

## Reciprocal differences in female recombination associated with hybrid dysgenesis in *Drosophila Melanogaster*\*

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### SUMMARY

Large reciprocal differences in recombination frequencies were observed in  $F_1$  female progeny of a number of strain crosses. These reciprocal effects on recombination were found in several intervals of all three major chromosomes; they were greatest in intervals proximal to or spanning the centromere but were also found in some distal regions. The direction of recombination change was not consistent over different chromosomal intervals. There was no clear trend for reciprocal recombination differences to be associated with change of interference values between adjacent intervals. A close association was found between reciprocal female recombination effects, male recombination and other component traits of hybrid dysgenesis. However, reciprocal differences in female recombination were not restricted to dysgenic crosses. Backcross experiments demonstrated that reciprocal differences in the centromeric region of chromosome III were the result of increased crossing-over in dysgenic  $F_1$  hybrids rather than decreased crossing-over in the reciprocal hybrids. It is concluded that genotype–cytoplasm interaction can be a major factor influencing recombination frequencies and that the use of interstrain hybrids for recombination measurement may lead to errors in the estimation of intrastrain frequencies.

### 1. INTRODUCTION

A syndrome of associated aberrant traits has been observed in the  $F_1$  hybrid progeny of many pairs of strains of *Drosophila melanogaster* (Kidwell, Kidwell & Sved, 1977). These aberrant traits included recessive lethal mutation, female and male sterility, male recombination, segregation distortion, chromosomal aberration and non-disjunction. The manifestation of dysgenic traits tends to be non-reciprocal and, at least in the case of sterility, highly dependent on temperature. The name *hybrid dysgenesis* has been given to this related group of phenomena (Kidwell & Kidwell, 1976; Sved, 1976). The strain interactions which result in hybrid dysgenesis most commonly, but not invariably, occur when females from long-established laboratory strains are crossed with males from strains more recently derived from nature. Some type of rapid and widespread evolutionary divergence seems to have occurred between the two groups of strains.

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Observation of significant frequencies of male recombination (e.g. Hiraizumi, 1971; Voelker, 1974; Waddle & Oster, 1974; Kidwell & Kidwell, 1975*a*; Woodruff & Thompson, 1977) has led to the obvious question of whether female recombination frequencies are affected in dysgenic hybrids. Hiraizumi *et al.* (1973) initially reported no observable changes in female recombination frequencies in crosses exhibiting male recombination. Later, Slatko & Hiraizumi (1975) claimed that female recombination changes were associated with male recombination induction. Because of wide variations in female recombination values among different strains (e.g. Hofmanova, 1974; Kidwell, 1972; Sinclair, 1975) it is a questionable procedure to use other strain crosses or published map distances as standards of comparison for such tests. Fortunately, the finding of reciprocal differences in other dysgenic traits suggested appropriate controls for measuring female recombination in dysgenic hybrids, the F<sub>1</sub> female progeny of the non-dysgenic reciprocal crosses. These reciprocal cross females are expected, on average, to possess the same chromosomal constitutions and to differ from dysgenic females only in the source of their maternal cytoplasm.

This paper documents the evidence for including female recombination changes among the manifestations of hybrid dysgenesis and demonstrates that cytoplasm-genotype interaction can be an important factor in determining female recombination frequencies. It also provides information on the distribution and nature of recombination changes in dysgenic F<sub>1</sub> females and evidence that their manifestation is essentially restricted to interstrain hybrids.

## 2. MATERIALS AND METHODS

The following strains of *Drosophila melanogaster* were employed:

Ames I, wild type (Gowen & Johnson, 1946).

Canton-S, wild type (Lindsley & Grell, 1968).

Cranston, wild type (Kidwell, Kidwell & Ives, 1977).

Harwich, wild type (Kidwell & Kidwell, 1976).

