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Mutant alleles of the meiotic locus, *mei-9*, differ in degree of effects on rod chromosome magnification and ring chromosome transmission in *Drosophila*

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Summary

Two mutant alleles of the meiotic locus, *mei-9*, have been examined for their effect on magnification of a rod X^{bb} chromosome and transmission of a ring X^{bb} chromosome under magnifying conditions. Our results indicate that the effects of these two mutations are allele-specific: *mei-9*^{a} strongly inhibits both rod chromosome magnification and ring chromosome loss under magnifying conditions, while *mei-9*^{b} has a smaller inhibitory effect on rod chromosome magnification and on the transmission of ring chromosomes under magnifying conditions. These observations can be explained by a difference in leakiness between the two alleles. Our results demonstrate that mutants defective in excision repair and repair replication inhibit ribosomal gene magnification. This suggests that a component of the excision repair pathway is involved in the process of magnification.

1. Introduction

A number of mutations that affect meiotic recombination or DNA repair in *Drosophila* have been isolated in screens for abnormal chromosome segregation, mutagen-sensitivity, or sensitivity to γ-rays (Baker & Carpenter, 1972; Smith, 1973; Boyd *et al.* 1976*a*; Smith 1976; Nguyen, Green & Boyd, 1978). Some of these recombination- or repair-defective mutations have been tested for their effect on magnification, a system of heritable increase in 18S + 28S rRNA gene number that occurs in germline cells of rDNA-deficient or *bobbed* (*bb*) *Drosophila* (Ritossa, 1968). The primary mechanism of magnification is most likely unequal sister chromatid exchange (Tartof, 1974; Endow, Komma & Atwood, 1984) with infrequent *X-Y* homologous recombination contributing to the recovery of *bobbed magnified* (bb^m) (Endow & Komma, 1986).

Hawley & Tartof (1983) and Hawley *et al.* (1985) examined the effect on magnification of recombination- or repair-defective mutations at seven loci and observed that mutants at three loci (*mei-41*, *mus-101*, *mus-108*) inhibit magnification. Since *mei-41*, *mus-101* and *mus-108* are defective in post-replication repair (Boyd & Setlow, 1976; Boyd & Shaw, 1982), Hawley *et al.* (1985) concluded that rDNA magnification requires a function that is involved in postreplication repair. Hawley & Tartof (1983) and Hawley *et al.* (1985) further observed that *mei-9*^{b}, a repair replication-defective mutation (Nguyen & Boyd, 1977), had little or no effect on magnification. This observation was in apparent conflict with a previous report that *mei-9*^{a} inhibits magnification (Polito *et al.* 1982).

We have re-examined the two previously studied alleles of *mei-9* for their effect on rod X^{bb} chromosome magnification and, further, tested their effect on ring X^{bb} chromosome transmission under magnifying conditions. Our results show that there is a difference between the two *mei-9* alleles with respect both to rod chromosome magnification and ring chromsome transmission under magnifying conditions: *mei-9*^{a} strongly inhibits both rod X^{bb} chromosome magnification and ring X^{bb} chromosome transmission under magnifying conditions, while *mei-9*^{b} has a smaller inhibitory effect on rod chromosome magnification and on the transmission of ring chromosomes under magnifying conditions. Our results confirm the report by Polito *et al.* (1982) that *mei-9*^{a} inhibits magnification and demonstrate that *mei-9*^{b} has a small, but detectable, inhibitory effect on magnification. The observation that *mei-9*^{a}, which has been characterized

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as normal in post-replication repair but defective in excision repair (Boyd, Golino & Setlow, 1976*b*) and repair replication (Nguyen & Boyd, 1977), and *mei-9*^{b}, which is defective in repair replication (Nguyen & Boyd, 1977), inhibit magnifying events suggests that a component of the excision repair pathway is involved in rDNA magnification.

2. Materials and methods

(i) Drosophila *stocks*

Most of the mutations used in these studies are described in Lindsley & Grell (1968) or Lindsley & Zimm (1985).

