 0030-0950/78/0006-0310$2.00/0

LACK OF AN INTERCHROMOSOMAL EFFECT ASSOCIATED WITH SPONTANEOUS RECOMBINATION IN MALES OF *DROSOPHILA MELANOGASTER*[1]

R. C. WOODRUFF, BARTON SLATKO[2] and JAMES N. THOMPSON, JR.[3], Department of Biological Sciences, Bowling Green State University, Bowling Green, OH 43403

Abstract. It is shown that the frequency of spontaneous male recombination in two different lines of *Drosophila melanogaster* (*OK1* and *T-007*) are not subject to an interchromosomal effect. Second-chromosome male recombination in these lines was not affected by heterozygosity for the multiple third-chromosome inversions *In(3LR)TM3* or *In(3LR)Ubx*^{130}, which do affect recombination in females. It seems, therefore, that a large fraction of spontaneous recombination in males of *D. melanogaster* occurs by some mechanism other than that in females. We discuss the possibility that the mechanism is chromosome breakage and reunion, and that these breakage events may be caused by a microorganism.

OHIO J. SCI. 78(6): 310, 1978

Contrary to previous reports (Morgan 1912, 1914), spontaneous recombination does occur in some males of *Drosophila melanogaster*. Many stocks of *D. melanogaster* that are newly derived from natural populations show male recombination when outcrossed, although at frequencies much lower than those in females (Cardellino and Mukai 1975, Henderson *et al* 1978, Hiraizumi 1971, Hiraizumi *et al* 1973, Kidwell and Kidwell 1975a, 1975b, 1976, Kidwell *et al* 1977, Slatko and Hiraizumi 1973, 1975, Sochacka and Woodruff 1976, Sved 1976, Thompson and Woodruff 1978a, 1978b, Voelker 1974, Waddle and Oster 1974, Woodruff and Thompson 1977, Yamaguchi 1976, Yamaguchi and Mukai 1974). For a review of this topic, see Thompson and Woodruff (1978b).

It is not clear, however, by what mechanism these male recombination events occur. Do they, for example, occur by the same mechanism that produces recombination in females or by some other exchange event (possibly chromosome breakage and reunion)? To determine whether male recombination occurs by the same process as in females, we can examine those parameters affecting frequencies of recombination in females, and ask whether male recombination is similarly affected.

It is known that the frequency of recombination can be increased by structural heterozygosity of other nonhomologous chromosomes—termed the interchromosomal effect (for reviews, see Lucchesi and Suzuki 1968, Lucchesi 1976). In *Drosophila*, only spontaneous recombination in females has been tested for interchromosomal effect. It was our objective, therefore, to determine whether there is an interchromosomal effect on spontaneous recombination in males of *D. melanogaster*. We have observed that male recombination in two different isolated chromosome lines is not influenced by an interchromosomal effect.

MATERIALS AND METHODS

The mutant genes, chromosome rearrangements and wild-type chromosome lines used in this study are listed as follows (see Lindsley and Grell 1968, for detailed descriptions): *aristaless* (*al*, 2L–0.0, antenna), *black* (*b*, 2L–48.0, body color), *brown* (*bw*, 2R–104.5, eye color), *cinnabar* (*cn*, 2R–57.5, eye color), *dumpy* (*dp*, 2L–13.0, wing shape), *veinlet* (*ve*, 3L–0.2, short wing veins), *Inversion(3LR)Third Multiple-3* (*In(3LR)TM3*, 3rd chromosome inversions, balancer, contains the markers y^+, *ri*, p^p, *sep*, bx^{34e}, *Sb* and *Ser*), *Inversion(3LR)Ultra-*

[1]Manuscript received 13 January 1978 and in revised form 21 June 1978 (#78-4).

[2]Present address: Department of Genetics, University of California at Davis, Davis, CA 95616.

