INTERCHROMOSOMAL EFFECTS OF HETEROCHROMATIC DELETIONS ON RECOMBINATION IN *DROSOPHILA MELANOGASTER*

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ABSTRACT

It is now known that partial deletions of the satellite sequences in *X*-chromosome heterochromatin result in a significant decrease in intrachromosomal recombination in the proximal region of the *X* chromosome of *D. melanogaster* (YAMAMOTO and MIKLOS 1978). It is important to ask then if the loss or gain of heterochromatin on the *X* also alters recombination in other chromosomes of the genome (interchromosomal effects). I have looked for such alterations by measuring recombination in chromosome *3*. The results clearly indicate that the partial loss of *X*-chromosome heterochromatin not only decreases crossing over in the proximal region of the *X* chromosome itself, but also increases the frequency in chromosome *3*, especially in the euchromatic regions around the centromere. Furthermore, the greater the deficiency of *X* heterochromatin, the higher is recombination in chromosome *3*. This finding not only provides further evidence in support of the hypothesis that heterochromatin, in this case mainly composed of satellite DNA, regulates the recombination system, but it demonstrates that when the satellite content of one chromosome of the *D. melanogaster* genome is altered, there is an alteration in the crossover characteristics of other chromosomes in the same complement. If the amount of satellite DNA in a genome is being continuously altered, then one can predict that the recombination system is also being continually perturbed. Thus, the changing gene combinations produced indirectly by increases or decreases of heterochromatin are among the components available to organisms to break up or form new gene combinations upon which selection can act.

THE functions of constitutive heterochromatin, which is composed of satellite DNA, has long been a mystery despite the many hypotheses that have been proposed (WALKER 1971a,b; YUNIS and YASMINEH 1971; HSU 1975; JONES 1976; SINGH, PURDOM and JONES 1976; HATCH *et al.* 1976; MIKLOS and NANKIVELL 1976; PEACOCK *et al.* 1977; FRY and SALSER 1977). Most of these hypotheses, however, have yet to be critically examined in appropriate experimental systems. Because of this, there is an astonishing lack of data on which they can be evaluated.

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In *Drosophila melanogaster*, the amount of heterochromatin and its arrangement in the genome can easily be manipulated genetically, and by this means the predictions of some of the hypotheses on satellite DNA function have actually been tested either genetically or cytologically (YAMAMOTO and MIKLOS 1977, 1978; YAMAMOTO 1979). The results to date indicate that, while some hypotheses, such as satellite DNA involvement in homolog recognition, are untenable, others, such as the role of heterochromatin in regulating the recombination system, have hard data in their support.

Recently, YAMAMOTO and MIKLOS (1978) demonstrated that the systematic alteration of the heterochromatic content of the *X* chromosome yielded significant changes in the frequency and the position of crossing over within the *X* chromosome itself. When an *X* chromosome is deficient for some of its heterochromatin, both homozygous deficient and heterozygous deficient females exhibit substantially decreased recombination frequencies in the *X* chromosome, especially in the proximal regions near the centromeric heterochromatin. It has been known since the time of BEADLE (1932) that the centromere itself has an inhibitory effect on recombination in its vicinity. It was concluded, therefore, that the satellite sequences in the heterochromatin could function simply as a spacer between the centromere and the euchromatin. Thus, the amount of heterochromatin would influence the extent of recombination inhibition in the proximal region of the *X* chromosome.

Furthermore, since spontaneous SCE events were shown to occur in a satellite DNA-rich ring *Y* chromosome, YAMAMOTO and MIKLOS (1978) suggested that ongoing unequal SCE within the heterochromatin could be one mechanism to increase or decrease the amount of satellite DNA on a chromosome and thereby to alter the amount of recombination.

There have been extensive studies on alterations in the recombination system within structurally rearranged chromosomes (intrachromosomal effects), as well as on effects between chromosomes (interchromosomal effects). The well-established phenomena in this regard are summarized for Drosophila by LUCCHESI and SUZUKI (1968) and SUZUKI (1973). Most commonly, some types of chromosomal rearrangements, such as inversions, translocations and compound chromosomes, are known to cause alterations in recombination on other members of the genome. For example, increases in crossing over in the third chromosome, especially around the centromere, are known to be induced by such rearrangements. In general, rather complex systems have been used to study such interchromosomal effects. These have involved inversions and translocations having different breakpoints in either heterochromatin or euchromatin, systems in which, the amount of heterochromatin has varied, the variation has not been systematic. Generally, however, such chromosomal rearrangements have failed to assess directly the interchromosomal influence of heterochromatin on either recombination or segregation.