The ring X^{bb} chromosome, *R(1)2 y ct bb*, has been described previously (Endow, *et al.* 1984). The ring chromosome has a strong *bb* allele, but is viable and fertile with the Y^{bb-}, chromosome, which was used for magnifying conditions. Derivatives of *R(1)2 y ct bb* were constructed for the experiments described here.

A stock of *mei-9*^{a} marked with *echinus* (*ec*) and *ruby* (*rb*) was obtained from J. M. Mason. *mei-9* is at map position 6·5, *ec* is at 5·5 and *rb* is at 7·5. Recombinant ring X^{bb} chromosomes carrying *mei-9*^{a} were obtained by screening for *y rb* males among offspring of *R(1)2 y ct bb/ec mei-9*^{a} *rb* females. The presence of *mei-9*^{a} in the recombinant chromosomes was confirmed by testing the offspring of the *y rb* males for sensitivity to methyl methanesulfonate (MMS) according to the method of Boyd *et al.* (1976*a*).

mei-9^{a} was transferred by recombination from the ring X^{bb} chromosome onto the bb^{2} rod chromosome, which was obtained from R. S. Hawley. y^{+} *rb* recombinant chromosome were recovered among offspring of *R(1)2 y mei-9*^{a} *rb* bb/bb^{2} females, and were tested for MMS sensitivity to confirm the presence of *mei-9*^{a}.

A stock carrying *mei-9*^{b} on a chromosome marked with *yellow* (*y*) (0·0), *crossveinless* (*cv*) (13·7) and bb^{2} (66·0) was obtained from R. S. Hawley. *y* was eliminated by recombination with a *w m f* chromosome by screening for y^{+} *cv* males among offspring of *y mei-9*^{b} *cv* $bb^{2}/w\ m\ f$ females. Since *mei-9* (6·5) is between *w* (1·5) and *cv* (13·7), it was probable that these recombinants would carry *mei-9*^{b}. *mei-9*^{b} was then transferred by recombination onto the ring X^{bb} chromosome by screening for *y cv* males among offspring of *R(1)2 y ct bb/mei-9*^{b} *cv* females. The offspring of two males carrying *y cv* recombinant chromosomes were tested for MMS sensitivity to confirm the presence of *mei-9*^{b}, and both chromosomes were found to be sensitive. The original *y mei-9*^{b} *cv* bb^{2} chromosome was used in tests of rod X^{bb} chromosome magnification.

Ring and rod chromosomes were tested for *bb* with the Y^{bb-} or $B^{S}Y^{bb-}$ chromosome, or with $In(1)sc^{4L}sc^{8R}$ (see below). Only chromosomes with strong *bb* alleles were used in these studies. Allelism of *mei-9*^{a} and *mei-9*^{b} was confirmed by their failure to complement for MMS sensitivity in females.

The structure of rod and ring chromosomes carrying *mei-9* was confirmed by cytological examination of larval neuroblast tissue from individuals of the stocks at several times during the course of these studies.

(ii) *Rod chromosome magnification tests*

bb^{2} males carrying either the meiotic mutation or its wild-type allele, and either Y^{bb-} or a bb^{+} *Y* were mated to $sc^{4L}sc^{8R}/y^{2}w^{bf}$ or $sc^{4L}sc^{8R}/sc^{4L}sc^{8R}/B^{S}Y$ females carrying the $In(1)sc^{4L}sc^{8R}$, $y\ sc^{4}\ sc^{8}\ cv$ chromosome (referred to as $sc^{4L}sc^{8R}$). The $sc^{4L}sc^{8R}$ chromosome is deficient for the nucleolus organizer region and is used to test for reversion of *bb* to bb^{m} or bb^{m+}. Female offspring carrying the bb^{2} and $sc^{4L}\ sc^{8R}$ chromosomes were examined for magnification of the bb^{2} chromosome. These females were classified as *bb*, bb^{m} or bb^{m+} by measurement of posterior scutellar bristles (Komma & Endow, 1986) and by noting the presence or absence of abdominal etching. bb^{m} (*bobbed magnifed*) refers to any improvement in phenotype relative to the parental *bb* fly, while bb^{m+} indicates reversion to wild type. *bb*, bb^{m}, and bb^{m+} offspring typically had bristle lengths of 10–12, 16–17 and 18–20 units on an arbitrary scale, respectively, compared with $bb^{+}/sc^{4L}sc^{8R}$ females with bristle lengths of 19–20 units. Offspring of single-pair matings were examined in order to monitor the frequency of clusters of bb^{m} offspring from a single male.