Oregon R-C, wild type (Lindsley & Grell, 1968). Subdivided into two independently maintained subcultures in 1971 referred to as Oregon R-C (I) and Oregon R-C (II). The two subcultures were assumed to be genetically identical when these experiments were initiated.

*y*<sup>2</sup> *cv* *v* *f* *car*, a chromosome I multiply marked stock: yellow, *y* (0.0); cross-veinless, *cv* (13.7); vermilion, *v* (33.0); forked, *f* (56.7); carnation, *car* (62.5).

*al* *cl* *b* *c* *sp*<sup>2</sup>, a chromosome II multiply marked stock: aristaless, *al* (0.01); clot, *cl* (16.5); black, *b* (48.5); curved, *c* (75.5); speck-2, *sp*<sup>2</sup> (107.0).

*Gl* *Sb*/LVM, a stock carrying the dominant markers: Glued, *Gl* (3-41.4); Stubble, *Sb* (3-58.2).

*rucuca*, a chromosome III multiply marked stock (Lindsley & Grell, 1968).

The Cranston and Harwich strains were collected from the wild in 1964 and

1967 respectively. The remaining strains had been laboratory-maintained for many years.

Female recombination was measured according to the method of Bridges & Brehme (1944). A maize-meal/molasses/agar medium seeded with live yeast was employed. Propionic acid was used as a mould-inhibitor. The temperature was maintained at  $25 \pm 1$  °C.

3. RESULTS

(i) *Recombination frequencies in reciprocal crosses*

For each of two wild-type strains, Cranston and Oregon R-C (I) or (II), reciprocal crosses were made with three multiply marked stocks: *y<sup>2</sup>cv v f car, al cl b c sp<sup>2</sup>* and *rucuca* for monitoring recombination in chromosomes I, II and III respectively. The parental strains were mass-mated in half-pint milk bottles. Reciprocal crosses are designated according to previous usage:

Cross A	Cross B
Marker ♀♀ × wild-type ♂♂	Wild-type ♀♀ × marker ♂♂

Between 10 and 20 F<sub>1</sub> virgin females from each cross were individually back-crossed to males of the appropriate marker stock. Reciprocal crosses were always tested concurrently under external conditions as identical as possible.

Recombination frequencies are presented in Table 1 for four intervals of the X chromosome. It was found that carnation could not be reliably scored in the

Table 1. *Mean percentage recombination in four intervals of the first chromosome in F<sub>1</sub> female progeny from reciprocal crosses between y<sup>2</sup> cv v f car and two wild-type strains*

Interval	Cross						Standard map distance (cM)†
	y <sup>2</sup> cv v f car Cranston			y <sup>2</sup> cv v f car Oregon R-C (I)			
	A	B	A-B	A	B	A-B	
<i>y<sup>2</sup>-cv</i>	9.1	10.3	-1.2	9.9	12.0	-2.1**	13.7
<i>cv-v</i>	15.5	17.7	-2.2*	17.9	22.8	-4.9**	19.3
<i>v-f</i>	19.8	20.5	-0.7	18.7	21.4	-2.7	23.7
No. progeny counted	2375	2248	—	2490	3111		
<i>f-car</i>	6.6	6.8	-0.2	4.6	6.9	-2.3**	5.8
No. progeny counted	1633	2344	—	2386	2378		

\* *P* < 0.05; \*\* *P* < 0.01.

† Lindsley & Grell (1968).

presence of vermilion due to partial suppression of the carnation phenotype. Recombination in the *f-car* interval was therefore scored in a separate experiment using a marker stock bearing *f* and *car* only, which was derived from the *y<sup>2</sup>cv v f car* stock. For both groups of wild-type/marker hybrids, and in every measured interval, recombination in the B cross exceeded that in the A cross. These differ-

ences were at least significant in the *cv-v* interval for both wild-type hybrids and highly significant in the  $y^2-cv$  and *f-car* intervals for Oregon R-C (I) hybrids.