It is important to know if interchromosomal effects can be caused by systematic alterations of the *X* chromosome centromeric heterochromatin itself. If this is the case, it would clearly indicate the possibility that the increases or decreases of

satellite DNA *per se*, on any chromosome, can modulate the recombination properties of other chromosomes, thus exerting some control over the genetic variation in natural populations.

MATERIALS AND METHODS

Chromosomes

Heterochromatically deleted X *chromosomes:* The deleted *X* chromosomes used in this study contain a deficiency for the nucleous organizer and some of the basal *X* heterochromatin. They are Df(1)bobbed lethal, Df(1)bobbed lethal-158 and Df(1)bobbed lethal-452 (LINDSLEY, EDINGTON and VON HALLE 1960; LINDSLEY and GRELL 1968). Hereafter, they are designated as bb^{l}, bb^{l158} and bb^{l452}. These chromosomes are free of euchromatic rearrangements and the relative size of their proximal heterochromatin to that of a normal *X* chromosome is as follows: bb^{l} (0.8), bb^{l452} (0.7), bb^{l158} (0.2). Thus, they differ in the amount of heterochromatin they contain (see Figure 1 in YAMAMOTO and MIKLOS 1978). All bobbed lethal chromosomes used carry the marker *y*.

Normal chromosomes: Chromosomes used in these experiments were: (1) a normal wild-type *X* chromosome, (2) a normal *X* chromosome with the marker, *y* (yellow), (3) *Dp(1;1)sc*^{v1}, *y*·*y*+, a heterochromatically normal *X* chromosome, (4) a marked *Y* chromosome, $B^{S}Y$, and (5) a normal chromosome 3 marked with *ru* (roughoid), *h* (hairy), *st* (scarlet), p^{p} (pink-peach), *ss* (spineless) and e^{s} (ebony-sooty). Hereafter, this chromosome is designated as "threepl" and was obtained from the Bowling Green Stock Center. *Dp (1;1)sc*^{v1}, *y*·*y*+ is cytologically indistinguishable from a normal *X* chromosome in length at mitotic metaphase, but it carries a very small euchromatic duplication including the wild-type allele of *y* at the tip of the short right arm of the *X* chromosome.

Stock construction

The same genetic background is essential in determining the interchromosomal effects between the experimental and the control flies. For this reason, the experimental females were strictly synthesized in the following way to control their genetic background.

The crossing procedure was as follows:

bobbed lethal/bobbed lethal/$B^{S}Y$ ♀ × *Dp(1;1)sc*^{v1}/$B^{S}Y$ ♂

↓

Dp(1;1)sc^{v1}/bobbed lethal ♀ × "threepl" ♂

↓

$$\frac{\text{bobbed lethal}}{\text{normal } X} ; \frac{\text{normal 3}}{\text{"threepl"}} \text{ ♀} \quad \text{(Exp.)}$$

$$\frac{Dp(1;1)sc^{v1}}{\text{normal } X} ; \frac{\text{normal 3}}{\text{"threepl"}} \text{ ♀} \quad \text{(Cont.)}$$

Full sibs

The distinction between the control and experimental genotypes was made by assessing the ratio of the number of sons with yellow body color to those with wild-type body color among the progeny of a single female. In all experimental crosses, two- to three-day-old virgin females were individually crossed to two Canberra wild-type males and subsequently transferred three times to new food under standard conditions in shell vials of 25°. All progenies were scored for recombination frequencies in chromosome 3.

Analysis of the data

Tetrad analyses were calculated as previously described, and 95% confidence limits were determined from the formula used by SUZUKI (1962), except that a minor but important alteration was made in that formula so as not to yield a negative square root.