(iii) *Ring chromosome transmission tests*

Transmission of ring X^{bb} chromosomes was monitored by mating *y car* females to males carrying the ring chromosome with the meiotic mutation or its wild-type allele, and either Y^{bb-} or a bb^{+} *Y*. Male (M) and female (F) offspring were counted, and ring loss relative to nonmagnifying conditions was calculated as equal to

$$\frac{(\text{F:M ratio with } Y^{bb+}) - (\text{F:M ratio with } Y^{bb-})}{\text{F:M ratio with } Y^{bb+}},$$

(Endow *et al.* 1984).

3. Results

(i) *Effect of* mei-9 *on rod chromosome magnification*

Two alleles of the repair-defective meiotic mutation, *mei-9*, were tested for their effect on magnification of a rod *X* chromosome carrying a strong *bb* allele. The *bb* phenotype is characterized by short, thin thoracic bristles, delayed development and, in severe cases, distorted pigmentation of the abdominal tergites. These characteristics presumably arise as a consequence of a decreased rate of protein synthesis, and can be partially or completely alleviated by an increase in ribosomal genes. The classical quantitative study of

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Stern (1929) first showed that bristle length is correlated with dosage of *bb*. Ritossa (1968), in his initial report of magnification, as well as in subsequent studies (Ritossa & Scala, 1969; Henderson & Ritossa, 1970; Ritossa *et al.* 1971; Boncinelli *et al.* 1972), showed using filter saturation hybridization that magnified revertants of *bb* were increased in rDNA content relative to the starting chromosome. Because of the strong correlation between improvement in phenotype and increased ribosomal gene content, phenotypic traits, in particular, bristle length, have become frequently used criteria of magnification (see, for example, Atwood, 1969; Tartof, 1974; Locker, 1976). Measurement of posterior scutellar bristle length provides a reliable index of magnification (Komma & Endow, 1986).

In the experiments reported here, rDNA-deficient (magnifying) males carrying the Y^{bb-} chromosome together with the meiotic mutation on a bb^2 *X* chromosome were mated to females carrying the $sc^{4L}sc^{8R}$ chromosome, which is completely deficient for rDNA. $bb^2/sc^{4L}sc^{8R}$ offspring of these matings were scored for their phenotype with respect to *bb* by posterior scutellar bristle length and by noting the presence or absence of abdominal etching. Results of these experiments are presented in Table 1. As controls for these experiments, the bb^2 chromosome was tested for the production of *bobbed magnified* in rDNA-nondeficient (nonmagnifying) and rDNA-deficient (magnifying) flies carrying *mei-9*$^+$. $y\ bb^2/B^SY$ males that are nondeficient for rDNA produced no bb^m or bb^{m+} among 162 *bb* offspring, indicating that the frequency of spontaneous reversion of *bb* to bb^m or bb^{m+} is less than 0·006 (Table 1). By comparison, the bb^2 chromosome in combination with Y^{bb-} in rDNA-deficient males resulted in frequencies of $bb^m + bb^{m+}$ of 0·391 and 0·490 (Table 1). The frequencies of bb^{m+} in these experiments were 0·226 and 0·246. These values are in agreement with the frequencies of 0·19 and 0·173 reported previously for reversion of the bb^2 chromosome to bb^{m+} (Hawley & Tartof, 1983; Hawley *et al.* 1985). The distribution of bb^m and bb^{m+} produced by individual males is skewed toward the production of $\geqslant 2$ revertants per male with 80–85% of individuals producing two or more magnified offspring. The recovery of multiple revertants from individual males has been attributed to the occurrence of magnification in premeiotic germline cells (Tartof, 1974).

