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An *SD* (Segregation Distribution) – *MR* (Male Recombination) chromosome isolated from a natural population of *Drosophila melanogaster*

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SUMMARY

A second chromosome line of *Drosophila melanogaster* (*S-90*), isolated from a northern California natural population, is able to induce (1) an increased frequency of X-chromosome visible mutations, (2) male recombination activity subject to reciprocal cross suppression, and (3) strong meiotic drive from heterozygous males. Based upon several lines of evidence (including the response to suppressor chromosomes of both systems) we conclude that *S-90* contains both *SD* (Segregation Distortion) and *MR* (*P* or *I*) chromosome activity. The two systems appear to behave independently and simultaneously, and a small centromeric region of the *S-90* chromosome appears to contain the major genetic elements of both systems.

INTRODUCTION

In recent years, a syndrome of unusual genetic phenomena has been observed among the hybrid individuals produced in certain interstrain matings of *Drosophila melanogaster*. The chromosomes (symbol *MR*) which contain the ability to induce this syndrome are often present in natural populations at frequencies of 30–100 % (Cardellino & Mukai, 1975; Yamaguchi, 1976; Matthews & Hiraizumi, 1978; Woodruff & Thompson, 1980; Kidwell, Kidwell & Sved, 1977). The anomalies associated with *MR* chromosomes include male recombination, mutation induction, male and/or female sterility, meiotic disturbances (including altered parameters of recombination and disjunction) and reduced transmission ratios from heterozygous males. These anomalies often occur only in one of the two reciprocal parental mating types and have been descriptively grouped under a syndrome known as *hybrid dysgenesis* (Hiraizumi *et al.* 1973; Kidwell, 1982 Kidwell *et al.* 1977; Slatko, 1978, Sved, 1979).

In addition to *MR* chromosomes in natural populations, chromosomes capable

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of altering the distribution of homologues among progeny also exist. The most extensively studied of these meiotic drive systems is termed Segregation Distorter (SD) (Sandler & Novitski, 1957). These chromosomes tend to be in rather low frequencies (1–5%, Sandler, Hiraizumi & Sandler, 1959; Hartl & Hartung, 1975). These chromosomes contain the genetic capability to show *increased* transmission ratios from heterozygous males, and are also associated with other phenomena such as mutation induction (Hartl & Hiraizumi, 1976).

Both *MR* chromosomes and *SD* chromosomes appear to share several interesting features including defects in spermio-genesis (Matthews, 1981), and a polygenic genetic system responsible for the phenotypic effects for each system (Ganetzky, 1977; Slatko & Hiraizumi, 1975, 1978; Matthews *et al.* 1978). Both *MR* and *SD* appear to have several major genetic elements located in centromeric hetero- and euchromatin (Ganetzky, 1977; Slatko & Hiraizumi, 1975; Slatko & Green, 1980; Periquet & Anxolabehere, 1982). Other parallels include mutation induction (Slatko & Hiraizumi, 1973; Hiraizumi, 1961; Hartl & Hiraizumi, 1976), and the existence of suppression systems (Matthews & Slatko, 1983; Kataoka, 1967; Martin & Hiraizumi, 1979). These two systems, in fact, may be related in some as yet unknown way (Matthews & Slatko, 1982; Hiraizumi, 1979; Martin & Hiraizumi, 1979).

This report describes one chromosome isolated from a northern California population, *S-90*, which appears to contain both *MR* and *SD* characteristics, based upon several criteria. The finding of such *SD/MR* chromosomes raises interesting questions concerning the relationships of the two systems and the genetic structure of natural populations of *Drosophila*.

MATERIALS AND METHODS

Descriptions of *D. melanogaster* strains used for the present study are listed in Lindsley & Grell (1968).

Special chromosomes utilized include the following:

C(1)DX, y f. A tandem acrocentric compound X chromosome line in which females must carry a Y chromosome to survive and thus produce sons which inherit patroclinous X chromosomes. It will be abbreviated as *C(1)DX* in this report.