RESULTS

The general outline of the experiments and the properties of the chromosomes involved are shown in Figures 1 and 2. The regions in which recombination was measured are shown on the mitotic map of chromosome 3 (Figure 1), together with the amount of recombination in each region as estimated from controls. The relevant experimental and control chromosomal constitutions are presented in Figure 2, the object of the experiment being to determine if recombination in chromosome 3 is altered by deletions of the *X* heterochromatin. It is already known that recombination is decreased in the proximal region of the *X* chromosome, when *X* chromosomes are heterozygous or homozygous for heterochromatic deletions (YAMAMOTO and MIKLOS 1978).

Table 1 summarizes the map distances in the third chromosomes measured in females heterozygous for the heterochromatically deficient bobbed-lethal *X* chromosome and a normal *X* chromosome. In order to compare those map distances more meaningfully in each set of experiments, I have taken the ratio of the experimental values obtained to their respective control values, as has been customary in previously published interchromosomal effects.

Comparison of the map distances in each region of chromosome 3 reveals a typical interchromosomal effect, that is, recombination is maximally enhanced in the region spanning the centromere. In the case of the largest *X* heterochromatic deficiency (bb^{l158}), recombination in region 3 is 52% higher than the control, with values in other regions being much lower (Figure 3). Similarly, between bb^{l452} and its control, with the exception of region 5, the crossover values in the experimental case are again larger than the control, but the maximum increase is only 14%.

The ratio of experimental to control map distances (*E/C*) in any region is a convenient means of assessing alterations in recombination. The results in Table 1, where the experimental and control females are full sibs, clearly shows the

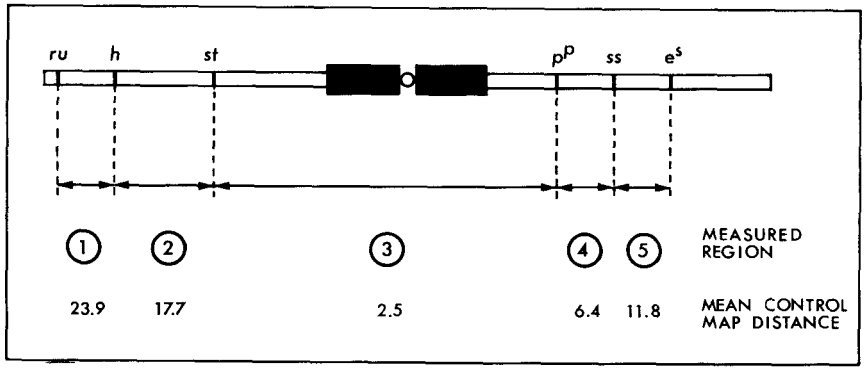

FIGURE 1.—Diagrammatic representation of the regions in which recombination was measured, and the locations of "threepl" markers on the mitotic chromosome 3. The thin white blocks represent euchromatin and the thick black blocks represent heterochromatin. The mean control map distance in each region is also presented.

