Crossing Over in Males of Higher Diptera (Brachycera)

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Achiasmatic meiosis, or meiosis without crossing over, is characteristic of all higher Diptera males (suborder Brachycera). However, several cases of spontaneous crossing over in males have been reported in several different species. An examination of the published data suggests that recombinant chromosomes recovered from heterozygous males are usually the result of spontaneous crossing over in premeiotic cells. Mitotic, or somatic, crossing over probably occurs at a low frequency in all Diptera. When the crossover occurs in a gonial cell line, the recombinant chromosomes can be recovered in the gametes as presumptive meiotic crossovers. In cases where there is a translocation between the Y chromosome and an autosome, the segregation of the chromosomes from the translocation complex can produce aneuploids that phenotypically appear to be crossovers. Chromosome rearrangements and insertion elements, including the male sex-determining factor in *Musca domestica* and *Megaselia scalaris,* increase the frequency of exchange. *Drosophila ananassae* appears to be an exception to the above. Genetic evidence in *D. ananassae* suggests that crossing over is a meiotic event and is controlled by a series of suppressors and enhancers.

Crossing over serves a dual function in most meiotic systems. First, it generates the genetic variability necessary for the survival of a species. Second, after the exchange event, the resulting chiasmata maintain the synapsed state between homologues during the latter stages of prophase and into metaphase. When crossing over is eliminated or greatly reduced, segregation at the first meiotic division becomes highly irregular, resulting in a high frequency of aneuploid gametes.[2,34] In female *Drosophila melanogaster,* complex models have been derived to explain the relationship between genetic exchange, nonhomologous pairing, and nondisjunction.[16,55]

In invertebrates, the heterogametic sex of a large number of species is achiasmatic[75] and lacks meiotic crossing over. However, chromosome segregation in the achiasmatic sex is normal, suggesting a separate meiotic system to ensure the generation of regular haploid gametes.

According to White,[75] achiasmatic meiosis is the general rule for males of all "higher" Diptera (suborder Brachycera). In recent years, a number of cases of spontaneous crossing over in males has been reported in several different species. Many of these studies were confounded either by a reduced penetrance of the mutants used in the crosses or by the involvement of rearranged chromosomes, usually translocations. Various offspring can appear to be the result of a meiotic crossover but are in fact the result of events other than meiotic exchange.

The following is an analysis of species in which crossing over in males has been reported, with an emphasis on the possible mechanism responsible for the exceptional offspring.

Review

Musca domestica

Several cases of crossing over in male houseflies have been reported, primarily, if not exclusively, in strains in which the male-determining factor from the Y chromosome has been transposed to an autosome and the males are now XX. Both Kerr[35] and Sullivan[70] demonstrated a low frequency of crossing over on chromosome 3, and Inoue and Hiroyoshi[30] found a low frequency of crossing over on chromosome 1. In Sullivan's experiments,[70] it is not clear whether the males were XY or XX. In one set of crosses, Sullivan[70] recovered six recombinants from three males out of a total of 63 pair matings. This clustering suggests a premeiotic origin of the recombinants.

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Lester et al[39,40] reported an extremely high frequency of recombination in XX males derived from a wild caught population. Crossover frequencies of 30.7 and 9.3% were reported between the male-determining factor (*M*) and two linked second-chromosome mutants. These frequencies are much higher than any others that have been reported in *Musca*. Additionally, the reciprocal recombinant classes were very unequal, 5 and 465 in one case and 1 and 362 in the other.

The results of these experiments suggest that the presumptive recombinants were actually aneuploid segregants from a translocation heterozygote. In any translocation heterozygote, a significant proportion of the progeny will be aneuploid as a result of the segregation pattern from the translocation complex. If the resulting aneuploids include only a small portion of the genome, they will survive, with the hyperploid (the aneuploid with a duplication) surviving at a higher rate than the hypoploid (the aneuploid with a deficiency). If the wild caught stock carried a translocation between the Y chromosome and chromosome 2, with the second-chromosome breakpoint located between the mutant loci and the centromere, terminal aneuploids with the phenotypes observed would be generated. The unequal recovery of the reciprocal classes is consistent with this interpretation, because the larger class would have been hyperploid, and the smaller class would have been hypoploid.