Second chromosome recombination frequencies are given in Table 2 for the Cranston and Oregon R-C (I) hybrids. In addition, comparable data are provided

Table 2. Mean percentage recombination in four intervals of the second chromosome in  $F_1$  female progeny from reciprocal crosses between *al cl b c sp<sup>2</sup>* and three wild-type strains

Wild-type strain	Direction of cross	Interval				No. progeny counted
		1 <i>al-cl</i>	2 <i>cl-b</i>	3 <i>b-c</i>	4 <i>c-sp<sup>2</sup></i>	
Cranston	A	14.1	23.6	21.5	30.4	1472
	B	18.0	22.0	12.9	23.9	2837
	A-B	-3.9**	1.6	8.6**	6.5**	
Oregon R-C (I)	A	17.2	27.6	22.9	28.9	1578
	B	15.3	22.7	19.6	31.1	1886
	A-B	1.9	4.9**	3.3*	-2.2	
ID†	A	3.8	2.5	20.2	31.8	711
	B	3.9	2.5	18.1	32.8	1489
	A-B	-0.1	0	2.1	-1.0	
Standard map distance (cM)‡		16.5	32.0	27.0	31.5	

\*  $P < 0.05$ ; \*\*  $P < 0.01$ .

† Data from Kidwell & Kidwell (1975b).

‡ Lindsley & Grell (1968).

from an earlier study involving crosses with the same marker stock and the ID mutator line (Kidwell & Kidwell, 1975b). This line was known to carry an inversion in the left arm of chromosome II with breakpoints 31B-38C (Kidwell, Kidwell & Nei, 1973). In both Cranston and Oregon R-C (I) hybrids, significant differences between reciprocal crosses were found in at least half of the tested intervals. In both cases, A-cross female recombination exceeded that of the B cross in the centromeric (*b-c*) interval. In other intervals there seemed to be no consistency in the direction or size of reciprocal differences. These results are in contrast with the ID line crosses which produced no significant reciprocal differences in any tested interval.

Recombination frequencies for six third chromosome intervals are presented in Table 3. Significant differences between reciprocal crosses were not observed in the Oregon R-C (I)/*rucuca* hybrids. In the Cranston/*rucuca* hybrids they were restricted to the pericentric (*st-cu*) interval and the adjacent *cu-sr* interval. In both these intervals, cross A hybrids showed large increases in recombination, compared with cross B hybrids. In every interval, Cranston B recombination frequencies were closer to the Oregon R-C (II) values than were those of Cranston A, the known dysgenic hybrid. This suggests that reciprocal differences between Cranston A and B hybrids are due to increases in Cranston A recombination frequencies rather than decreases in Cranston B values. Further evidence to support this idea will be presented in a later section.

Crosses between Cranston and Oregon R-C (II) and *Gl Sb/LVM*, another chromosome III marker stock, gave patterns of recombination in the centromeric region (Table 4) very similar to those of the *rucuca* tests (Table 3). Reciprocal differences were large and highly significant between the Cranston hybrids but small and non-significant between the Oregon R-C (II) hybrids.

Table 3. Mean percentage recombination in six intervals of the third chromosome in  $F_1$  female progeny from reciprocal crosses between *rucuca* and two wild-type strains

Interval	Cross						Standard map distance†
	<i>rucuca</i> /Cranston			<i>rucuca</i> /Oregon R-C (II)			
	A	B	A-B	A	B	A-B	
1 <i>ru-h</i>	23.8	24.4	-0.6	25.3	24.6	0.7	26.5
2 <i>h-st</i>	19.6	17.9	1.7	15.9	15.4	0.5	17.5
3 <i>st-cu</i>	7.6	3.6	4.0**	2.4	3.8	-1.4	6.0
4 <i>cu-sr</i>	12.7	8.9	3.8**	8.2	7.5	0.7	12.2
5 <i>sr-e<sup>a</sup></i>	10.1	9.3	0.8	8.2	8.9	-0.7	8.5
6 <i>e<sup>a</sup>-ca</i>	32.6	35.4	-2.8	36.6	34.8	1.8	30.0
No progeny counted	1475	1720		1298	784		

\*\*  $P < 0.001$ .