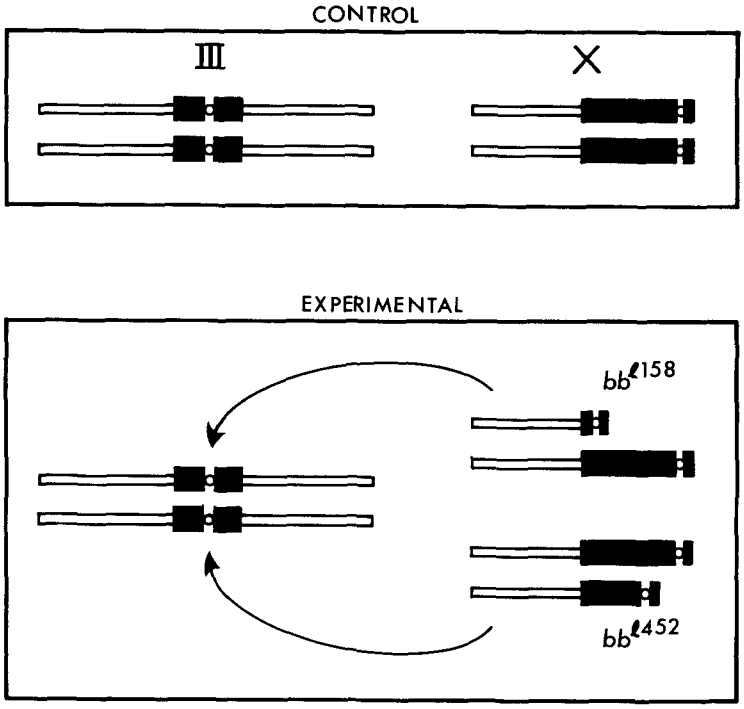

FIGURE 2.—Diagrammatic representation of the relevant chromosomal constitutions of the control and the experimental systems. The relative size of the proximal heterochromatin of the two deleted *X* chromosomes, bb^{l158} and bb^{l452}, to that of a normal *X*, is 0.2 and 0.7, respectively.

increase in recombination in chromosome 3 in the region spanning the centromere. Cases in which the experimental and control genotypes are not full sibs also yield very similar results, though the effect is certainly modified by the background genotype (Table 2). Once again, there is an almost identical increase in recombination in region 3 in the bb^{l158}/normal *X* genotype. The homogeneity of the results is seen in Table 3 where the three control results and the three

TABLE 1

Recombination measured in chromosome 3 in genotypes of the constitution normal X/normal X (control) or deleted X/normal X (experimental) females, which are sisters

	Female genotype	Map Distances Region 1 *ru-h*	Region 2 *h-st*	Region 3 *st-p^p*	Region 4 *p^p-ss*	Region 5 *ss-e^s*	Total Progeny
	bb^{l452}/normal *X*	23.9	18.5	3.2	7.6	11.3	3955
Exp. 1	Control	23.1	16.9	2.8	6.6	11.3	3610
	% Increase	3%	9%	14%	15%	—	
	bb^{l158}/normal *X*	25.8	20.3	3.2	9.0	12.3	3757
Exp. 2	Control	24.6	18.5	2.1	7.1	12.2	2328
	% Increase	5%	10%	52%	27%	1%	

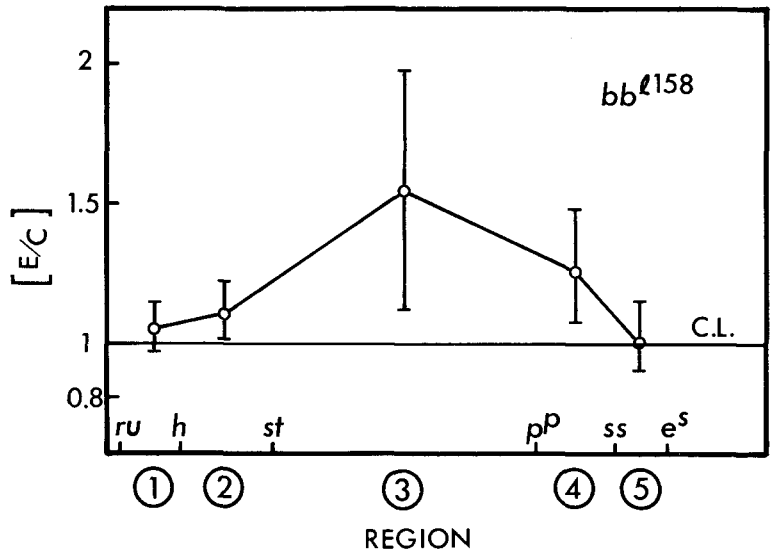

FIGURE 3.—Ratio of map distance in each region in a female heterozygous for bb^{l158} and a normal X to that of its control map distance (E/C). C.L. indicates the control level. 95% confidence limits are shown by vertical lines.