In(1)FM7, y^{31d} w^a v B. A multiply inverted balancer X chromosome. This chromosome will be abbreviated as *FM7* for this report.

S-90. A second chromosome line isolated from a single female captured at the Gunlach Bunshue winery in the Sonoma Valley, California, and provided to us by W. Marks. A single second chromosome from this female was isolated by conventional procedures, and is now kept as a balanced lethal stock *S-90/CyO, dp^{1VI} pr cn^2*. This line is abbreviated as *S-90/Cy* in this report.

A standard cornmeal-agar food was used for all experiments. Flies used for experimental matings were usually less than six days old. All experiments were carried out at room temperature (24 °C).

Male recombination induced by the *S-90* chromosome or its recombinant derivatives was measured as follows: */Cy* males were taken from the stock and crossed to *cn bw* females and single F_1 */cn bw* males were back-crossed to a harem

of virgin *cn bw* females. The frequency of male recombination was determined from F_2. The transmission frequency of the chromosome in question (*) was assayed from the same crosses, from */*cn bw* heterozygotes to their progeny. The measure of transmission frequency, k, is the frequency of * among the total non-recombinant F_2 progeny. When segregation and transmission is Mendelian $k = 0{\cdot}50$. When * is transmitted in excess, k approaches 1·0. All k values presented in this report have been corrected for viability by utilizing the procedure described by Ganetzky (1977), as follows. The number of *cn bw* progeny observed for the cross */*cn bw* ♂ × *cn bw* ♀♀ was multiplied by the ratio */*cn bw* observed from the reciprocal cross. This adjusted number of *cn bw* flies was used to calculate the k values from the original cross.

EXPERIMENTS AND RESULTS

S-90 was tested for male recombination induction as part of a screen for such activity among numerous second chromosome lines for a northern California population. Utilizing the mating scheme described in Materials and Methods it was found that *S-90* is able to induce male recombination with a frequency of 0·0021 (Table 1). In addition to male recombination induction, *S-90* also appeared to show an increased transmission ratio above the expected Mendelian 1 : 1 ratio ($k = 0{\cdot}95$), a result similar to those observed with *SD* chromosomes (Hartl & Hiraizumi, 1976; Ganetzky, 1977).

To ascertain whether the male recombination activity and transmission frequency associated with *S-90* might be subject to a 'reciprocal cross effect', *S-90/Cy* females from the stock were crossed to *cn bw* males and from these matings *S-90/cn bw* males were back-crossed to *cn bw* females. The results of these matings are also included in Table 1. It appears that the segregation distortion phenotype is not influenced by whether the *S-90* chromosome is of paternal or maternal origin whereas there is an apparent five-fold decrease in male recombination frequency. Similar reduced but still non-trivial frequencies of male recombination were reported for several *MR* strains when assayed for reciprocal effects (Kidwell *et al.* 1977).

In the control matings it was noticed that female recombination was strongly inhibited in the right arm of the *S-90/cn bw* heterozygous females, suggesting the presence of an inversion in the *S-90* chromosome. Aceto-orcein squash preparations were made from the salivary glands of third-instar larvae of *S-90/cn bw* flies and a small inversion (breakpoints of 52B–56F on the cytological map) was observed. This inversion appears to be identical to a previously known one, *In(2R)NS*, often associated with *SD* chromosomes (Lindsley & Grell, 1968).

It should be noted that the recombinant chromosomes generated in *S-90/cn bw* males follow the distortion pattern observed for the parental (non-recombinant) chromosomes (Table 1). This suggests that the distortion and male recombination events can simultaneously occur in the same meiocyte or its derivative in spermiogenesis. It is known that, at least for one *MR* chromosome, the reduced k value associated with the *T-007* second chromosome is not present among the recombinant progeny (Hiraizumi, 1979). This suggests that the increased k

associated with *S-90* and the male recombination induction may be due to separate genetic systems.