[3]Present address: Department of Zoology, University of Oklahoma, Norman, OK 73019.

bithorax^{130} (*In(3LR)Ubx*^{130}, 3rd chromosome inversions, balancer, contains the markers *Ubx*^{130} and *e*^{s}), *Texas–007* (*T–007*, a second chromosome line isolated from a natural population; this line contains the genetic element *Mr*, which is responsible for male recombination induction (Slatko and Hiraizumi 1975)), *Oklahoma–1* (*OK1*, a wild-type strain that shows recombination in males (Woodruff and Thompson 1977)).

The data described in this paper are from two independent studies, one carried out at Austin, Texas by B. Slatko and the other at Cambridge, England and Norman, Oklahoma by R. C. Woodruff and J. N. Thompson, Jr. Different *D. melanogaster* lines exhibiting male recombination were used by the two groups. The *OK1* line, which shows a frequency of male recombination on the second chromosome between *dp* and *bw* of about 3–4% (Woodruff and Thompson 1977), was used by the Cambridge-Norman group, whereas the Austin study used a male recombination line, *T–007*, which shows a frequency of male recombination of about 1% on the second chromosome between *cn* and *bw* (Hiraziumi 1971, Slatko and Hiraziumi 1975).

In the Cambridge-Norman study, all stocks were maintained at 25±2°C on a standard oatmeal, molasses, agar, nipagin medium, whereas in the Austin study, flies were maintained at room temperature (23°–24°C) on a standard cornmeal-agar food, supplemented with propionic acid.

To determine whether third chromosome inversion heterozygosity affects the frequency of recombination in males, one experiment was performed with the *T–007* line (Experiment A) and two experiments were performed using the *OK1* line (Experiments B and C). The inversion chromosomes used in these experiments were *In(3LR)Ubx*^{130} (also called *In(3LR)TM2*) and *In(3LR)TM3*. Both of these multiple inversion chromosomes have been shown to cause an interchromosomal effect in females (Lewis 1952). In addition, we have observed that *In(3LR)TM3* does increase the frequencies of second chromosome recombination in *Tokyo* and *T–007* bearing females (see table 1).

RESULTS

Experiment A: Effect of In(3LR)TM3 on second chromosome recombination in T–007/cn bw males

In experiment A, males carrying the *T–007* second chromosome were mated to heterozygous *In(3LR)TM3* females, which were also homozygous for the second chromosome markers *cn* and *bw*. From this parental mating, individual F_1 *T–007/cn bw; +/+* males were selected and backcrossed to *cn bw* females. In addition, individual F_1 *T–007/cn bw* male sibs, which were also heterozygous for *In(3LR)TM3*, were selected and backcrossed to *cn bw* females. Parental flies were discarded after seven days, and all progeny were scored no later than the 19th day after matings were initiated. The age of flies at the time of mating was 3–5 days old.

By this mating scheme, heterozygous F_1 *T–007/cn bw* males, in the presence and absence of *In(3LR)TM3*, were scored for male recombination induction between the *cn* and *bw* second chromosome markers. Results of these matings are shown in table 2. It can be seen that there was no significant difference in the frequencies of recombination between *T–007/cn bw* males with or without *In(3LR)TM3* ($X_1^2=3.12$, $0.1>P>0.05$).

It has been reported that large clusters of recombinants could be recovered from *T–007* heterozygous males (Hiraizumi *et al* 1973). Since clustered events could influence the interpretation of these results, it was important to determine whether large clusters of male recombinants were recovered in this experiment. Since individual F_1 males were used to detect male recombination, it is possible to identify clustering by determining whether the distribution of recombinants per male fits a Poisson distribution. If clusters do occur, then the distribution of recombinants will not fit

TABLE 1

The influence of In(3LR)TM3 *on second-chromosome recombination in* Tokyo *and* T–007 *bearing females.*

Region	Tokyo + / al dp cn bw + ♀	Tokyo TM3 / al dp cn bw + ♀	T–007 + / al dp cn bw + ♀	T–007 TMS / al dp cn bw + ♀
al–dp	0.1317	0.1745	0.0754	0.1518
dp–cn	0.3459	0.3798	0.3675	0.4051
cn–BW	0.4284	0.4394	0.4212	0.4609
N=	(1139)	(1427)	(1657)	(1291)

a Poisson distribution (Hiraizumi *et al* 1973, Kale 1969, Woodruff and Thompson 1977).