TABLE 2

Recombination measured in chromosome 3 in genotypes that are not sibs, but that have genetic backgrounds similar to those of Experiments 1 and 2

	Female genotype	Map Distance Region 1 *ru-h*	Region 2 *h-st*	Region 3 *st-p^p*	Region 4 *p^p-ss*	Region 5 *ss-e^s*	Total Progeny
Exp. 3	normal X/normal X	23.9	19.5	2.4	8.1	11.4	1580
Exp. 4	bb^{l}/normal X	24.7	17.8	1.7	9.2	13.6	1116
Exp. 5	bb^{l158}/normal X	26.3	19.1	3.6	7.6	12.2	1236
Exp. 6	bb^{l158}/normal X	23.8	18.5	3.1	7.5	13.8	1780

TABLE 3

Summary of the five recombination experiments involving normal X/*normal* X *and* bb^{l158}/*normal* X *genotypes*

Female genotype	Expt. No.	Region 1	Region 2	Region 3	Region 4	Region 5
normal X/normal X	Exp. 1	23.1	16.9	2.8	6.6	11.3
	Exp. 2	24.6	18.5	2.1	7.1	12.2
	Exp. 3	23.9	19.5	2.4	8.1	11.4
bb^{l158}/normal X	Exp. 2	25.8	20.3	3.2	9.0	12.3
	Exp. 5	26.3	19.1	3.6	7.6	12.2
	Exp. 6	23.8	18.5	3.1	7.5	13.8

experimental results relating to bb^{l158}/normal X females are summarized. It is evident from the Figure 3 that the deletion of the proximal heterochromatin of the X chromosome has resulted in an increase in recombination in chromosome3, especially in the markers close to the centromere.

One question that immediately arises in evaluating the interchromosomal effect observed in bb^{l158} and bb^{l452} heterozygotes is whether the increase in crossing over in region 3 is due simply to the lack of a nucleolus organizer. Another heterochromatic deficient X chromosome, bb^{l}, was employed in which the deficiency is even smaller than that in bb^{l452}. The results of using this chromosome are shown in Table 2. Even though in this case bb^{l}/normal X does not have a full-sib control, its recombination values are very close to those of the control. In fact region 3 is slightly lower than that of the control. Thus, a deficiency of virtually only the nucleolus organizer itself does not cause such an interchromosomal effect. Therefore, the increase in bb^{l158}/normal X and bb^{l452}/normal X is due to the deficiency of the proximal heterochromatin *per se*.

The results of experiments 1 and 2 are remarkable. As the size of the heterochromatic deficiency in the X chromosome increases, recombination in most

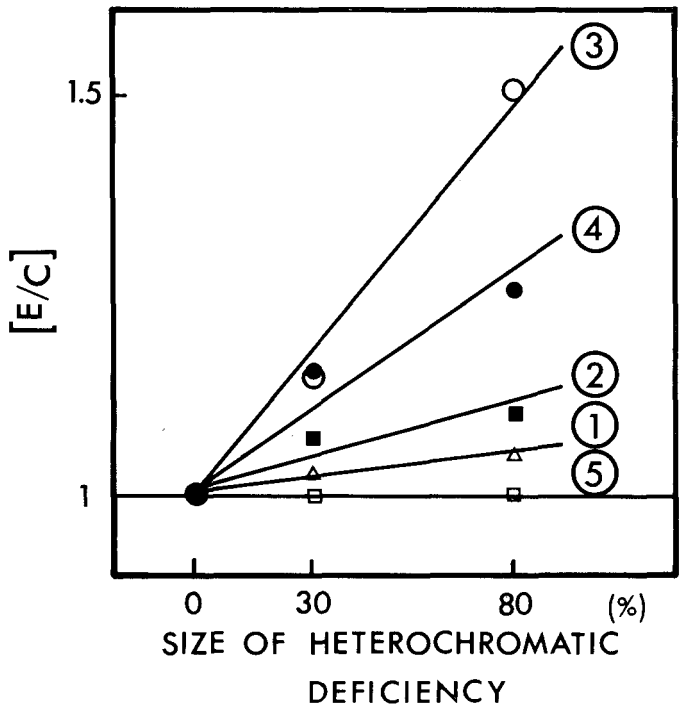

FIGURE 4.—Comparisons of the recombination levels (E/C) in each region of chromosome 3 for a control, bb^{l452} (30% deficiency in heterochromatin) and bb^{l158} (80% deficiency in heterochromatin) heterozygotes. Clearly, as the distance from the centromere increases, there is less of an interchromosomal effect. Within each region the effect decreases from bb^{l158} through bb^{l452} to the control value, that is, the effect in a region decreases as the size of the experimental X heterochromatin approaches that of the control.