Because both *SD* and *MR* chromosomes are known to produce mutator lines (Hiraizumi, 1961; Hartl & Hiraizumi, 1976; Slatko & Hiraizumi, 1973; Green, 1979*a*; Kidwell *et al.* 1977; Yannopoulos, 1978; Simmons & Lim, 1980; Berg, Engels & Kreber, 1980; Engles & Preston, 1981; Woodruff, Slatko & Thompson, 1982), *S-90* was assayed for the ability to induce visible mutations along the X

Table 1. *Transmission frequencies* (k) *and percentage of male recombination observed in* S-90/cn bw *heterozygotes carrying an* S-90 *chromosome of paternal* (*A cross*) *or maternal* (*B cross*) *origin*

Type of cross	*k* value	Male recombination (%)	No. of recombinations		*N*
			cn	*bw*	
A	0·95	0·21 (0·16)	5 (4)	22 (15)	12899
B	0·95	0·08 (0·04)	0·0	5 (4)	8525

The *k* values were adjusted for viability as described in Materials and Methods. Figures in parentheses indicate the minimum estimates of recombination, where clusters are considered as one event. *N* is the number of progeny scored. For full details consult the text.

chromosome. Forty-eight males from the *S-90* stock were singly mated to 5 virgin y^2 sn^3 ras^2 *v* females. The matings were transferred to fresh food and each male was given five new females. This procedure was repeated for a total of five transfers and the parents were discarded after 7 days of egg-laying. The female progeny from these matings were scored for the presence of any of the four visible markers and also for any dominant mutants. Among 32,307 progeny, 24 *sn*, 5 *ras* and 13 dominant mutations (12 Minute and 1 Notch) were recovered. Some of the mutations did occur in clusters, suggesting a possible pre-meiotic origin for at least a portion of them. The increased mutation frequency at *sn* and *ras* is characteristic for *MR* chromosomes (Green, 1977*b*, *c*).

In the light of the above findings it could be suggested that the *cn* and *bw* flies recovered in the male recombination experiments were of mutation rather than recombination origin. This was especially a consideration since 3 of 5 presumptive *cn* recombinants observed among the progeny of the *S-90/cn bw* males also appeared to be of Minute phenotype. On the basis of similar observations Green & Shepherd (1979) have suggested that they may be due to short deletions.

To ensure that the majority of *cn* and *bw* flies recovered were in fact true recombinants, *S-90* males were tested for recombination induction in the usual way utilizing a multiply marked second chromosome, *al dp b pr c px sp*, instead of *cn bw*. Eight exceptions of independent origin, from among the 969 progeny produced in these matings, all appeared to be true recombinants, as multiple marker exchange was observed.

As it was of interest to separate the *SD* and *MR* components genetically, experiments were designed to examine the interrelationships of these two systems. Slatko & Hiraizumi (1978) and Matthews & Slatko (1983) have reported the isolation and characterization of an *X* chromosome which contains the capability for suppressing several qualitative characteristics of the *MR* phenotype, including

male recombination induction. To test the effect of *X* chromosome suppressors of *MR* (symbol: (*X*)) upon *S-90*, (*X*)/*FM7*; *cn bw*/*Cy* females were crossed to *S-90*/*Cy* males. From these matings, F_1 (*X*)/*Y*; S-*90*/*Cy* males were crossed to *C(1)DX*; *cn bw* females and F_2 (*X*)/*Y*; *S-90*/*cn bw* males were selected and back-crossed to *cn bw* females. Progeny from this cross were scored for male recombination induction and *K* value. This scheme ensures the paternal origin of both (*X*) and *S-90*, necessary because of the unidirectional suppression often observed with *MR* chromosomes (Kidwell *et al.* 1977; Engels, 1979*b*).