Rubini et al[64] reviewed the occurrence of male recombination, mosaics, and gynandromorphs. Based on genetic and cytological observations, it was proposed that these classes are due to mitotic, or somatic, recombination between homologous chromosomes in mitotically dividing gonial cells. Cytologically, exchanges between homologues in mitotic cells have been observed.[64] The somatic association of homologues characteristic of Diptera presumably facilitates this process of mitotic recombination.

Megaselia scalaris

Megaselia scalaris lacks a heteromorphic pair of sex chromosomes; both males and females contain three pairs of homologues.[45] Maleness is determined by a sex-determining factor[44] (*M*), which can be located in any one of the three chromosomes and is capable of being transposed. Transposition usually occurs at frequencies of less than 1%, and the element is always inserted into one end of each linkage group.[46] Transposition is probably premeiotic, as clusters of transpositions are recovered.[46] Cytologically, male meiosis has been described as being typical and chiasmatic.[47] No frequencies have been given.

Crossing over in males has been demonstrated for each chromosome pair. Generally, the frequency of crossing over between any two linked genes in males is much lower than is the case for the same region in females.[8]

The crossover products are recovered in clusters,[47] indicating that the exchange event is premeiotic. An apparent polar effect on crossover distribution was reported,[8] with exchanges at the end of the chromosome containing *M* being more frequent. The frequency of recombination also depends on which chromosome contains *M*.[47] Recombination is lower for a chromosome with *M* than it is when *M* is in a nonhomologous chromosome.

In an experiment designed to measure both transposition of *M* and crossing over in chromosome 1, no transpositions were recovered from nearly 26,000 flies, and the frequency of crossing over was 0.023%. In another experiment with the same markers, the frequency of transposition was 0.057% (13 of 23,007) and the crossover frequency was 0.15%.[45] The correlated behavior of both events suggests a common mechanism.

Ceratitis capitata

Ceratitis capitata, like *Megaselia*, has been reported to have a chiasmatic meiosis in males.[57] The genome ($n = 6$) averaged 12 chiasmata per cell. This suggests that meiotic crossing over should occur at a relatively high rate, but genetic tests have not borne this out.[9,60–62,67] In the absence of any known chromosomal rearrangements, crossing over in males is either rare[60–62] or absent.[9]

Considerable effort has been spent developing genetic systems for the biological control of *C. capitata*. These systems usually involve a translocation between an autosome and the Y chromosome. Crossing over between linked genes is increased when the males are heterozygous for a Y-autosome translocation.[60,62]

In population cages, the Y-autosome translocations are unstable and frequently break down. The translocation-bearing males were heterozygous for a recessive autosomal marker, and the females were homozygous for the mutant. Three cases have been reported in which the translocation system broke down.[62] The result was a dramatic increase in the overall frequency of the mutant phenotype. Mutant males became predominant, and wild type females appeared, but at a much lower frequency. When the fertility of wild type and mutant males was compared, it was found that the wild type males were semisterile and the mutant males were not. This observation suggests that the mutant males no longer carried the translocation.

It is interesting to speculate about the origin of these males. Crossing over in the male should result in the mutant allele being transferred to the translocated chromosome and the wild type allele being transferred to the nontranslocated autosome. In this situation, one would expect selection for the wild type phenotype. However, just the opposite was found: The mutant phenotype replaced the wild type phenotype.

In *Ceratitis*, the Y chromosome is responsible for sex determination, XXY individuals are phenotypically normal fertile males, and single X individuals are apparently lethal.[77] It is also likely that the X chromosome contains relatively few genes. This is based on fact that only one X-linked gene has been described,[66] and this gene was discovered only recently.

Thus, the breakdown of the translocation can be explained by a heterochromatic crossover between the X and the Y chromosomes. This is diagrammed in Figure 1. The crossover will generate an attached-XY chromosome and a reciprocal chromosome that lacks both the Y chromosome male-determining gene(s) and the X chromosome gene(s). After the exchange, the offspring would be XXY (free X/attached-XY) males and a genetically single X individual that is lethal. Thus, phenotypically mutant but translocation-free males would be generated.