The distribution of male recombination events per male among the progeny of *T–007/cn bw; In(3LR)TM3/+* males was not significantly different from the expected Poisson distribution (index of dispersion $X^2_{85}=85.02$, $P>0.95$). There was, therefore, no large clustering in the progeny of these males. Conversely, the distribution of recombinants from *T–007/cn bw; +/+* males was significantly different from the expected Poisson distribution (index of dispersion $X^2_{140}=241.23$, $P\ll0.001$), suggesting the occurrence of large clusters of male recombinants. This result agrees with previously reported data (Hiraizumi *et al* 1973).

To determine whether clustering of recombinants affects the interpretation concerning the lack of an interchromosomal effect on male recombination, any male which gave more than one recombinant progency was considered to have produced a cluster, and each cluster was counted as a single recombination event. Using this procedure, the recombination frequency in this experiment becomes 37/6406 (0.0058) for progeny from *T–007/cn bw; +/+* males, and 17/3466 (0.0049) for progeny from *T–007/cn bw; In(3LR)TM3/ +* males. These frequencies are not significantly different ($X^2_1=0.31$, $0.7>P>0.5$). This indicates that, even if the phenomenon of clustering is maximally accounted for, there is no interchromosomal effect on male recombination.

Experiments B and C: Effect of In(3LR)TM3 and In(3LR)Ubx^{130} on second chromosome recombination in OK1/ dp b cn bw males

The mating scheme used to test the influence of *In(3LR)TM3* and *In(3LR)Ubx*^{130} on second chromosome recombination in *OK1* males is similar to the mating scheme used for experiment A. *OK1* males were mated to homozygous *dp b cn bw* females, which were also heterozygous for *In(3LR)TM3* (experiment B) or *In(3LR)Ubx*^{130} (experiment C). Heterozygous *OK1/dp b cn bw* males were selected from these parental matings, and five F_1 males were mated to 10 *dp b cn bw; ve* females in half-pint bottles. After 4–5 days, the parents were transferred to fresh bottles for 4–5 more days and were subsequently discarded. F_2 progeny were scored for recombination. In addition, heterozygous *OK1/dp b cn bw* F_1 male sibs, which were also heterozygous for the appropriate multiply rearranged third chromosome, were similarly mated and scored. By these mating schemes, heterozygous *OK1/dp b cn bw* males were scored for male recombination in the presence and absence of the inverted third chromosome. All F_2 progeny were scored for male recombination no later than 18 days after F_1 crosses were initiated. All presumptive recombinants were tested by backcross matings to *dp b cn bw; ve* flies.

The results of experiment B are shown

TABLE 2

Experiment A: Frequencies of Second-Chromosome Recombination for Crosses with T–007/cn bw; In(3LR)TM3/+ *Males and Their* T–007/cn bw; +/+ *Male Sibs.*

Males Tested†	No. F_1 Males Tested	No. Progeny	No. Males Showing Recombination	No. Recom. Progeny	Recom.* Freq.	k†† Value**
T–007/cn bw; +/+	142	6425	37	56	0.0086	0.331
T–007/cn bw; TM3/+	86	3467	17	19	0.0055	0.269

†Males were mated to *cn bw* females.

††k=proportion of non-recombinant +/*marker* progeny from crosses of +/*marker* males to *marker/marker* females (with no segregation distortion, $k=0.5$).

*$X^2_1=3.12$, $P>0.05$.

**$F_{(1,35)}=10.92$, $P>0.01$.

in table 3. There was no significant change in the frequencies of male recombination in the presence of *In(3LR)-TM3* ($X_1^2=0.64$, $0.5>P>0.3$). This was true for all three intervals of the second chromosome that were examined for recombination (*dp-b*, P=0.04 by Fisher's exact probability test; *b-cn*, $X_1^2=0.02$, $0.9>P>0.8$; *cn-bw*, $X_1^2=0.27$, $0.7>P>0.5$). Furthermore, the distribution of recombination events was similar to that previously reported for *OK1/dp b cn bw* males (Woodruff and Thompson 1977); *i.e.*, the majority of male recombination events occurred in the centromeric region (*b-cn*) and in the right arm (*cn-bw*).