rDNA-deficient males carrying *mei-9*^{a} on the bb^2 chromosome produced a frequency of $bb^m + bb^{m+}$ of 0·049 in two separate experiments (Table 1). This frequency is 8- to 10-fold lower than for the bb^2 chromosome without *mei-9*^{a}. The frequencies of bb^{m+} in these experiments are 0·012 and $< 0{\cdot}016$ (Table 1), representing a 20-fold or greater reduction in frequency compared to the bb^2 chromosome without *mei-9*^{a}. Clusters of bb^m or bb^{m+} produced by individual males are greatly reduced in frequency, but not eliminated, in the presence of *mei-9*^{a}: In one experiment, no clusters of bb^m were recovered from 10 males, while in a second experiment, 20% of individual males ($n = 19$) produced $\geqslant 2$ revertants. The difference between the two experiments is probably due to the smaller number of fertile individuals in the first experiment compared with the second ($P > 0{\cdot}1$).

rDNA-deficient males carrying *mei-9*^{b} produce $bb^m + bb^{m+}$ revertants of bb^2 at frequencies of 0·194 and 0·305, with frequencies of bb^{m+} of 0·069 and 0·177 (Table 1). These values for reversion of bb^2 to bb^{m+} in *mei-9*^{b} males are similar to the frequencies of 0·18 and 0·14 reported by Hawley & Tartof (1983) and Hawley *et al.* (1985), although the value of 0·069 is somewhat lower than those reported previously. Values for reversion to $bb^m + bb^{m+}$ represent a 1·5- to 2·5-fold decrease in *bb* reversion frequency compared with the bb^2 chromosome without *mei-9*^{b}. The frequency of bb^{m+} in the presence of *mei-9*^{b} is reduced by 1·5- to 3·5-fold. The distribution of *bb* revertants produced by single males is also altered relative to mei-9$^+$ flies, with

Table 1. *Effect of* mei-9 *on rod chromosome magnification*

Paternal genotype	$X/sc^{4L}sc^{8R}$ progeny			No. of males producing 0, 1 or $\geqslant 2$ $bb^m + bb^{m+}$			Frequency of $bb^m + bb^{m+}$ revertants	Frequency of bb^{m+} revertants
	bb	bb^m	bb^{m+}	0	1	$\geqslant 2$		
$y\ bb^2/B^SY$	162	0	0	0	0	0	$< 0{\cdot}006$	$< 0{\cdot}006$
bb^2/B^SY^{bb-}	148	40	55	2	0	11	0·391	0·226
$y\ bb^2/Y^{bb-}$	199	95	96	1	3	14	0·490	0·246
rb mei-9^a bb^2/B^SY^{bb-}	327	13	4	11	4	4	0·049	0·012
rb mei-9^a bb^2/Y^{bb-}	58	3	0	7	3	0	0·049	$< 0{\cdot}016$
y cv mei-9^b bb^2/B^SY^{bb-}	362	56	31	10	4	9	0·194	0·069
y cv mei-9^b bb^2/Y^{bb-}	114	21	29	3	5	7	0·305	0·177

Males of the indicated genotypes were mated in single pairs to females carrying the $sc^{4L}sc^{8R}$ chromosome, and the $X/sc^{4L}sc^{8R}$ offspring were scored for their phenotype with respect to *bb* based on measurement of posterior scutellar bristles and the presence or absence of abdominal etching. The distribution of the bb^m and bb^{m+} offspring among individual males is indicated together with the overall frequency of $bb^m + bb^{m+}$ and the frequency of only bb^{m+}.

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an increased number of males producing no bb^m or bb^{m+}. However, although the proportion of males producing magnified revertants is reduced, multiple bb^m are produced by 40–45% of individuals.

In brief, these data show that *mei-9*^{a} strongly inhibits rod chromosome magnification, reducing the frequency of magnified revertants by roughly 8- to 10-fold, and reducing by roughly 75% the number of individual males producing multiple revertants. In contrast, *mei-9*^{b} reduces the frequency of $bb^m + bb^{m+}$ by only 1·5- to 2·5-fold, and reduces by approximately 50% the number of individual males producing clusters of revertants.