TABLE 4

Partial tetrad analysis of chromosome 3 in deleted X/normal X females and their controls

Female genotype	E_0	E_1	E_2	E_3
bb^{l452}/normal *X*	9.0	55.7	32.7	2.6
Control	14.4	51.6	32.2	1.8
bb^{l158}/normal *X*	6.4	49.6	41.0	3.0
Control	13.9	46.8	36.9	2.4

regions of chromosome 3 also increases, with a maximum effect in region 3 (Figure 4). The effect occurs in all regions, but with different magnitudes, as is seen by the slope of the lines in Figure 4.

Partial tetrad analysis of the data also shows that recombination on chromosome 3 is increased. In standard terminology, a noncrossover event is denoted E_0, a single crossover E_1, and so forth. Since the entire third chromosome was not marked for recombination, only a partial tetrad analysis is possible. Even so, it is abundantly clear from Table 4 that, as the amount of *X* heterochromatin is decreased (*i.e.*, size of deficiency is increased), crossing over in chromosome 3 increases as seen from the lowered frequency of no-exchange tetrads (E_0's). The decrease of E_0's and the increase of E_1's and E_2's has been found in many other cases involving interchromosomal effects on recombination (REDFIELD 1957; STEINBERG and FRASER 1944; SUZUKI 1962, 1963).

In summary, then, in a very strictly controlled genetic background and using only full sibs, the increase in recombination in chromosome 3 appears to be directly correlated with the size of the heterochromatic deficiency in the *X* chromosome.

DISCUSSION

Previously published interchromosomal effects have been studied in a variety of chromosomal constitutions of *Drosophila melanogaster* females. They have also involved many different chromosomal rearrangements, such as inversions, translocations, compound chromosomes and an added *Y* chromosome. It is mainly because of the complications of the experimental systems in which interchromosomal effects have been previously demonstrated that the influence of the amount of centromeric heterochromatin on recombination has not been recognized an important factor in generating interchromosomal effects. However, prior to the present study, no systematic experiments have been carried out to determine specifically whether the amount of heterochromatin *per se* is capable of influencing recombination interchromosomally. The results of simple systematic deletions of the proximal heterochromatin *per se*, in a strictly controlled background, have clearly demonstrated that the amount of heterochromatin certainly affects the recombination system.

As has been previously and extensively discussed (STEINBERG 1936; SCHULTZ and REDFIELD 1951; LUCCHESI and SUZUKI 1968; SUZUKI 1973), interchromosomal effects appear to be quite complicated in nature. Particular chromosome changes may cause effects that are specific in both manner and intensity. As RAMEL (1962), SUZUKI (1962, 1963) and WILLIAMSON (1966) have suggested, more than one mechanism appears to be required to explain the variety of these effects. Irrespective of the precise mechanism by which it occurs, however, alterations in the amount of satellite DNA in the centromeric heterochromatin of the *X* chromosome certainly cause changes in recombination in chromosome *3*. It is likely that the deletion of *X* chromosome basal heterochromatin will also affect crossing over in chromosome *2*, since we know that interchromosomal effects generated by *X* chromosome rearrangements influence both chromosomes *2* and *3*. Additionally, a heterochromatic deletion in an autosome may also lead to interchromosomal effects comparable to that caused by deletions of the *X* chromosome because, in the case of the *X* heterochromatin, the effect is dependent simply on the size of the heterochromatin segment involved and not on its satellite DNA content. Thus, mechanisms that cause the amount of satellite DNA to be altered have interesting consequences in as far as the recombination system is concerned. It is possible that alterations in the amount of heterochromatin in any chromosome of the genome may affect recombination in every other chromosome.