It has been reported that large clusters of recombinants (as large as 33% of the total progeny from a single male mating) are recovered from *OK1/dp b cn bw* males (Woodruff and Thompson 1977). Since (as in experiment A) clustered events could influence the interpretation of our results, it is important to determine

TABLE 3

Experiment B: Frequences of Second-Chromosome Recombination from Crosses with OK1/dp b cn bw; In(3LR)TM3/ve *Males and Their* OK1/dp b cn bw;ve/ve *Male Sibs.**

	Region of Second Chromosome Tested for Recom.			Total Recom. Frequencies	*k* Value
	dp-b	*b-cn*	*cn-bw*		
OK1/dp b cn bw;ve/ve					
Bottle 1	0	1 dp b cn^+ bw^+	2 dp^+ b^+ cn^+ bw 2 dp b cn bw^+	5/995(0.0050)	(341 0.34 —— 995
Bottle 2	2 dp b^+ cn^+ bw^+ 2 dp b^+ cn bw^+	2 dp b^+ cn bw^+	3 dp^+ b^+ cn^+ bw 2 dp b^+ cn bw^+	12/1452(0.0075)	693 0.48 —— 1452
Bottle 3	0	1 dp^+ b^+ cn bw	0	1/557(0 0018)	(253 0.45 —— 557
Bottle 4	0	0	4 dp^+ b^+ cn^+ bw 2 dp b cn bw^+	6/1139(0.0053)	(508 0.45 —— 1139
Bottle 5	0	2 dp^+ b^+ cn bw	4 dp^+ b^+ cn^+ bw 6 dp b cn bw^+	12/1909(0.0063)	(981 0.45 —— 1909
Totals	4 (0.0007)**	6 (0.0010)•	25 (0.0043)■	35/6087(0.0057)†	(2776 0.46 —— 6052
OK1/dp b cn bw;TM3/ve					
Bottle 1	0	4 dp^+ b^+ cn bw	8 dp^+ b^+ cn^+ bw 3 dp b cn bw^+	15/1550(0.0097)	(706 0.46 —— 1550
Bottle 2	0	1 dp^+ b^+ cn bw	1 dp b cn bw^+	2/1778(0.0011)	(866 0.49 —— 1778
Bottle 3	0	0	2 dp^+ b^+ cn^+ bw 2 dp b cn bw^+	4/1737(0.0023)	(822 0.47 —— 1737
Bottle 4	0	1 dp^+ b^+ cn bw 3 dp b cn^+ bw^+	6 dp^+ b^+ cn^+ bw 3 dp b cn bw^+	13/1908(0.0068)	(1033 0.54 —— 1908
Totals	0**	9 (0.0013)•	25 (0.0036)■	34/7007(0.0049)†	(3427 0.49 —— 6973

*Males were mated to *dp b cn bw;ve* females.

**P=0.04.

•$X_1^2=0.20$, P>0.05.

■$X_1^2=0.01$, P>0.05.

†$X_1^2=0.64$, P>0.05.

whether large clusters of recombinants were recovered in this experiment.

An analysis of the results in table 3 suggest that no large clusters were recovered in experiment B. This is apparent from the distribution of male recombinants for each interval per bottle, *i.e.*, from the progeny of five males and ten females. From crosses with *OK1/dp b cn bw; In(3LR)TM3/ve* males, the largest possible cluster of recombinants for any one bottle was 11 among 1550 total progeny (0.0071) in bottle 1, whereas from *OK1/dp b cn bw; ve/ve* male sibs, the largest possible cluster was 10 among 1909 progeny (0.0052) in bottle 5. Thus, the results from both crosses of experiment B show little evidence of large clustered recombination events.