(ii) *Effect of* mei-9 *on ring* X^{bb} *chromosome transmission*

The effect of two alleles of *mei-9* on ring X^{bb} chromosome transmission was monitored by comparing female: male (F:M) ratios among offspring produced by rDNA-deficient and non-deficient males. In these experiments transmission of the ring chromosome is monitored under magnifying and non-magnifying conditions. Loss of ring X chromosomes in male meiosis is expected to result in a decrease in ring X/X female offspring and an increase in X/O male offspring upon mating ring X/Y males to X/X females. In previous experiments (Endow *et al.* 1984) we used males carrying a ring X chromosome together with a dominantly marked Y in order to distinguish X/Y from X/O males. These experiments demonstrated that the number of ring X/X female + X/O male offspring recovered was not equivalent to the number of X/Y male offspring, as expected if all of the ring-X and nullo-X,Y gametes had been recovered. We attributed the ring loss that is not recovered as X/O males to zygote lethality or sperm dysfunction due to abnormal ring X chromosomes (Endow *et al.*, 1984). In these previous experiments, X/O males comprised 0·01–0·05 of the total offspring. Because the relative number of X/O males is small, the F:M ratio, which is equal to

$$\frac{\text{ring } X/X \text{ females}}{X/Y + X/O \text{ males}},$$

approximates

$$\frac{\text{ring } X/X \text{ females}}{X/Y \text{ males}} = \frac{\text{ring } X}{Y}.$$

The F:M ratio can thus be used as an approximation of the relative number of ring X gametes recovered among the offspring, with
1 − (F:M ratio) = frequency of ring loss.
The increase in ring loss under magnifying conditions is equal to

$$\left[\begin{array}{c}\text{Frequency of ring loss under}\\ \text{magnifying conditions}\end{array}\right] - \left[\begin{array}{c}\text{Frequency of ring loss under}\\ \text{nonmagnifying conditions}\end{array}\right]$$
$$= (\text{F:M ratio with } Y^{bb+}) - (\text{F:M ratio with } Y^{bb-}).$$

Normalizing the increase in ring loss under magnifying conditions to the amount of ring loss under non-magnifying conditions allows comparison with ring loss under nonmagnifying conditions, and results in the expression

$$\frac{(\text{F:M ratio with } Y^{bb+}) - (\text{F:M ratio with } Y^{bb-})}{\text{F:M ratio with } Y^{bb+}}$$
$$= 1 - \left(\frac{\text{F:M ratio with } Y^{bb-}}{\text{F:M ratio with } Y^{bb+}}\right)$$

(Endow *et al.* 1984). We previously denoted this value as the amount of ring X loss attributed to Y^{bb-}; more accurately, this denotes the relative increase in ring X loss due to magnifying conditions. In the experiments described here, we use the F:M sex ratio in the presence or absence of Y^{bb-} to calculate the relative

Table 2. *Effect of* Y^{bb-} *on sex ratio in ring-bearing males with* mei-9

Male parent	Offspring ♀	Offspring ♂	♀:♂ ratio	Relative loss of X attributed to Y^{bb-}
R(1)2 y c: bb/Y	1596	2535	0·63	
R(1)2 y ct bb/Y^{bb-}	539	1242	0·43	0·32
R(1)2 y mei-9^a *rb bb/Y*	1019	1815	0·56	
R(1)2 y mei-9^a *rb bb/Y*$^{bb-}$	1478	2654	0·56	0·0
R(1)2 y cv mei-9^b *bb/Y*	1606	2629	0·61	
R(1)2 y cv mei-9^b *bb/Y*$^{bb-}$	1352	2833	0·48	0·21

Males of the indicated genotypes were mated to *y car* females, and female and male offspring of each mating were scored. Relative loss of the ring X chromosome as a consequence of magnifying conditions is calculated as $1-[(F':M')/(F:M)]$, where F′:M′ and F:M are the female:male ratios of the offspring in the presence and absence of the Y^{bb-} chromosome, respectively.

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increase in ring loss under magnifying conditions compared with nonmagnifying conditions.

