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Effects of autosomal inversions on meiotic exchange in distal and proximal regions of the X chromosome in a natural population of *Drosophila melanogaster*

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Summary

We have investigated the interchromosomal effect of the naturally-occurring paracentric inversions *In(2L)t* and *In(3R)P* on meiotic recombination in two regions of the X chromosome in *Drosophila melanogaster*. Previous authors have suggested that the rate of recombination at the tip of the X chromosome may be substantially higher in some natural populations than values measured in the laboratory, due to the interchromosomal effect of heterozygous autosomal inversions. This suggestion was motivated by observations that transposable elements are not as common at the tip of the X chromosome as predicted by recent research relating reduced meiotic exchange to increased element abundance in *D. melanogaster*. We examined the effects of heterozygous *In(2L)t* and *In(3R)P* on recombination at both the tip and base of the X chromosome on a background of isogenic major chromosomes from a natural population. Both inversions substantially increased the rate of recombination at the base; neither one affected recombination at the tip. The results suggest that the presence of inversions in the study population does not elevate rates of crossing over at the tip of the X chromosome. The relevance of these results to ideas relating transposable element abundance to recombination rates is discussed.

1. Introduction

Recent observations have revealed that transposable elements (TEs) in *Drosophila melanogaster* tend to be more abundant in chromosomal regions in which recombination is reduced (Langley *et al.* 1988; Charlesworth & Lapid, 1989; Charlesworth *et al.* 1992*a*, *b*; Eanes *et al.* 1992; Sniegowski & Charlesworth, unpublished data). These observations are consistent with a theory that per-genome copy numbers of *Drosophila* TEs are contained, in part, by the deleterious fitness consequences of ectopic meiotic exchanges between TE sequences (Langley *et al.* 1988).

An exception to the observed association between TE abundance and reduced recombination in Drosophila is found at the tip of the X chromosome. Recombination is known to be highly suppressed in this region in laboratory females with standard karyotypes (Lindsley & Sandler, 1977), yet TEs are not over-abundant there in samples of X chromosomes from natural populations (Langley *et al.* 1988; Charlesworth & Lapid, 1989). It has been suggested that the interchromosomal effect of inversion heterozygosity (Lucchesi, 1976) might greatly increase recombination at the tip of the X chromosome, relative to its base, in natural populations that harbour autosomal inversions at substantial frequencies (Langley *et al.* 1988; Montgomery *et al.* 1991). Such an increase in recombination, provided it also entails an increase in ectopic exchange, could provide an explanation for why the tip of the X chromosome has not accumulated excess TE sequences.

Non-uniform interchromosomal effects of inversion heterozygosity on meiotic exchange along the X chromosome are well-known in *D. melanogaster* (Sturtevant, 1919; Schultz & Redfield, 1951; Lucchesi & Suzuki, 1968). Different chromosomal rearrangements, however, do not have identical interchromosomal effects, and there is no evidence that increases in recombination are generally greater at the tip of the X chromosome than elsewhere. We tested this possibility using chromosomes extracted from a natural population known to have both considerable inversion heterozygosity and no excess of TEs at the tip of the X chromosome. We measured rates of recombination at the tip and base of the X chromo-

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some in females that were heterozygous for a major autosomal inversion or homozygous for standard sequence autosomes. We made no effort to test recombination in females heterozygous for inversions at both major autosomes, since such karyotypes are expected to be rare in the population and hence will have a negligible effect in determining the population-wide distribution of recombination rates along the X chromosome.

2. Methods

(i) *Study population and wild-type chromosomes*

The wild-type chromosomes for this study were derived from stocks collected near Beltsville, Maryland by Dr Jerry Coyne in 1986 and 1987. This population has been the subject of previous studies of TE abundance and chromosomal distribution (Charlesworth & Lapid, 1989; Charlesworth *et al.* 1992*a*, *b*). There is substantial polymorphism for autosomal paracentric inversions in this population. We chose the inversions *In(2L)t* and *In(3R)P* for the present study because they are the highest in frequency and hence most likely to affect population-wide recombination rates. These inversions are present at frequencies of about 10% in the region of our collection site (Mettler *et al.* 1977; B. Charlesworth, unpublished data).

