
(1*b*)	*dp*; *ve* × 31.1/*CyL*^{4}	18	20	2251	14 (0·62)	0	7 × 1 + 2 × 2 + 3 = 14	6
		25	19	2357	58 (2·46)	1 (0·04) cn	16 × 1 + 6 × 2 + 4 × 3 + 3 × 4 + 5 = 57	16
		28	28	2392	85 (3·55)	2 (0·08) cn	22 × 1 + 10 × 2 + 7 × 3 + 2 × 4 + 5 + 7 = 83	26
(2*a*)	*CyL*^{4}/*dp* × 23.5/*CyL*^{4} (♀♀ from the cross *CyL*^{4}/*Pm* × *dp*; *ve*)	25	14	2125	31 (1·46)	1 (0·05) cn	5 × 1 + 2 × 2 + 3 + 18 = 30	7
(2*b*)	*CyL*^{4}/*dp* × 31.1/*CyL*^{4} (♀♀ from the cross *CyL*^{4}/*Pm* × *dp*; *ve*)	25	18	2541	1 (0·04)	0	1	1
(3)	*CyL*^{4}/*dp* × 31.1/*CyL*^{4} (♀♀ from the cross *dp*; *ve* × *CyL*^{4}/*Pm*)	25	20	2290	66 (2·88)	0	21 × 1 + 9 × 2 + 6 × 3 + 4 + 5 = 66	18

* *dp*: stands for the whole *dp b cn bw* chromosome.
† No. of single recombinants × size of clusters.

The results are in good agreement with the corresponding with those of female sterility (see Table 1).

(iii) *Chromosomal abnormalities during male meiosis*:

It was found that male recombination induced by 31 . 1 MRF is always associated with chromosome breakage occurring mainly during meiosis. When 31 . 1/CyL^4 males were mated to virgin *dp b cn bw*; *ve* females, chromosome bridges and/or fragments were found both in anaphase I and II in frequencies of 37·8 and 39·8 % respectively (Table 3, cross 1).

In order to test whether 23 . 5 MRF induces the same phenomenon, we crossed 23 . 5/CyL^4 males to virgin *dp b cn bw*; *ve* females. Male pupae were then collected for testes preparations. The cultures were kept in a 25 °C incubator. Among the 105 anaphases I examined, 10 (9·5 %) were found to have bridges and/or fragments. There were 8 abnormal anaphases II out of 142 scored (5·6 %) (Table 3, cross 2).

4. DISCUSSION

The data presented in this paper have clearly demonstrated that the two male recombination factors, isolated from the same natural population, exhibit specific differences in their activities. 23 . 5 MRF induces female sterility in a wider range of temperature with no consistent relationship between sterility and temperature being detected. In contrast, 31 . 1 MRF is mainly effective at higher developmental temperatures and a positive correlation between female sterility and temperature has been consistently observed. Based on this evidence 31 . 1 MRF appears to be similar to other sterility male recombination factors studied (Engels & Preston, 1979; Schaefer, Kidwell & Fausto-Sterling, 1979; Matthews & Gerstenberg, 1979). The finding that 23 . 5 MRF induces female sterility at low temperatures (18 °C) provides evidence that the factor is active in the female embryo at such temperatures, while this effect does not seem to occur with 31 . 1 MRF. The observed low sterility and the absence of male recombination at 28 °C in cross 1a can be interpreted by the hypothesis that 23 . 5 factor is active to a lesser extent in the heterozygous F_1 23 . 5/*dp b cn bw*; +/*ve* offspring at 28 °C. However, the other results obtained with 23 . 5 MRF (see Table 1) provide evidence that this reduced activity is not solely due to the factor itself. A different interaction between the laboratory strains used and 23 . 5 MRF must be the reason.

The suppression of 23 . 5 MRF in cross 8 but not in 7a (Table 1) indicates that the 31 . 1/CyL^4 strain has acquired its own cytoplasmic resistance to 23 . 5 MRF which is different from that of the CyL^4/*Pm* stock.

Considered together the lower frequencies of abnormal anaphases, the extreme cases where the non crossover phenotypes were absent and the large clusters of single and identical double recombinants induced by 23 . 5 MRF, but not by 31 . 1 MRF, we can hypothesize that 23 . 5 MRF expresses itself mainly pre-meiotically whereas 31 . 1 MRF acts mainly during meiosis.

The extreme cases may be explained by two hypotheses: either deletion or

Table 3. *Chromosomal abnormalities at male meiosis induced by 31 . 1 and 23 . 5 MRF*

Type of mating ♀ × ♂	No. of pupae examined	Anaphase I		Anaphase II	
		Bridges and/or fragments (%)	Normal (%)	Bridges and/or fragments (%)	Normal (%)
(1) *dp ; ve** × 31 . 1/*CyL*^{4}	36	†42 (37·8)	69 (62·2)	72 (39·8)	109 (60·2)
(2) *dp ; ve* × 23 . 5/*CyL*^{4}	38	10 (9·5)	95 (90·5)	8 (5·6)	134 (94·4)

* *dp*: refers to the entire *dp b cn bw* chromosome II.

† Numbers not in brackets show the no. of Anaphases I or II examined.

crossing over events occurred in one original polé cell, or on an early germ cell stage; in the latter case, all mature spermatozoa could have descended from this early germ cell. Both the previously mentioned hypotheses are somehow in contrast with what is known about the formation of the pole cells and the generation of the germ cell line (Sonnenblick, 1950). However, Sonnenblick does not exclude instances in Drosophila whereby all pole cells have generated from one original nucleus. Moreover, the hypothesis proposed by Whittinghill (1955) and Kidwell & Kidwell (1975) does not seem to stand for the cases of the present study where loss of chromosomes does not take place.

These described earlier as 'reduced' ovaries possess normal ovarioles as well as ovarioles containing only germaria (Yannopoulos, 1978*c*). Because affected ovaries vary in size, according to the number of normal ovarioles, it can be hypothesized that the MRF factors studied do not act at specific stage during female germ line development. They can affect all or some of the precursor oogonial cells (pole cells) or even oogonial cells in different stages of development. Whether the defective development of the ovaries is due to the same reason which induces male recombination (chromosome breakage) is still obscure.

In an attempt to interpret the specific differences in the activities of these two MRF studied, we should mention that the data, up to now, that is: (*a*) their chromosome transmission (Kidwell, Kidwell & Sved, 1977; Yannopoulos, 1979), (*b*) the induction of unstable mutations (Green, 1977; Sinclair & Green, 1979), and (*c*) their ability for transposition (Yannopoulos, 1979) support the view that they are transposable genetic elements integrated into the chromosomes. However, it is still not clear whether MRFs are elements intrinsic to the *Drosophila* genome or whether they constitute foreign genetic elements, i.e. viruses or episomes (Voelker, 1974; Waddle & Oster, 1974; Roberts, 1976) integrated into the chromosomes. At any rate, independently of their nature, being genetic elements they can mutate. So, we can hypothesize that 31 . 1 and 23 . 5 MRF may be mutants of the same original element.

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