The results of these crosses indicate that the suppressor X chromosome (*X*) does suppress the frequency of male recombination, but not the transmission distortion activity associated with *S-90*. From among 2688 progeny, only two recombinants (both *bw*) were obtained (a frequency of 0·0007) and the *k* value was 0·98.

Suppressor-X chromosomes of *SD* have also been isolated and characterized (Martin & Hiraizumi, 1979; Kataoka, 1967) and one *SD* suppressor-X [symbol: (*X**)] was utilized to test the effect upon *S-90*. (*X**)/*FM7*; *cn bw*/*Cy* females were crossed to *S-90*/*Cy* males and from these parental crosses (*X**)/*Y*; *S-90*/*cn bw* males were selected and back crossed to *cn bw* females. Progeny from these crosses were scored for male recombination induction and *k* value. The results of these crosses indicated that (*X**) suppressed neither the *SD* phenotype nor the *MR* phenotype. Among 12816 progeny from 78 matings there were 28 recombinants (24 *bw* and 4 *cn*) (a frequency of 0·0022) and the *k* value was 0·91. Non-suppression of *S-90* was to be expected, as (*X**) apparently only suppresses SD chromosomes whose right arms have been replaced with a laboratory chromosome such as *cn bw*. This experiment did, however, indicate that *MR* phenotype associated with *S-90* was not suppressed.

An additional experiment was performed with (*X**), similar to that described above, but utilizing and *S-90* chromosome whose right arm was replaced by recombination with a standard laboratory *pr cn* chromosome. This derivative, *S-90*, *cn-3* was tested with (*X**) as described above. From 4632 progeny a *k* value of 0·48 (2223 *cn* flies) was obtained. Male recombination frequencies were not assayed in this experiment. It should be mentioned that *S-90*, *cn-3* shows a *k* of 0·97 against *cn bw* in the absence of (*X**). The results from these experiments suggest that the suppressor-X chromosomes of each system [(*X*) or (*X**)] suppress only the phenotypes of their respective system and further confirm the duality of systems present within the *S-90* chromosome.

A genetic mapping experiment for the *SD* genetic elements was performed, by crossing *S-90*/*Cy* males to *al dp b Tft pr Bl c px sp*/*Cy* females, selecting F_1 *S-90*/*al dp b Tft pr Bl c px sp* females and crossing them to *al dp cn bw*/*Cy* males. From these crosses, various Recombinant Chromosome Genotypes (RCGs) generated in the F_1 females were selected and balanced over *Cy*. Stocks were maintained as *RCG*/*Cy* × *RCG*/*Cy*. The mapping data represent 12 independently isolated cross-overs in the *Tft–pr–Bl* interval of the second chromosome. Results of testing each transmission frequency and male recombination induction in *RCG*/*cn bw* heterozygotes are presented in Table 2.

Of six *Tft* pr^+ Bl^+ crossover types, two show high distortion, two show no distortion and two show intermediate levels of distortion. The five reciprocal

crossover types, *Tft*$^+$ *pr Bl* all show *k* values close to 0·50. The single *Tft*$^+$ *pr*$^+$ *Bl* crossover type showed no distortion.

These results suggest that the *Tft*$^+$ *pr*$^+$ *Bl*$^+$ interval of the *S-90* chromosome does contain genetic elements affecting distortion, similar to the *SD* elements (Ganetzky, 1977 and personal communication). One complexity in these data concerns the *Tft*$^+$ *pr*$^+$ *Bl* genotype, which was expected to be a distorter chromosome. As this chromosome is insensitive to distortion (data not shown), it

Table 2. *Transmission frequencies* (k *values*) *and percentages of male recombinations observed* (*between the markers* cn *and* bw) *among* 12 *recombinant derivatives of* S-90 *generated in the* Tft-Bl *interval*