The wild-type X chromosome used here was used in an earlier study (Charlesworth & Lapid, 1989), which describes the details of its extraction. The wild-type second and third chromosomes (standard arrangement, *In(2L)t* and *In(3R)P*) were isolated as described in Charlesworth *et al.* (1992*a*). All wild-type chromosome stocks were initially marked with the fourth chromosome recessive spa^{pol} to guard against contamination during laboratory maintenance.

(ii) *Proximal X chromosome markers*

The markers *f* (located at 56.7), *car* (located at 62.5) and *su(f)* (located at 65.9) were used to assay exchange frequency at the base of the X chromosome. A chromosome bearing these markers was created by mating virgin *f car/f car* females to *f su(f)* males (stocks obtained from the National Drosophila Stock Center, Bowling Green, Ohio) and selecting F_2 males showing carnation eyes and the suppressed *forked* phenotype (near-wild-type bristles). *car* is located cytologically at 18D1-2, which is the distal boundary of the X chromosome proximal region examined for increased TE abundance in the *in situ* hybridization surveys of Langley *et al.* (1988) and Charlesworth & Lapid (1989).

(iii) *Distal X chromosome markers*

To assay recombination at the tip of the X chromosome, a tester stock bearing the recessives *y*, *pn* and *spl* was obtained from the National Drosophila Stock Center, Bowling Green, Ohio. These markers are at 0.0, 0.8 and 3.0 on the X chromosome (standard map positions from Lindsley & Zimm, 1992). *spl* is located cytologically at 3C7 (Lindsley & Zimm, 1992) which is near the proximal boundary of the X chromosome distal region tested for increased TE abundance in the *in situ* hybridization surveys of Langley *et al.* (1988) and Charlesworth & Lapid (1989).

(iv) *Preliminary crosses*

To facilitate genetic manipulation of the isogenic wild second and third chromosomes, we constructed a stock isogenic for the wild X chromosome, bearing the standard laboratory balancer/recessive lethal second and third chromosome combinations *SM1, Cy/Pm; TM6, Ubx/Sb*, and a fourth chromosome with the recessive markers *ey* and *ci* (obtained from the National Drosophila Stock Center). The individual balancer chromosome stocks had previously been placed, by repeated backcrossing, on a cytoplasmic and genetic background derived from the wild-type, outbred laboratory stock *IV* (described in Charlesworth & Charlesworth, 1985*a*) in order to avoid hybrid dysgenesis in crosses involving wild males. The *ey ci* fourth chromosome was substituted for the spa^{pol} chromosome in our balancer stock because eye colour phenotypes proved difficult to distinguish during genetic manipulation on the spa^{pol}/spa^{pol} background.

The *X/X; SM1, Cy/Pm; TM6, Ubx/Sb; ey ci/ey ci* double balancer stock was used to construct lines isogenic for chromosomes X, 2 and 3 from the wild population following the scheme illustrated in Fig. 1. The following combinations of isogenic wild chromosomes derived from the Maryland study population, marked with the *ey ci* fourth chromosome, were constructed and subsequently maintained: *X/X; 2/2; 3/3, X/X; In(2L)t/In(2L)t; 3/3,* and *X/X; 2/2; In(3R)P/In(3R)P*.