† Lindsley & Grell (1968).

### (ii) Direction of female recombination change

Theoretically, the observed reciprocal differences could have occurred through either enhancement of recombination frequencies in one cross or reduction in the reciprocal cross. Comparison with standard map distances cannot reliably differentiate between these alternatives because of strain variability from the published values. An experiment was therefore designed to differentiate between these two theoretical alternatives in the case of a third chromosome pericentric interval. The design of this experiment also enabled us to test another hypothesis: that recombination changes are restricted to interstrain hybrids and are not exhibited in the progeny of intrastrain matings (see Kidwell, Kidwell & Sved, 1977).

Two wild-type strains, Canton-S and Harwich, were chosen for the experiment; from previous extensive experience these strains were known to interact strongly together and produce dysgenic  $F_1$  hybrids from the cross of Canton female  $\times$  Harwich male. These two strains were first independently mated in reciprocal combinations with the *Gl Sb/LVM* marker stock.  $F_1$  females were then back-crossed to males of the wild-type parental strain and recombination frequencies estimated. Significant reciprocal differences were recorded for both sets of matings (Table 4) but the magnitude of the difference was greater for the Harwich than the Canton-S crosses. *Gl Sb*  $F_1$  males were selected from both sets of B crosses and these were separately back-crossed to females of their respective parental wild-type line, Harwich or Canton-S. Female progeny of these matings were tested for recombina-

nation between *Gl* and *Sb*. This cycle of *Gl Sb* male selection and backcrossing was repeated every generation, resulting in lines which were essentially Harwich and Canton-S except for a third chromosome bearing *Gl* and *Sb*. Tests of female recombination in both back-cross lines were made in generations 1, 2, 3, 4, 5, 9 and 12. In addition, reciprocal intercrosses between the Harwich and Canton-S back-cross lines were made at generations 4, 8 and 11 and the intercross female progeny were tested concurrently with the backcrosses at generations 5, 9 and 12 respectively. The results are presented in Table 5. Successive backcrossing to both the

Table 4. *Mean recombination values in the third chromosome pericentric interval Gl Sb, † of F<sub>1</sub> females from reciprocal crosses between Gl Sb/LVM and five wild-type strains*

Wild-type strain	Cross		
	A	B	A-B
Cranston	18.1 (3814)	10.0 (4131)	8.1**
Oregon R-C (II)	10.7 (2187)	9.2 (2372)	1.5
Canton-S	13.8 (3144)	8.9 (4130)	4.9**
Harwich	18.4 (2528)	9.7 (2803)	8.7**
Ames	17.2 (1642)	9.7 (3413)	7.5**

\*\*  $P < 0.01$ .

† Standard map distance 16.8 cM (Lindsley & Grell, 1968).

Table 5. *Percent recombination between Gl and Sb after successive generations of backcrossing to Harwich and Canton-S females*

Generation tested	Harwich♀♀		Canton♀♀	
	Harwich/ <i>Gl Sb</i> ♂♂	Canton/ <i>Gl Sb</i> ♂♂	Harwich/ <i>Gl Sb</i> ♂♂	Canton/ <i>Gl Sb</i> ♂♂
F <sub>1</sub> (B)	9.7 (2803)	—	—	8.9 (4130)
2	10.8 (2367)	—	—	11.8 (2185)
3	10.5 (1147)	—	—	11.8 (1956)
4	10.8 (1789)	—	—	12.4 (1662)
5	12.1 (1314)	11.7 (1834)	20.0 (925)	12.2 (1344)
9	9.6 (1433)	12.2 (1798)	27.4 (776)	12.7 (2087)
12	9.3 (900)	10.1 (898)	25.6 (1279)	13.2 (1030)

Harwich and Canton-S stocks produced no large changes in female recombination frequencies. In both sets, these values were within the 10–12% range as were the progeny of all three generations of Harwich female × Canton-S male intercrosses. In marked contrast, females from all three generations of reciprocal intercrosses (Canton-S females × Harwich males) produced recombination frequencies which were approximately double those of all the other tests. In addition, the low numbers of progeny observed in the high recombination intercross groups are a partial reflexion of the high sterility and low productivity of these hybrid females, providing confirming evidence of hybrid dysgenesis.