Robinson and van Heemert[58] induced several Y-autosome translocations and followed their behavior in population cages for 10 generations. All the lines but one were stable. Exceptional males from the unstable line were sterile.

Saul[65] also induced several Y-autosome translocations. He recovered four stable lines, each of which yielded a low frequency of exceptional males. These exceptional males were delayed in development and were smaller than the normal; upon progeny testing, they were sterile. Saul[65] suggested that these males might be aneuploid segregants that were hyperploid for part of the autosome. The single unstable stock of Robertson and van Heemert[58] was possibly due to the same cause.

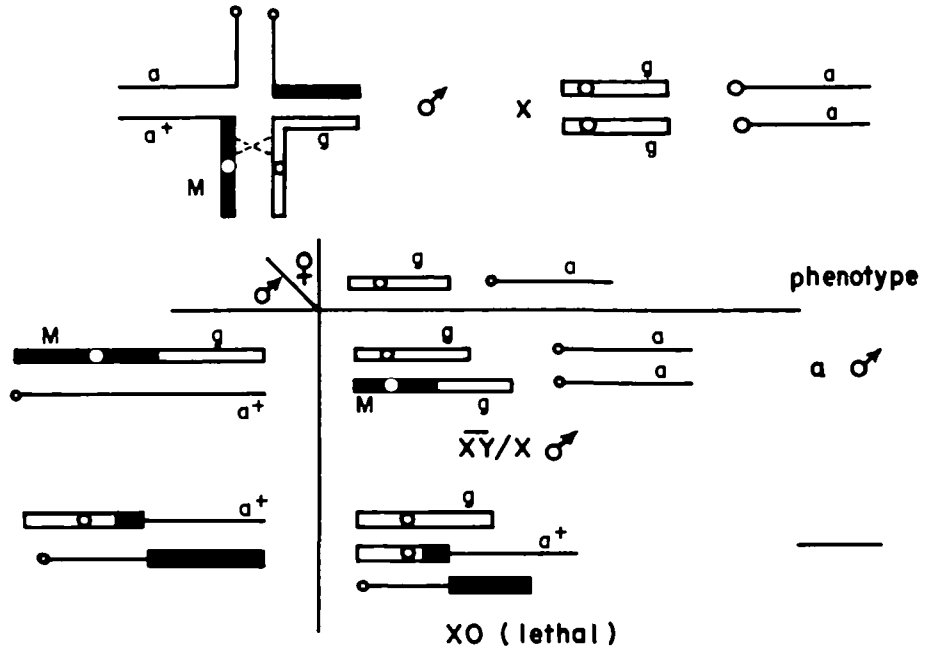

Figure 1. Proposed origin of mutant males from heterozygous *Ceratitis capitata* Y-autosome translocation males crossed to normal females. *M* represents the male-determining factor on the Y chromosome, *g* represents X-linked genes, and *a* represents an autosomal gene. See text for explanation. Open bar = X chromosome; solid bar = Y chromosome; line = autosome; open circle = centromere.

Lucilia cuprina

As in other Diptera, crossing over occurs in males at a low frequency, at least for chromosome 3.[12] Y-autosome translocations enhance the frequency of crossing over, although in all cases the overall frequency is quite low compared with that of females.

Not only does translocation heterozygosity in males enhance crossing over, but the translocations are prone to breaking down.[12] The males were heterozygous for a complex translocation involving two autosomes and the Y chromosome (Figure 2). Males were heterozygous for two recessive mutants, whereas females were homozygous. After a few generations in mass rearing cages, the stocks began to break down, with mutant males being the most frequent exceptional class.

When the exceptional males were examined cytologically and genetically, it was discovered that many of the males either had lost the translocation or had "reverted" to a *T(Y;5)* translocation. The breakpoint in chromosome 5 of the *T(Y;5)* appeared to be identical to that in *T(Y;5;3)*.