The *y pn spl* and *f car su(f) X* chromosome marker combinations were placed on backgrounds harbouring isogenic wild autosomes as follows. For the *y pn spl* chromosome, virgin *y pn spl/y pn spl* females were crossed to males of the genetic constitution *+; SM1,Cy/+; TM6,Ubx/+; ey ci/ey ci*, and their male offspring of constitution *y pn spl; SM1,Cy/+; TM6,Ubx/+; ey ci/+* were backcrossed to virgin *y pn spl/y pn spl* females to establish a stock of constitution *y pn spl/y pn spl; SM1,Cy/+; TM6, Ubx/+*, which was maintained by selection. In succeeding generations, individuals of this stock homozygous for the *ey ci* fourth chromosome were interbred, and the resulting *y pn spl/y pn spl; SM1,Cy/+; TM6,Ubx/+; ey ci/ey ci* stock was maintained by selection. Virgin females of this stock were then crossed to males of the triply isogenic *X/X;*

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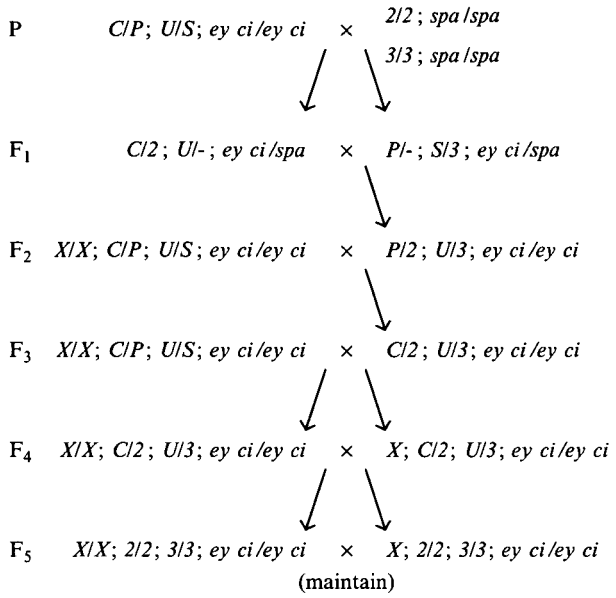

Fig. 1. Breeding scheme used to construct a stock isogenic for chromosomes X, 2 and 3 from the wild population, bearing the *ey ci* fourth chromosome. In the figure, *C* represents the *SM1,Cy* balancer chromosome, *P* represents *Pm*, *U* represents the *TM6,Ubx* balancer chromosome and *S* represents *Sb*.

2/2; *3/3*; *ey ci/ey ci* stock. Female offspring of constitution *X/y pn spl*; *SM1,Cy/2*; *TM6,Ubx/3*; *ey ci/ey ci* and males that were *y pn spl*; *SM1,Cy/2*; *TM6,Ubx/3*; *ey ci/ey ci* were selected from these crosses and interbred. Their male and female offspring showing the *y pn spl* phenotype and bearing the isogenic major autosomal complement *2/2*; *3/3* derived from the wild population were interbred, and the resulting *y pn spl/y pn spl*; *2/2*; *3/3*; *ey ci/ey ci* stock was retained for use in crosses detailed below.

Because of the difficulty of unambiguously recovering nonrecombinant *f car su(f)* chromosomes after possible recombination with the wild X chromosome, a different scheme was used to place this combination of loci in the same genotype as the isogenic wild autosomes. Virgin *f car su(f)/f car su(f)* females were crossed to males of the genetic constitution +; *SM1,Cy/+*; *TM6,Ubx/+*; *ey ci/ey ci* and their male offspring of constitution *f car su(f)*; *SM1,Cy/+*; *TM6,Ubx/+*; *ey ci/+* were then backcrossed to virgin *f car su(f)/f car su(f)* females to establish a stock of constitution *f car su(f)/f car su(f)*; *SM1,Cy/+*; *TM6,Ubx/+*, which was maintained by selection. In succeeding generations individuals of this stock homozygous for the *ey ci* fourth chromosome were interbred, and the resulting *f car su(f)/f car su(f)*; *SM1,Cy/+*; *TM6,Ubx/+*; *ey ci/ey ci* stock was maintained by selection. Virgin females from this stock were then crossed to males from the triply isogenic *X/X*; *2/2*; *3/3*; *ey ci/ey ci* stock and males of the constitution *f car su(f)*; *SM1,Cy/2*; *TM6,Ubx/3*; *ey ci/ey ci* were selected for use in crosses to obtain heterozygous females.