These results show unambiguously that the observed intercross reciprocal

differences are attributable to A cross increases rather than B cross decreases in female recombination frequency. The female recombination results closely parallel those of male recombination in the same interval. Significant frequencies of male recombination were observed concurrently in the progeny of the same Canton-S female  $\times$  Harwich male intercrosses but not in the progeny of the reciprocal cross nor in either of the backcross lines (see Kidwell, Kidwell & Sved, 1977).

(iii) *Coincidence values in reciprocal crosses*

It was of interest to ascertain whether changes in interference between adjacent chromosome intervals accompanied reciprocal differences in recombination values. Estimates of coincidence coefficients and their standard errors (Bailey, 1961) are presented in Table 6 for the second chromosome and Table 7 for the third

Table 6. *Coincidence estimates for adjacent second chromosome intervals*

Intervals*	Cranston/ <i>al cl b c sp</i> <sup>2</sup>		Oregon R-C (I)/ <i>al cl b c sp</i> <sup>2</sup>	
	A	B	A	B
1 and 2	0.22 $\pm$ 0.06	0.19 $\pm$ 0.04	0.35 $\pm$ 0.06	0.21 $\pm$ 0.05
2 and 3	0.86 $\pm$ 0.09	0.88 $\pm$ 0.09	0.81 $\pm$ 0.07	0.98 $\pm$ 0.09
3 and 4	0.62 $\pm$ 0.07	0.49 $\pm$ 0.07	0.69 $\pm$ 0.07	0.61 $\pm$ 0.06

\* See Table 2 for identification of intervals.

Table 7. *Coincidence estimates for adjacent third chromosome intervals*

Intervals*	Cranston/ <i>rucuca</i>		Oregon R-C (II)/ <i>rucuca</i>	
	A	B	A	B
1 and 2	0.25 $\pm$ 0.02	0.11 $\pm$ 0.04	0.21 $\pm$ 0.06	0.23 $\pm$ 0.08
2 and 3	1.18 $\pm$ 0.19	1.01 $\pm$ 0.27	0.20 $\pm$ 0.20	0.65 $\pm$ 0.35
3 and 4	1.20 $\pm$ 0.26	0.37 $\pm$ 0.25	0.40 $\pm$ 0.39	0.89 $\pm$ 0.59
4 and 5	0.16 $\pm$ 0.09	0.07 $\pm$ 0.07	0	0
5 and 6	0.23 $\pm$ 0.06	0.14 $\pm$ 0.05	0.08 $\pm$ 0.04	0.20 $\pm$ 0.09

\* See Table 3 for identification of intervals.

chromosome. For second chromosome recombination values, interference was high between intervals 1 and 2, low between intervals 2 and 3 and moderate between intervals 3 and 4. There was no significant differences between reciprocal crosses nor between the Cranston and Oregon R-C tests. In the third chromosome, the only significant reciprocal difference in coincidence values was between intervals 3 and 4 in the Cranston tests.