Male genotype	*k* value	Male recombination (%)	No. of recombinants	*N*
S-90/cn bw	0·95	0·21 (0·16)	27 (19)	12899
Tft pr$^+$Bl$^+$-1/cn bw	0·92	0·20 (0·18)	11 (10)	5452
Tft pr$^+$Bl$^+$-16/cn bw	0·99	0·16 (0·04)	9 (2)	5460
Tft pr$^+$Bl$^+$-7/cn bw	0·63	0·01 (0·01)	1 (1)	8745
Tft pr$^+$Bl$^+$-14/cn bw	0·57	0·05 (0·05)	6 (6)	12311
Tft pr$^+$Bl$^+$-20/cn bw	0·49	0·33 (0·14)	19 (8)	5753
Tft pr$^+$Bl$^+$-21/cn bw	0·50	0·23 (0·22)	13 (12)	5564
Tft$^+$pr Bl-3/cn bw	0·46	0	0	14362
Tft$^+$pr Bl-4/cn bw	0·52	0	0	10543
Tft$^+$pr Bl-8/cn bw	0·55	0	0	6·050
Tft$^+$pr Bl-12/cn bw	0·50	0	0	6·532
Tft$^+$pr Bl-10/cn bw	0·47	0	0	6·539
Tft$^+$pr$^+$Bl-9/cn bw	0·53	0	0	8·310

The *k* values were adjusted for viability as described in Materials and Methods. Figures in parentheses indicate the minimum estimates of recombination, where clusters are considered as one event. *N* is the number of progeny scored. For full details consult the text.

could be assumed that there may be elements on *S-90*, in addition to *SD* on the *Tft*$^+$ *pr*$^+$ *Bl* chromosome, which are also necessary for distortion to occur. Because these elements must also be present on a reciprocal RCG chromosome, as a confirmation, this *Tft*$^+$ *pr*$^+$ *Bl* genotype was made heterozygous with the *Tft pr*$^+$ *Bl*$^+$*–21* chromosome in females, and backcrossed to *Cy/Pm* males. Fifteen independent progeny males of the genotype *Tft*$^+$ *pr*$^+$ *Bl*$^+$, were selected and balanced over *Cy*. Each independent isolate was tested for distortion and male recombination ability. As shown in Table 3, ten of the fifteen *Tft*$^+$ *pr*$^+$ *Bl*$^+$ chromosome show full distortion and five show low levels or no distortion.

These results also strongly suggest that elements responsible for distortion lie in the *Tft*$^+$ *Bl*$^+$ interval of the *S-90* chromosome. The reason that the *Tft*$^+$ *pr*$^+$ *Bl* chromosome fails to show distortion is unknown.

With respect to male recombination activity the mapping is less precise due to the low frequencies of occurrence of recombinants and the presence of *In(2R)NS*. Nevertheless the results of these experiments (Tables 2 and 3) do show that the elements responsible for the majority of male recombination induction also map to the *Tft*$^+$ *Bl*$^+$ interval of the *S-90* chromosome, perhaps between *pr*$^+$ and *Bl*$^+$. As several RCGs show one phenotype (male recombination induction) and not the other (distortion), it appears that the *SD* and *MR* elements are separable, and therefore at least partially distinct.

DISCUSSION

The results presented above demonstrate the existence of a second chromosome line isolated from a natural population (*S-90*) which contains both *MR* and *SD* elements. The evidence that the distortion is actually due to *SD* elements can be summarized as follows. (1) Analysis of recombinant progeny localizes several genetic elements responsible for distortion between the *Tft* and *Bl* loci. These

Table 3. *Transmission frequencies* (k *values*) *and* % *of male recombination* (*between the markers* cn *and* bw) *among the fifteen* Tft^+ pr^+ Bl^+ *recombinants generated between the* Tft^+ pr^+ Bl-9 *and* Tft pr^+ Bl^+-21 *chromosomes*