(v) *Crosses to obtain heterozygous females and recombinant offspring*

To produce females heterozygous for the proximal X chromosome marker loci with and without inversion heterozygosity in a major autosome, virgin females from the stocks isogenic for the major chromosomes derived from the Maryland population were mated to males of the constitution *f car su(f)*; *SM1,Cy/2*; *TM6,Ubx/3*; *ey ci/ey ci*. Virgin female offspring carrying neither balancer chromosome, as shown in Table 1, were then selected and backcrossed to males of the original *f car su(f)* stock. The resulting offspring were classified phenotypically.

To obtain females heterozygous for the distal X chromosome marker loci with and without inversion heterozygosity in one of the major autosomes, virgin females from the *y pn spl/y pn spl*; *2/2*; *3/3*; *ey ci/ey ci* stock were mated to males from the different stocks made triply isogenic for wild-type major chromosomes from the Maryland population. The resulting chromosomal constitutions of female offspring are shown in Table 2. These females were either backcrossed to tester *y pn spl* males and their male and female offspring classified phenotypically; or crossed to males from a wild-type stock, in which case only male

Table 1. *Recombination in the* f car su(f) *region*

	Progeny phenotypes		
f car su(f)/X females	Parental	*Forked*; wild-type eye	*Forked*; carnation eye
In(2L)t/2; *3/3*	5214	185 (3·3%)*	154 (2·8%)*
2/2; *3/3*	1482	34 (2·2%)	22 (1·4%)
2/2; *In(3R)P/3*	4848	172 (3·3%)**	158 (3·1%)***
2/2; *3/3*	4455	107 (2·3%)	90 (1·9%)

Asterisks indicate significantly higher recombinant fractions in offspring of inversion heterozygotes (two-tailed Fisher's exact tests: $*P < 0{\cdot}05$; $**P < 0{\cdot}01$; $***P < 0{\cdot}001$). The overall fraction of recombinants (both classes pooled) is significantly higher ($P < 0{\cdot}001$) in progeny of inversion heterozygotes in both comparisons.

Table 2. *Recombination in the* y pn spl *region*

	Progeny		
y pn spl/X females	*y pn spl*, + + +	*y* + +, + *pn spl*	*y pn* +, + + *spl*
In(2Lt)/2; *3/3*	8007	10 (0·1%)	146 (1·8%)
2/2; *3/3*	4329	5 (0·1%)	68 (1·5%)
2/2; *In(3R)P/3*	8940	6 (0·07%)	145 (1·6%)
2/2; *3/3*	3503	7 (0·2%)	69 (1·9%)

No significant differences in recombination rate were observed between inversion heterozygotes and non-heterozygotes.

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offspring could be classified. (Some crosses were made with wild-type males because crosses with *y pn spl* males go poorly and produce relatively few offspring.)

(vi) *Progeny rearing and scoring*

Flies for all crosses were raised on standard agar-yeast-soyameal-cornmeal medium in 8-dram vials at 25 °C on a 12-h light–dark cycle. Three female parents per vial were used for the crosses providing recombinant progeny. Crosses with and without inversion heterozygosity were started at the same time, and vials were kept in a random arrangement on the same incubator shelf throughout the experiment. Parents were removed from vials upon appearance of offspring pupae, and progeny were counted until ten days after first eclosion. Vials for crosses were coded such that scoring of progeny was done without knowledge of whether the female parent was an inversion heterozygote.

3. Results

Results are shown in Tables 1 and 2. The total number of offspring scored was 42156. All significance levels reported in comparisons of recombination rate between inversion heterozygotes and non-heterozygotes were obtained by two-tailed Fisher's exact tests; within each table, probabilities were adjusted by the sequential Bonferroni method (Rice, 1989).