The *T(Y;5)* males were postulated to be the result of an unequal exchange or fusion between different pieces of the translocated Y chromosome. Rehealing at site 1 (Figure 2) between the Y chromosome pieces will generate the *T(Y;5)* and a free chromosome 3, possibly with a small insert of Y material. The free Y chromosome and the free chromosome 5 would be the result of a breakdown of the *T(Y;5)*, comparable to the initial breakdown of the *T(Y;5;3)* complex.

An alternative explanation is that crossing over between the X and the Y chromosomes is responsible for the exceptional chromosomal types. Both *XS* and *YS* are heterochromatic, whereas the long arms contain distal, nonheterochromatic regions.[4,73] The only known X chromosome gene has been shown to be located in the proximal region of the euchromatic region of the long arm.[43] The male-determining factors are located close to the centromere.[5]

The expected progeny following an exchange between the X and Y chromosomes are shown in Figure 2. An exchange at site *a* will generate a *YL.XS* chromosome and an *XL.5L* chromosome. The model assumes that this pseudo-Y (*YL.XS*) will be biologically equivalent to a normal Y chromosome. The pseudo-Y can be recovered with normal third and fifth chromosomes, thus generating the translocation-free exceptional males. The reciprocal *XL.5L* chromosome can be recovered with the other two elements of the translocation as a *T(X;5;3)*, which would be recovered as a wild type exceptional female. Wild type females were not recovered until late in this two-year study.

A crossover at site *b* would generate the *T(Y;5)* translocation. The reciprocal product would be an *X-3* translocation. The *T(X;3)* would be recovered initially as a mutant female.

While this model explains each chromosomal type recovered, it also generates one phenotypic class that was rarely observed. The appealing feature of this model is that all exceptional classes are the result of the same genetic event: an exchange between homologous chromosomes.

Drosophila

Spontaneous crossing over in males is rare in most *Drosophila* species. This has been shown by direct studies in *D. virilis*,[37] *D. melanogaster* (see below), *D. subobscura*,[56] and *D. willistoni*.[13] In *D. subobscura* and *D. willistoni*, the evidence is cytological. In both cases, males heterozygous for inversions were examined. In *D. subobscura*, one testis out of 300 was found with a cluster of anaphase I cells with chromosome bridges. In *D. willistoni*, 961 F_1 larvae from males heterozygous for nonoverlapping inversions were examined for their inversion phenotypes. Four progeny carrying a recombinant class of inversions were observed. The four exceptions were recovered from different sets of parents, suggesting a meiotic origin of the crossovers.

Two cases of a relatively high frequency of crossing over in males have been reported. One case was *D. ananassae* (see below), and the other was *D. littoralis*.[42] In *D. littoralis*, crossing over in two different pairs of chromosomes was essentially the same in both sexes.

Drosophila melanogaster. Hannah-Alava[18] summarized control crosses for the autosomes through 1968 (see page 127 in reference 18 for listing of sources). Combining this data with other autosomal controls[19,27,58,76] reported since 1968 gives an overall frequency of 0.002% for each of the major autosomes.

Stern and Doan[69] and Lindsley[41] examined spontaneous crossing over between the X and Y chromosomes in males carrying an X chromosome with a heterochromatic duplication. In both cases the exceptions were recovered in clusters. The frequency of exchange events (assuming that a cluster represents one exchange event) was on the order of 0.02%.

It is interesting to note that crossing over between the X and Y chromosomes occurs more frequently than it does between autosomes, although in each case the X chromosome was rearranged. Unfortu-