Table 2 shows data for experiments in which transmission of the *R(1)2 bb* chromosome was monitored by examining offspring of males carrying Y^{bb-} or a $bb^+ Y$, and *mei-9*^{a} or *mei-9*^{b}. The control for these experiments is the *R(1)2 bb* chromosome with *mei-9*$^+$. Males carrying the *R(1)2 y ct bb* chromosome and a bb^+ *Y* chromosome produce a F:M ratio of 0·63. In the presence of Y^{bb-}, the F:M ratio produced by males carrying *R(1)2 y ct bb* is 0·43, indicating that the Y^{bb-} chromosome results in increased loss of the ring chromosome. The relative increase in ring loss due to magnifying conditions is calculated as

$$1-\left(\frac{\text{F:M ratio with } Y^{bb-}}{\text{F:M ratio with } bb^+ Y}\right),$$

and is 0·32 for the *R(1)2 y ct bb* chromosome with *mei-9*$^+$.

The F:M ratio among offspring produced by males carrying a ring *X* with *mei-9*^{a} and Y^{bb-} is the same as for males carrying the ring and Y^{bb+} (Table 2). This means that no added loss of the ring X^{bb} chromosome occurs in rDNA-deficient (magnifying) males carrying *mei-9*^{a} compared with rDNA-nondeficient (nonmagnifying) males with *mei-9*^{a}. *mei-9*^{a} thus prevents or inhibits the loss of the ring X^{bb} chromosome that occurs under magnifying conditions.

Males carrying a ring chromosome with *mei-9*^{b} and Y^{bb-} produce lower F:M ratios among their offspring compared with males carrying the *mei-9*^{b} ring and Y^{bb+}, indicating that ring chromosome loss is increased under magnifying conditions (Table 2). The relative increase in ring loss under magnifying conditions is calculated as 0·21. Thus the loss of ring X^{bb} chromosomes that occurs in males with Y^{bb-} also occurs in males with *mei-9*^{b} and Y^{bb-}, although the relative amount of loss for the *mei-9*^{b} ring is somewhat lower than for the ring with *mei-9*$^+$. We conclude that *mei-9*^{b} does not prevent the loss of ring X^{bb} chromosomes that is observed under magnifying conditions; its inhibitory effect on ring *X* loss under magnifying conditions is much smaller than that of *mei-9*^{a}.

These results demonstrate that one allele of *mei-9*, *mei-9*^{a}, strongly inhibits the loss of ring X^{bb} chromosomes that occurs under magnifying conditions, while a second allele, *mei-9*^{b}, has a smaller inhibitory effect on ring chromosome transmission under magnifying conditions.

4. Discussion

Our results demonstrate that two alleles of the meiotic mutation, *mei-9*, differ in the degree of their effects on magnification. One allele, *mei-9*^{a}, reduces rod X^{bb} magnification by 8- to 10-fold and strongly inhibits the loss of ring X^{bb} chromosomes under magnifying conditions. A second allele, *mei-9*^{b}, has a smaller inhibitory effect both on magnification of rod X^{bb} chromosomes, reducing it by 1·5- to 2·5-fold, and on transmission of ring chromosomes under magnifying conditions.

In these studies we used two methods to measure magnification. The first, magnification of a rod X^{bb} chromosome, reflects both premeiotic and meiotic events, with premeiotic events evidenced by the recovery of clusters of bb^m. The second method, ring X^{bb} chromosome transmission, is a measure of meiotic magnification, assuming that premeiotic loss of ring *X* chromosomes is cell-lethal. Increased ring *X* chromosome loss in rDNA-deficient males is most likely a consequence of induced sister chromatid exchange in the ring (Endow *et al.* 1984); a deficiency of ring *X*/*X* females is produced among the offspring, resulting in lower F:M ratios. Our results indicate that *mei-9*^{a} has a strong inhibitory effect on rod chromosome magnification, including the recovery of clusters of bb^m, and on ring chromosome loss in magnifying flies, as expected if the *mei-9*$^+$ product were required for magnification in both premeiotic and meiotic cells. *mei-9*^{b} also inhibits both rod chromosome magnification and ring *X* chromosome loss under magnifying conditions, but its effect is not as strong as that of *mei-9*^{a}.