(iv) *Correlation of female and male recombination*

The female recombination experiments described here were originally designed to test two types of strain crosses with different male recombination properties. F<sub>1</sub> A crosses involving Cranston had previously yielded high frequencies of male recombination while those involving Oregon R-C had shown only trivial levels of

male recombination (Kidwell & Kidwell, 1975*a*). Only after the female recombination experiments were completed was it discovered that the two substrains of Oregon R-C, designated I and II, had quite different potentials with respect to male recombination and other dysgenic traits. Oregon R-C (I), which was used in the first and second chromosome female recombination tests, subsequently exhibited 0.24 and 0.66 % minimum male recombination in A cross combinations with *al cl b c sp<sup>2</sup>* and *rucuca* respectively. In contrast, Oregon R-C (II), which was used in the third chromosome female recombination test, showed no male recombination with either marker stock. Thus, crosses between Oregon R-C (I) males and marker stock females are dysgenic but those between Oregon R-C (II) males and the same females do not exhibit hybrid dysgenesis.

The female recombination results reported in earlier sections are summarized in Table 8 in comparison with reciprocal cross male recombination frequencies. In

Table 8. *Comparisons of female recombination reciprocal differences with those of male recombination*

Parental cross	Reciprocal differences in female recombination	Minimum frequency of male recombination (%)	
		Cross A	Cross B
Cranston × <i>y<sup>2</sup> cv v f car</i>	Yes	Not measurable	
Oregon R-C (I) × <i>y<sup>2</sup> cv v f car</i>	Yes	Not measurable	
Cranston × <i>al cl b c sp<sup>2</sup></i>	Yes	0.83	0.07
Oregon R-C (I) × <i>al cl b c sp<sup>2</sup></i>	Yes	0.24	—
ID × <i>al cl b c sp<sup>2</sup></i>	No	0.08	0.04
Cranston × <i>rucuca</i>	Yes	1.39	0.02
Oregon R-C (II) × <i>rucuca</i>	No	0.02	0
Canton × Harwich ( <i>Gl Sb</i> )*	Yes	0.72	0.03

\* Backcross generation 12 (Table 5).

most testable strain combinations, female recombination reciprocal effects and male recombination were closely associated. There was generally internal consistency in the results in that all paternally derived chromosomes from the same wild-type stock behaved in the same way with respect to both male and female recombination. However, reciprocal differences in female recombination have occasionally been observed in some apparently non-dysgenic hybrids (e.g. the crosses *Gl Sb/LVM* × Canton-S and *Gl Sb/LVM* × Ames, reported in Table 4) and thus reciprocal differences may not be exclusively a characteristic of dysgenic crosses. The reason for the differences in behaviour between the two Oregon R-C stocks is not known. One possibility is contamination in the laboratory. This would usually be undetected in any wild-type stock. However, divergence attributable to independent genetic changes cannot be completely ruled out at this time.



## 4. DISCUSSION

The effect of many internal and external factors on recombination, particularly in proximal regions, is well documented (for review, see Bodmer & Parsons, 1962). Mather (1939) suggested that heterochromatic regions of chromosomes, such as the pericentric regions, may be highly sensitive to environmental change. Because of the drastic nature of the events leading to hybrid dysgenesis, it is therefore not particularly surprising that female recombination changes are included among its manifestations. In the present study, reciprocal differences were most prominent in, but not restricted to, proximal regions of the major autosomes. In the third chromosome, at least, proximal reciprocal differences were due to increases in A cross recombination values. Intervals in which B cross values exceeded those of the A cross tended to be located in the X chromosome and in distal regions of the autosomes. Further experiments will be required to determine the generality of these observations and to ascertain whether the differences in the X chromosome and distal intervals are attributable to reduced crossing over in the A cross or to increased crossing over in the B cross.

The possibility that the observed recombination patterns could be directly attributed to inversions or indirectly to their heterologous effects (Schultz & Redfield, 1951) can almost certainly be ruled out. Consistent reciprocal differences in inversion frequencies could only result from inversions newly occurring in  $F_1$  hybrids. It seems most unlikely that these could occur in specific regions at frequencies sufficiently high to account for the present results. In fact, perusal of individual female recombination data for dysgenic crosses provided no evidence for the presence of inversions in any tested chromosome region. Also, crosses involving the ID line, known to carry an inversion, exhibited no reciprocal differences.