(i) *Recombination at the base of the X chromosome*

Both inversions increased recombination at the base of the X chromosome (Table 1). Heterozygous *In(2L)t* increased the fraction of recombinants between *f* and *su(f)*, the outer markers used in this region, from 3·6% to 6·1% ($P < 0{\cdot}001$). Heterozygous *In(3R)P* increased the fraction of recombinants between *f* and *su(f)* from 4·2% to 6·4% ($P < 0{\cdot}001$). The increase in recombination was apparently evenly distributed between the intervals *f-car* and *car-f*; in other words, there was no evidence that one interval was more strongly affected by inversion heterozygosity than the other. (For the comparison of recombination increase in intervals *f-car* and *car-f* between *In(2L)t* heterozygotes and non-heterozygotes, $P = 0{\cdot}468$; for the same comparison between *In(3R)P* heterozygotes and non-heterozygotes, $P = 0{\cdot}653$.)

Since the *su(f)* allele is indistinguishable from its wild-type counterpart on a non-*f* background, it was not possible to detect double recombinants among these loci. For this reason, also, the fraction of recombinants detectable among the progeny of *f car su(f)/X* females is expected to equal half the total fraction of viable recombinants. Accurate discrimination between *f+* and *f su(f)* progeny was made difficult by some overlap in the bristle phenotypes of these two classes, and thus the data from homokaryotypic females should not be taken as measures of the 'true' map distances between the three loci. This difficulty in scoring could not have affected the direction of the results, however, since vials from the different crosses were coded to preclude observer bias.

(ii) *Recombination at the tip of the X chromosome*

As shown in Table 2, neither inversion increased the rate of recombination at the tip of the X chromosome, whether between the outer markers *y* and *spl* or in the intervals *y-pn* and *pn-spl*. A slight increase in the fraction of *y-pn* interval exchange progeny was observed in the offspring of homokaryotypic females relative to those heterozygous for *In(3R)P*, but this is not significant by a two-tailed Fisher's exact test ($P = 0{\cdot}059$) even before any correction for multiple tests is made. Although the low numbers of recombinants observed in the *y-pn* interval weakened statistical detection of increased exchange there, the results for the adjacent *pn-spl* interval give a clear indication that recombination is not increased when an inversion is present. No double-recombinant (*y+spl* or *+pn+*) progeny were observed.

4. Discussion

This study provides no indication that the relatively common paracentric inversions *In(2L)t* and *In(3R)P* increase the overall rate of recombination at the tip of the X chromosome, relative to its base, in the study population. Both inversions increased recombination at the base of the X chromosome when heterozygous, but had no detectable effect on recombination at the tip.

In contrast to our results for the tip region, Montgomery *et al.* (1991) found that simultaneous heterozygosity for the multiply-inverted laboratory balancers *SM1* and *TM3* increased normal meiotic recombination and transposon-associated rearrangement in the *y-spl* interval approximately 3-fold. It is, therefore, clear that meiotic exchange at the tip of the X chromosome can be affected by inversion heterozygosity in the autosomes. However, since Montgomery *et al.* (1991) only examined the effect of *SM1*; *TM3* heterozygosity on recombination at the tip of the X chromosome, the possibility that recombination at the base was equally or even more strongly affected cannot be ruled out. Moreover, their result represents an extreme case, since the multiple inversions on laboratory balancer chromosomes drastically reduce recombination with the homologue over the entire chromosome, with corresponding increases in interchromosomal effect (Roberts, 1976).

A possible alternative explanation for our results might be that the effect of inversion heterozygosity on recombination at the tip was 'masked' by some other genetic factor present on one of the inversion chromosomes or on one of the isogenic standard chromosomes. There is good evidence for substantial

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genetic variation for recombination rate in *Drosophila*. Charlesworth & Charlesworth (1985*a*, *b*) observed significant responses in *D. melanogaster* to artificial selection on recombination between two third-chromosome dominant visible mutations on a largely wild-type genetic background. Subsequent mapping experiments and analysis suggested that single genes or tightly linked factors with partially dominant effects were responsible for much of the observed response. (It would seem necessary to invoke dominant effects to explain any suppression of recombination in the present experiment, since each isogenic autosome was heterozygous in half of the parental females: see Tables 1 and 2.)