So far we have focused our attention to the effect of the alteration in amount of centromeric heterochromatin. Although centromeric localization of heterochromatin is most common, telomeric and interstitial heterochromtin and also supernumerary heterochromatic chromosomes, are known to exist. The interchromosomal effect on recombination in the situation analogous to the telomeric localization of heterochromatin has been studied by SUZUKI (1963). He demonstrated that when the heterochromatic short arm of the *Y* chromosome was appended to the distal euchromatic tip of the *X* (Y^sX), there was a very strong interchromosomal effect, especially around the centromeric regions of chromosome *3*. On the other hand, if the same short arm of the *Y* chromosome was appended to the *X* chromosome as a right arm ($X{\cdot}Y^s$), it no longer had an interchromosomal effect on chromosome *3*. These experiments imply that the position of heterochromatin on a chromosome can influence its capacity to produce interchromosomal effects. However, there is an additional complication, which depends on the mode of origin of these two chromosomes, Y^sX and $X{\cdot}Y^s$. It is likely that both chromosomes have different amounts and different arrangements of *X* and *Y* heterochromatin in their basal regions, and the results may be complicated by differences in both the amount and type of heterochromatin in the two chromosomes.

As a heterochromatic duplication, the totally heterochromatic *Y* chromosome has also been used in studying interchromosomal effects. SCHULTZ and REDFIELD (1951) found that an extra *Y* chromosome slightly increased recombination in the autosomes, although they measured its effects only in the centromeric regions. More extensive studies of this kind, utilizing heterochromatic duplications of different sizes, have also shown increases in the recombination on chromosome *2*

in females carrying a free duplication; the magnitude of recombination alteration is again proportionally correlated with the size of the heterochromatic duplications (MIKLOS and YAMAMOTO in preparation).

These results suggest two mechanisms by which the recombination frequency of a genome is influenced: (1) Every different localization of heterochromatin on a chromosome in its own fashion, affects the recombination system. (2) In a given situation, the amount of heterochromatin in a particular location is an important factor in the regulation of the recombination system.

In the *Drosophila melanogaster* female, another way in which alterations in the amount of heterochromatin modify the recombination system is an intrachromosomal control: the specific chromosome has decreased crossing over when it possesses some deficiency in its own centromeric heterochromatin (YAMAMOTO and MIKLOS 1978). Thus, changing the amount of the centromeric heterochromatin *per se* could be an important regulatory mechanism that operates in addition to the genic control of the recombination system. In grasshoppers, the association of heterochromatin with the regulation of chiasma position and frequency is well established (JOHN 1973; KLASTERSKA, NATARAJAN and RAMEL 1974), and in one case at least this heterochromatin has been shown to contain satellite DNA (MIKLOS and NANKIVELL 1976).

The mechanisms by which the amount of heterochromatin can be altered within a genome are not yet completely understood. A plausible mechanism would be unequal sister-chromatid exchange, since the spontaneous occurrence of sister-chromatid exchanges have been demonstrated cytologically in a ring *Y* chromosome that is entirely heterochromatic and rich in satellite DNA (YAMAMOTO and MIKLOS 1978). If some of these spontaneous sister-chromatid exchanges are unequal, they would constitute a mechanism by which continuous variation in the heterochromatin content could occur, and the recombination system would be continually perturbed, both intrachromosomally and interchromosomally, giving rise to continually changing patterns of gene combinations.

As yet, mammals have not been examined as to whether intra- and interchromosomal effects can be mediated *via* changes in satellite DNA content. However, the extensive variation in heterochromatin content in many mammals and in other organisms (summarized in YAMAMOTO 1979) makes such investigations very interesting.

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LITERATURE CITED

BEADLE, G. W., 1932 A possible influence of the spindle fiber on crossing-over in *Drosophila*. Proc. Natl. Acad. Sci. U.S. **18**: 160–165.

FRY, K. and W. SALSER, 1977 Nucleotide sequences of HS-α Satellite DNA from kangaroo rat *Dipodomys ordii* and characterization of similar sequences in other rodents. Cell **12**: 1069–1084.

HATCH, F. T., A. J. BODNER, J. A. MAZRIMAS and D. H. MOORE, 1976 Satellite DNA and cytogenetic evolution. DNA quantity, satellite DNA and karyotypic variations in kangaroo rats (Genus *Dipodomys*). Chromosoma **58**: 155–168.

HSU, T. C., 1975 A possible function of constitutive heterochromatin: The bodyguard hypothesis. Genetics **79**: 137–150.

JOHN, B., 1973 The cytogenetic systems of grasshoppers and locusts. Chromosoma **44**: 123–146.

JONES, K. W., 1976 Repetitive DNA sequences in animals, particularly primates. Chromosome Today **5**: 305–313.