Recombinant line	*k* value	Male recombination (%)	Number of recombinants	*N*
1	1·0	0·81	9	1092
2	0·99	0·21	2	785
3	0·99	0·17	2	1121
4	0·99	0·51	4	766
5	0·99	0·27	3	1073
6	0·99	0·0	0	351
7	0·99	0·41	5	1209
8	0·99	0·10	1	998
9	0·98	0·46	3	645
10	0·98	0·28	2	701
11	0·61	0·20	2	982
12	0·48	0·22	10	808
13	0·46	0·51	5	971
14	0·42	0·27	2	749
15	0·36	0·21	1	467

The *k* values were adjusted for viability as described in Materials and Methods. *N* is the number of progeny scored, excluding recombinants. Clusters could not be detected in these crosses. For details consult the text.

elements are separable from those responsible for male recombination induction. (2) Distortion only occurs in males, not in females heterozygous for *S-90* and *cn bw*, and is independent of the origin of the *S-90* chromosome (male or female) in the parental generation. (3) Distortion occurs among recombinant progeny of *S-90/cn bw* males, with the vast majority of the recombinants being genotypically *bw* rather than *cn*, whereas distortion associated with *MR* is not present among recombinant progeny (Hiraizumi, 1979). (4) Suppression of distortion using an appropriate *S-90* recombinant (*S-90, cn-3*) occurs with a suppressor-X chromosome [(*X**)] known to suppress the *SD* phenotype. (5) *S-90* contains *In(2R)NS*, often associated with *SD* chromosome isolated from the wild.

The evidence that the *MR* phenotype is actually due to a similar genetic system, as has been observed previously, rests on the following lines of evidence. (1) the observed induction of recombinants among the progeny of males heterozygous for *S-90*. (2) Parental reciprocal crosses show unidirectional suppression of male recombination induction. (3) Suppressor-X chromosome [(*X*)] known to suppress *MR* traits also suppress male recombination induction in *S-90* heterozygotes. (4) Mutation induction resulting in increased frequencies of *sn* and *ras* (and other visible mutations) occurs in appropriately crossed *S-90* heterozygous males (*S-90* heterozygous females were not tested) (5) Genetic elements responsible for the

majority of male recombination induction map to the *Tft-Bl* region of the *S-90* chromosome. Woodruff & Lyman (1980) report the finding of several *SD–MR* chromosomes from a natural population of *D. melanogaster* (from Bowling Green, Ohio) with characteristics similar to those reported here. Our results support their conclusion concerning the actual genetic nature of such chromosomes, with respect to *SD* and *MR*. It is of interest that one line upon which they extensively report, *BG-12*, also contains the pericentric inversion associated with some *SD* chromosomes found in nature (Hartl & Hiraizumi, 1976), although *S-90* does not.

It is known that most natural populations of *D. melanogaster* contain specific transposable DNA sequences which appear to be responsible for several phenotypes associated with hybrid dysgenesis, including male recombination. At present two such families of transposable elements are known, the *P* elements (Engels, 1979*a*; Bingham, Kidwell & Rubin, 1982; Rubin, Kidwell & Bingham, 1982), and the *I* elements (Bregliano *et al.* 1980; Bregliano & Kidwell, 1982) associated with the *P–M* and *I–R* systems of hybrid dysgenesis, respectively. We have found that the *S-90/Cy* line is able to induce *I* sterility and is resistant to *P* sterility induction (our unpublished observations). Although it is reasonable to assume that the *MR* activity of *S-90* is connected with transposons, its exact relationship to these systems of hybrid dysgenesis remains to be determined. It is suggested that *P* elements are invasive (Kidwell, Novy & Feeley, 1981; Bingham *et al.* 1982). The occurrence of chromosomes containing both *SD* and *MR* activity can be explained by assuming that an *SD* chromosome was invaded by elements similar to *P* or *I*.

The coupling of *SD–MR* chromosomes may contribute to the rapid increase in dysgenic hybrids at times of occasional hybridization among various strains and populations with overlapping interacting systems of hybrid dysgenesis, where *SD* provides a vehicle for transmission of *MR* chromosomes to the next generation. This may explain the rapid spread of some transposon families in natural populations.

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