The observation that one allele of *mei-9* has a marked effect on magnification, while a second allele has a much smaller effect, explains the conflicting conclusions regarding the role of *mei-9* in magnification reached by investigators who examined only one mutant allele (Polito *et al.* 1982; Hawley & Tartof, 1983; Hawley *et al.* 1985). Polito *et al.* (1982) observed that approximately 50% of *mei-9*^{a} males were *bb* after eight magnifying generations, when the process of magnification is normally complete. This indicated a strong inhibitory effect of the meiotic mutation on magnification. Hawley & Tartof (1983) and Hawley *et al.* (1985), on the other hand, observed frequencies of magnification for *mei-9*^{b} *bb* males that approximated frequencies for the same *bb* allele on a mei-9$^+$ chromosome. These workers concluded that *mei-9* had no effect on magnification, even though in one report a somewhat lower frequency of magnification was observed in the presence of *mei-9*^{b} than in its absence (Hawley *et al.* 1985). Using *bb* and *mei-9*^{b} alleles obtained from R. S. Hawley, we observe a more pronounced inhibitory effect of *mei-9*^{b} on magnification than reported by Hawley & Tartof (1983) and Hawley *et al.* (1985). We also observe a reduction in the frequency of clusters of bb^m in the presence of *mei-9*^{b}, strengthening our conclusion that *mei-9*^{b} inhibits rod chromosome magnification. Our conclusion that *mei-9*^{b} inhibits magnification is further supported by the observation that *mei-9*^{b} inhibits the loss of ring *X* chromosomes under magnifying conditions relative to *mei-9*$^+$ flies.

mei-9^a, which greatly inhibits magnification, has a strong inhibitory effect on recombination in females (Baker & Carpenter, 1972). *mei-9*^{b}, an independent

allele, affects recombination in females, but at a frequency of approximately 2-fold less. The magnitude of the difference between *mei-9*^{a} and *mei-9*^{b} is roughly the same for both homologous recombination and magnification, the basis of which is probably sister chromatid exchange. It therefore seems reasonable to conclude that the difference between these two loss-of-function alleles is simply a difference in leakiness. The alternative possibility exists, however, that the *mei-9*$^+$ product is multifunctional and that different alleles of *mei-9* affect different functions. For example, the *mei-9*$^+$ product has been implicated in excision repair of dimers and possibly also mismatches; its role in recombination may be to break cross-strands in recombination intermediates (Boyd *et al.* 1976*b*). Alleles that affect one or the other of these functions might have different effects on processes, such as magnification, that involve recombinational mechanisms.

These experiments demonstrate that *mei-9*$^+$ is active in male *D. melanogaster* despite the extremely low levels of homologous recombination observed in males, even under magnifying conditions (Ritossa, 1973; Endow & Komma, 1986). This must also be true of *mei-41*$^+$, a second locus implicated both in homologous recombination in females (Baker & Carpenter, 1972) and in magnification in males (Hawley & Tartof, 1983; Hawley *et al.* 1985). The lack of homologous recombination in male *D. melanogaster* must not be due to the absence of repair activities that are needed for exchange; it may instead reflect the absence of pairing structures or products that are needed for recombination to take place.

In summary, our results confirm the observation by Polito *et al.* (1982) that *mei-9*^{a} inhibits magnification and demonstrate that *mei-9*^{b} has a similar, although less severe inhibitory effect. They show that two reduction-of-function alleles at a given locus may not have the same effect on a given process, in this case, magnification. They also indicate that the *mei-9*$^+$ product is needed both premeiotically and meiotically for magnification. *mei-9*^{a} has been characterized as an excision repair- and repair replication-defective mutation (Boyd *et al.* 1976*b*; Nguyen & Boyd, 1977), while *mei-9*^{b} has been shown to be repair replication-defective. Since both *mei-9*^{a} and *mei-9*^{b} inhibit magnification, magnification must not be limited to components of post-replication repair systems, but must also involve a component of the excision repair system.

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