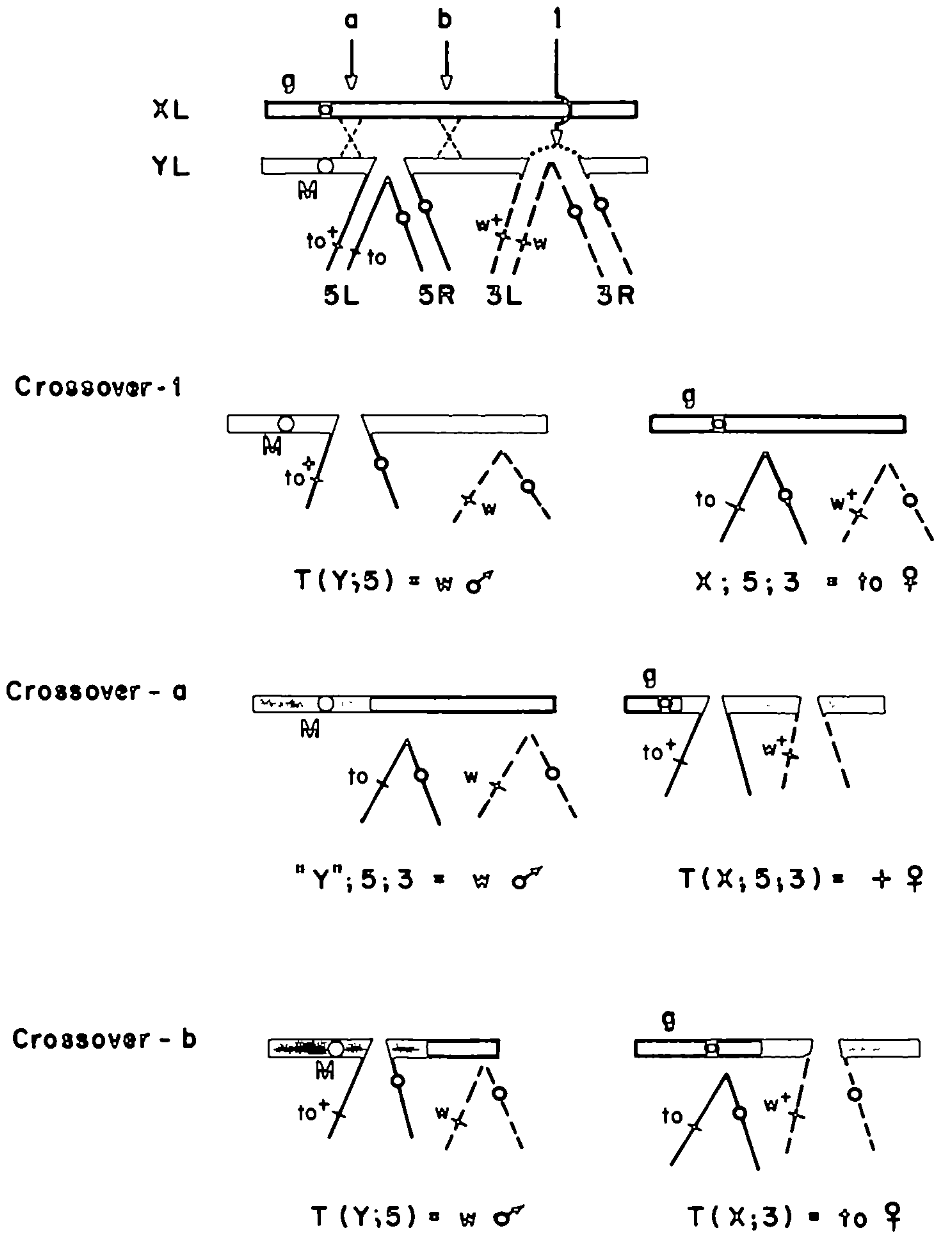

Figure 2. Proposed mechanism of breakdown of *T(Y;5,3)* translocation in heterozygous male of *Lucilia cuprina*. Gametes are listed following proposed crossovers. *M* represents the male-determining factor on the Y chromosome, *g* represents genes on the X chromosome, *to* represents the chromosome 5 gene *topaz* eye, and *w* represents the chromosome 3 gene *white* eye. See Figure 1 for legend to chromosomes. See text for explanation. Diagram modified from Foster et al.[12]

nately, there is no simple way to measure crossing over between a normal X chromosome and a Y chromosome, and so it is not clear whether crossing over is inherently higher for the sex chromosomes or whether the rearranged nature of these chromosomes increased the frequency of exchanges.