KLASTERSKA, I., A. T. NATARAJAN and C. RAMEL, 1974 Heterochromatin distribution and chiasma localization in the grasshopper *Bryodema tuberculata* (Fabr.) (Acrididae). Chromosoma **44**: 393–404.

LINDSLEY, D. L., C. W. EDINGTON and E. S. VON HALLE, 1960 Sex-linked recessive lethals in Drosophila whose expression is suppressed by the *Y* chromosome. Genetics **45**: 1649–1670.

LINDSLEY, D. L. and E. H. GRELL, 1968 *Genetic variations of* Drosophila melanogaster. Carnegie Inst. Wash. Publ. No. 627.

LUCCHESI, J. C. and D. T. SUZUKI, 1968 The interchromosomal control of recombination. Ann. Rev. Genet. **2**: 53–86.

MIKLOS, G. L. G. and R. N. NANKIVELL, 1976 Telomeric satellite DNA functions in regulating recombination. Chromosome **56**: 143–167.

PEACOCK, W. J., R. APPELS, P. DUNSMUIR, A. R. LOHE and W. L. GERLACH, 1977 Highly repeated DNA sequences: Chromosomal localization and evolutionary conservatism. pp. 494–506. In: *International Cell Biology*. Edited by B. R. BRINKLEY and K. R. PORTER. Rockefeller Univ. Press, New York.

RAMEL, C., 1962 Interchromosomal effects on crossing over in *Drosophila melanogaster*. I. Crossing-over. Hereditas **48**: 1–58.

REDFIELD, H., 1957 Egg mortality and interchromosomal effects on recombination. Genetics **42**: 712–728.

SCHULTZ, J. and H. REDFIELD, 1951 Interchromosomal effect on crossing over in *Drosophila*. Cold Spring Harb. Symp. Quant. Biol. **16**: 175–197.

SINGH, L., I. F. PURDOM and K. W. JONES, 1976 Satellite DNA and evolution of sex chromosomes. Chromosoma **9**: 43–62.

STEINBERG, A. G., 1936 The effect of autosomal inversions on crossing over in the *X*-chromosome of *Drosophila melanogaster*. Genetics **21**: 615–624.

STEINBERG, A. G. and F. G. FRASER, 1944 Studies on the effect of *X* chromosome inversions on crossing over in the third chromosome of *Drosophila melanogaster*. Genetics **29**: 83–103.

SUZUKI, D. T., 1962 Interchromosomal effects on crossing-over in *Drosophila melanogaster*. I. Effects of compound and ring *X* chromosomes on the third chromosome. Genetics **47**: 305–319. ——, 1963 Interchromosomal effects on crossing-over in *Drosophila melanogaster*. II. A reexamination of *X* chromosome inversion effects. Genetics **48**: 1605–1617. ——, 1973 Genetic analysis of crossing over and its relation to chromosome structure and function in *Drosophila melanogaster*. pp. 7–32. In: *Genetic Lectures*, Vol. 3. Edited by R. BOGARD, Oregon State Univ. Press, Corvalis.

WALKER, P. M. B., 1971a Origin of satellite DNA. Nature **229**: 306–308. ——, 1971b "Repetitive" DNA in higher organisms. Progr. Biophys. Molec. Biol. **23**: 145–190.

WILLIAMSON, J. H., 1966 Interchromosomal effects of autosomal translocations on recombination in *Drosophila melanogaster*. Genetics **54**: 1431–1440.

YAMAMOTO, M., 1979 Cytological studies of heterochromatin function in the *Drosophila melanogaster* male: Autosomal meiotic pairing. Chromosoma **72**: 293–328.

YAMAMOTO, M., and G. L. G. MIKLOS, 1977 Genetic dissection of heterochromatin in *Drosophila*: The role of basal X heterochromatin in meiotic sex chromosome behaviour. Chromosoma **60**: 283–296. ——, 1978 Genetic studies on heterochromatin in *Drosophila melanogaster* and their implications for the functions of satellite DNA. Chromosoma **66**: 71–98.

YUNIS, J. J. and W. G. YASMINEH, 1971 Heterochromatin, satellite DNA and cell function. Science **174**: 1200–1209.

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