In another study, Brooks & Marks (1986) analysed the effects on recombination rate of six second chromosomes extracted from a natural population of *D. melanogaster*. They found considerable variation among these chromosomes in their effects on recombination along standard, multiply marked X, second and third chromosomes, including evidence (as given above) that the factors modifying recombination had partially dominant effects. Brooks & Marks also noted that factors modifying recombination along the second chromosome displayed effects of equal sign on recombination along the other chromosomes, in contrast to the negative associations observed in the interchromosomal effect of inversions (Schultz & Redfield, 1951; Lucchesi, 1976). It should be noted, however, that another study found that a third-chromosome line selected to increase crossing over with its homologue nonetheless *decreased* crossing over on the second chromosome (Charlesworth *et al.* 1985), so that the pattern of positive associations of effects on recombination revealed in the Brooks & Marks (1986) study may not hold generally.

It is conceivable that a *trans*-acting suppressor of recombination such as those in the above studies was present on one of the isogenic wild autosomes used here. However, the magnitude of effects of the genetic factors altering recombination rates as measured in these studies does not seem sufficient to mask the kind of strong increase in recombination at the tip that was sought in our study. The greatest estimated difference in effects on recombination (difference between highest and lowest observed rates) among lines in the above studies was 38% for the *y-cv* interval on the X chromosome (Brooks & Marks, 1986; table 4). In the data reported here, heterozygous autosomal inversions induced an increase of approximately 60% in recombination at the base of the X chromosome (Table 1); it seems unlikely that even greater increases at the tip (that is, a positive result) could have been completely masked by the effects of recombination-modifying factors on the autosomes.

Another possible explanation for these results is that a dominant recombination suppressor (e.g. an inversion) on the isogenic wild-type X chromosome or the *y pn spl* chromosome was responsible for the insensitivity of recombination in the *y pn spl* region to autosomal inversion heterozygosity. No inversion was cytologically detectable on these chromosomes, but the possibility that a cytologically undetectable inversion was present cannot be ruled out.

On balance, the results of this experiment fail to provide an explanation for why the tip of the X chromosome does not conform to the general pattern of increased TE abundance in areas of reduced recombination that has been revealed by *in situ* hybridization surveys (Langley *et al.* 1988; Charlesworth & Lapid, 1989; Charlesworth *et al.* 1992*a*, *b*). We have obtained preliminary data bearing on another possible explanation that are also negative. It is well-known that changes in temperature alter the rate and distribution of recombination along the Drosophila chromosomes (Plough, 1917, 1921; Stern, 1926). We tested whether temperatures other than 25 °C increase recombination at the tip of the X chromosome by rearing standard karyotype females heterozygous for the *y pn spl* combination at 18 °C and 28 °C and counting recombinant progeny. Although the samples of progeny were relatively small (approximately 500 at each temperature), we found no evidence that recombination between *y* and *spl* was much higher at these temperatures than at 25 °C. Recombinant fractions for the *y-spl* interval were 2·2% at 18 °C and 1·4% at 28 °C (J. Burdette, personal communication); these are not statistically different from the 25 °C values reported in Table 2.

Because there remains a lack of data on whether the rate of ectopic exchange events is correlated with the rate of normal meiotic exchange events in different *Drosophila* chromosome regions, the results of this study cannot be interpreted as definitely inconsistent with the ectopic exchange model. It is possible that the rate of occurrence of ectopic exchange events involving TEs at the tips of chromosomes is comparable with that for middle regions. It is interesting to speculate that perhaps TEs at tips of chromosomes are free to interact ectopically with other TEs in the freely-recombining middle regions of chromosomes, even though normal meiotic exchange is suppressed at the tip.

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