The autosomal data suggest that the exchange event is meiotic, as no clusters have been recovered, whereas the sex chromosome data indicate a premeiotic origin for at least some of the exchange events. Whether the mechanism of spontaneous crossing over is different for the sex chromosomes and the autosomes is not known.

High rates of crossing over in autosomes have been described recently, caused by the action of various insertion elements.[26,36,76] Crossing over is only one of several abnormalities caused by these elements. The genetics and molecular biology of insertion elements have been studied extensively and described in detail in various reviews.[7,11,15,63] With respect to crossing over, the findings can be summarized as follows: Crossing over is primarily, if not totally, premeiotic,[10,74,76] different elements exhibit different crossover distributions,[68,76] and the exchanges are symmetrical.[32,71]

Drosophila ananassae. High-frequency crossing over was first described in this species in the late 1930s.[38,50] In the aftermath of World War II, most of the stocks were lost. Recently, new strains have been isolated, and male recombination is being reinvestigated.

Several modifiers of crossing over have been identified. Hinton[20] described a dominant third-chromosome enhancer, a dominant second-chromosome suppressor of the enhancer, and an additional dominant third-chromosome enhancer.[24] The two third-chromosome enhancers do not appear to be alleles.[24]

Matsuda and Tobari[48] described three modifiers of crossing over: a dominant second-chromosome enhancer, a dominant second-chromosome suppressor, and a third-chromosome modifier that decreases crossing over with age. The enhancer and the suppressor mapped to the same region in 2L, suggesting that they may well be alleles.[48] Hinton's suppressor[20] maps to the same region, and this raises the possibility that the two suppressors may be isolates of the same allele. In addition to the specific modifiers, there is evidence for polygenic modifiers,[20,48] sex-linked modifiers,[53] and an extrachromosomal, maternally inherited factor.[21]

It has been shown that reciprocal recombinant classes are recovered equally and that the recombinant chromosomes do not cluster.[33,54] These observations indicate that crossing over is meiotic.

Hinton and Downs[25] observed chiasmata in first-division meiotic figures, but no correlations between chiasmata and crossover frequencies could be made. Matsuda et al.[49] examined meiosis in high and low crossover strains and found a correlation between the chiasmata frequency and the crossover frequency.

Two separate investigations failed to find any synaptonemal complexes in males,[17] although these complexes were observed in females. A third investigation[51] reported incompletely developed synaptonemal complexes in leptotene and zygotene nuclei, but the published micrographs are difficult to interpret.

Another property of *ananassae* stocks is mutability.[22,23,38,72] A correlation between high male recombination and mutation was found in several different stocks,[72] but in a more detailed study various modifiers of mutation and crossing over were found to be different and independent of one

another.[24] One combination of a crossover enhancer and a mutator was found in which the two systems interacted to increase both crossing over and mutation.[24]

At least three mutator systems have been described, each consisting of a mutator function and a suppressor of mutation. One system was chromosomal, with a dominant third-chromosome mutator and an unlinked chromosomal suppressor.[22] The mutator functions in both sexes, but the suppressor acts only in females.[22] Another system includes a third-chromosome mutator and an extrachromosomal suppressor,[24] whereas the third system is entirely extrachromosomal.[20,23]

As noted by Hinton,[22-24] the superficial similarities between recombination and mutation in *D. ananassae* and hybrid dysgenesis in *D. melanogaster* warrant further comparison. Mutation and recombination in *D. ananassae* are controlled by different systems, which, with one exception, are independent. The recombination and mutation associated with hybrid dysgenesis are both the result of the interaction of the *P* (paternal) system of chromosomal elements and the *M* (maternal) cytotype. Both mutation and crossing over are premeiotic in dysgenic systems, whereas crossing over in *D. ananassae* appears to be meiotic. Mutation is probably also meiotic, but this has not been investigated systematically. Thus, recombination and mutation in *D. ananassae* appear to be fundamentally different from hybrid dysgenesis in *D. melanogaster.*

Discussion

Male recombination has been found in several different species of higher Diptera, including members of five superfamilies[1] as well as at least four members of the same genus (*Drosophila*). The only common feature of male recombination is that the frequency is quite low, usually less than 1%. In cases where male crossing over has not been observed,[6,9,14] the sample size has been small. Therefore, it is likely that recombination in males of all higher Diptera probably occurs at a low frequency.