Additional independent evidence for an association between reciprocal differences in female recombination and hybrid dysgenesis is provided by Broadhead and J. F. Kidwell (in preparation). They tested female recombination frequencies in the second chromosome distal interval *px-or* in reciprocal crosses between both Harwich and Ottawa and the *a px or* marker stock. Harwich/*px or*  $F_1$  A females showed more than 50% total sterility; in addition, productivity of fertile females was very low. Both fertility and productivity of the Harwich/*px or*  $F_1$  B females were normal and at about the same level as those of both Ottawa/*px or* reciprocal crosses. There were reciprocal differences in recombination for hybrids of the marker stock with both wild-type stocks, but the Harwich dysgenic cross reciprocal difference exceeded that of the non-dysgenic Ottawa cross.

The strong association of reciprocal differences in female recombination with male recombination and sterility suggests that the same underlying event may be affecting recombination in male and female A cross hybrids. However, the size of reciprocal differences in female recombination is often an order of magnitude larger than male recombination reciprocal differences in the same interval. If there is a common underlying cause for male recombination and female reciprocal

recombination effects, its action must therefore be greatly magnified in the case of females or inhibited in the case of males. Also, in common with male recombination, small reciprocal differences in female recombination may exist in all intervals but may not be detected in the case of females because of the masking effect of normal female recombination in experiments of limited size.

Increases in female recombination in dysgenic crosses might be attributable to gonial rather than meiotic crossing over. If gonial crossing over occurred, regional differences might be amplified by the resultant clustering of recombinants and the production of double crossovers by a two-step process (Whittinghill, 1955). The observation of some clustering in male recombination data (Hiraizumi *et al.* 1973; Kidwell & Kidwell, 1976) suggests a gonial origin for some crossover events. Sved (personal communication) has additional evidence that male recombination occurs at the four-strand stage of a premeiotic division. However, Woodruff, Henderson and Thompson (personal communication) observed widespread chromosome fragmentation at male meiosis which suggested an exclusively meiotic phenomenon. The hypothesis of gonial origin of female crossovers could not be tested in the present experiment.

Two earlier studies have provided evidence for cytoplasmic effects on recombination. Thoday & Boam (1956) observed a decline in recombination in the *w-f* region of the X chromosome which they attributed to heritable cytoplasmic effects. Lawrence (1958) examined X chromosome crossing over in two inbred strains, Oregon and Samarkand and their reciprocal F<sub>1</sub> crosses. Reciprocal F<sub>1</sub> differences were highly significant in the distal (*w-m*) segment but negligible in the proximal (*m-B*) segment. The distal region differences were in the direction expected if maternal cytoplasm was active in determining crossing over but the genotype was also involved, with the cytoplasm, in determining the recombination response to changes in temperature. However, the very large reciprocal differences observed in the present study seem to be unprecedented and they seem not to be restricted to crosses between newly collected wild stocks and laboratory stocks.

These findings raise some questions concerning the methods by which standard map distances were estimated. The classical method for measuring female recombination has generally been to employ an initial mating of the A type between wild-type males and marker females (Ives, personal communication) but in most published reports of female recombination, the detail of the initial cross direction seems usually to have been considered too inconsequential to mention (e.g. Sinclair, 1975). The results reported here suggest that standard map distances estimated from type A hybrids may not always be a true reflexion of recombination frequencies in intrastain crosses or in reciprocal interstrain crosses. Also, the extended standard map distances found by Bridges using the Florida wild stock have previously been attributed to the heterologous effects of high inversion frequencies (Ives *et al.* 1953). If both strain effects and cytoplasm-genotype interactions are important in determining recombination frequencies, it is not surprising if deviations from the standard map are found to be commonplace even in the absence of heterologous inversion effects.

The overall results suggest that reciprocal female recombination differences between interstrain hybrids may be widespread and even larger than previously established strain variability. Such large effects on recombination frequencies could have important implications for many types of experimentation and for the release of genetic variability in hybrids between interacting strains.

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