Two additional general conclusions emerge from this survey. First, genomic rearrangements and various insertion elements (including *M*, the sex-determining factor) enhance the frequency of recombination. Second, the recombinational event is usually, if not exclusively, premeiotic. *D. ananassae* and possibly *D. willistoni* are exceptions to these general conclusions. In *D. ananassae*, crossing over appears to be a regular meiotic event that is regulated by a series of crossover enhancers and suppressors, both chromosomal and extrachromosomal. *D. ananassae* may be responding to some selective pressure in nature either to incorporate a regular meiotic system of recombination into males or to maintain a low-level recombination system in males. Various crossover modifiers are segregating in most natural populations.[52]

In other species in which it has been possible to make the distinction, the exchange event has been premeiotic. Mitotic exchange appears to be a regular but rare event in all cells of Diptera.[3,64] Most likely, then, the mechanism responsible for the recombinant chromosomes is a premeiotic mitotic crossover event rather than a true meiotic exchange.

The genetic maps generated by male recombination have the same order as those from females, although the distances are much smaller. In *Musca*[29-31] and *Lucilia*,[12] the male map appears to be a uniform reduction of the female map. However, in *Megaselia*,[8] the reduction is polar, with the greatest reduction occurring at the end distal to the insertion of *M*.

Rearranged chromosomes, primarily translocations, enhance the frequency of crossing over. In studies of somatic crossing over in cuticular and eye tissue of *D. melanogaster*,[3,59] rearranged chromosomes and increased amounts of heterochromatin increased the frequency of mitotic crossing over.

In *D. melanogaster*, there are major differences in crossing over between the sex chromosomes and the autosomes. In the autosomes, crossovers are recovered infrequently and are not clustered, whereas for the sex chromosomes the frequency of exchange is much higher, and the recombinants are usually clustered. Since the X chromosomes used in these studies were rearranged, it is not clear whether the difference is due to the rearranged nature of the chromosomes or whether mitotic exchange is different for the sex chromosomes and for the autosomes. Comparable tests have not been carried out for the other species in this survey. Additionally, in *Drosophila*, spontaneous exchange for the autosomes occurs at a much lower frequency than is the case in any of the other genera examined.

In *M. domestica*, recombination has been reported in XX males, with the sex-determination factor (*M*) located in one of the autosomes. Crossing over has not been found in XY strains.[28] In *M. scalaris*, the chromosome containing the sex factor influences the frequency of recombination for each pair of homologues.

The transposable behavior of *M* in *M. domestica* and *M. scalaris* invites a comparison with the insertion sequences found in dysgenic strains of *D. melanogaster*. The dysgenic factors in *D. melanogaster* induce premeiotic exchanges, mutations, and sterility. Exchange is premeiotic in both *Musca* and *Megaselia*. The *M* element in *M. domestica* causes an increase in male recombination, and the location of *M* in *Megaselia* influences the frequency of recombination. However, neither sterility nor increased mutation has been reported in either species. Until the molecular biology of the *M* elements in *M. domestica* and *M. scalaris* has been studied, any further comparisons with the insertion elements of *D. melanogaster* would be premature.

In *Ceratitis* and *Lucilia*, translocation heterozygosity increases the frequency of male recombination. This is of particular concern because a major effort has been made in these two species to utilize Y-autosome translocations as part of a system of biological control of these two pests. The consequence of the increased recombination in translocation heterozygotes is a loss of desirable markers from males or females or both and a breakdown of the balanced translocation. Surviving terminal aneuploids also complicate these systems. Tight linkage of any desired autosomal genes to the translocation breakpoint should minimize most of the detrimental effects of recombination. Surviving aneuploids probably will be sterile or subviable; thus, their presence should prove to be of minor consequence. The survival of aneuploids can be eliminated by choosing translocations with nonterminal breakpoints.

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