Even if all possible clusters of male

TABLE 4

Experiment C: Frequencies of Second-Chromosome Recombination from Crosses with OK1/dp b cn bw; In(3LR)Ubx^{130}/ve *Males and Their* OK1/dp b cn bw;ve/ve *Male Sibs.**

	Region of Second Chromosome Tested for Recom.			Total Recom. Frequencies	*k* Value
	dp-b	*b-cn*	*cn-bw*		
OK1/dp b cn bw;ve/ve					
Bottle 1	0	1 dp^+ b^+ cn bw	2 dp b cn bw^+	3/536(0.0056)	0.52 (281–536)
Bottle 2	0	2 dp b cn^+ bw^+ 1 dp^+ b^+ cn bw	3 dp^+ b^+ cn^+ bw 4 dp b cn bw^+	10/1463(0.0068)	0.52 (757–1463)
Bottle 3	0	1 dp b cn^+ bw^+ 1 dp^+ b^+ cn bw	6 dp^+ b^+ cn^+ bw 4 dp b cn bw^+	12/1077(0 0111)	0.50 (541–1077)
Bottle 4	0	1 dp b cn^+ bw^+ 1 dp^+ b^+ cn bw	1 dp^+ b^+ cn^+ bw 1 dp b cn bw^+	4/392(0.0102)	0.53 (209–392)
Bottle 5	1 dp b^+ cn^+ bw^+ 2 dp^+ b cn bw	2 dp b cn^+ bw^+ 2 dp^+ b^+ cn bw	7 dp^+ b^+ cn^+ bw 5 dp b cn bw^+	17/1031(0.0184)	0.48 (490–1031)
Totals	3 (0.0007)**	12 (0.0027)•	33 (0.0073)■	48/4547(0.0105)†	0.51 (2278–4499)
OK1/dp b cn bw;Ubx^{130}/ve					
Bottle 1	0	0	0	0/112(0.0)	0.61 (68–112)
Bottle 2	0	0	1 dp^+ b^+ cn^+ bw 11 dp b cn bw^+	12/1457(0.0082)	0.53 (766–1457)
Bottle 3	0	1 dp b cn^+ bw^+ 1 dp^+ b^+ cn bw	30 dp^+ b^+ cn^+ bw 5 dp b cn bw^+	37/1172(0.0316)	0.54 (631–1172)
Bottle 4	1 dp b^+ cn^+ bw^+ 2 dp^+ b cn bw	18 dp b cn^+ bw^+ 23 dp^+ b^+ cn bw	27 dp^+ b^+ cn^+ bw 8 dp b cn bw^+	79/1652(0.0478)	0.52 (858–1652)
Bottle 5	0	0	4 dp^+ b^+ cn^+ bw 1 dp b cn bw^+	5/1422(0.0035)	0.58 (827–1422)
Totals	3(0.0005)**	43 (0.0074)•	87 (0.0150)■	133/5948(0.0224)†	0.54 (3150–5815)

*Males were mated to *dp b cn bw;ve* females.

**P=0.77.

•X^2_1=9.77, P<0.01.

■X^2_1=13..7, P<0.001.

†X^2_1=22.5, P<0.001.

recombinants are considered, there would still be no significant difference in the frequencies of recombination in these two types of males. For example, if the occurrence of more than one recombination event for a given interval is considered as a cluster, then the frequency of recombination in the *OK1/dp b cn bw; In(3LR)-TM3/ve* males (7/6980 = 0.0010) is still not significantly different from the frequency in their *OK1/dp b cn bw; ve/ve* sibs (9/5762 = 0.0016) ($X_1^2 = 0.40$, $0.7 > P > 0.5$). Thus, there seems to be no interchromosomal effect on male recombination in experiment B.

The results of experiment C, the effect of *In(3LR)Ubx*^{130} on second chromosome recombination in *OK1/dp b cn bw* males, are shown in table 4. It can be seen that there is a significant increase ($X_1^2 = 23.22$, $P < 0.001$) in the *number* of second chromosome male-recombinant progeny in the presence of *In(3LR)Ubx*^{130}; although, if large clusters are counted as single recombination events, then there is no apparent increase in the *frequency* of male recombination. This increase was due to significant increases in male recombination in the *b-cn* region ($X_1^2 = 0.86$, $P < 0.01$) and in the *cn-bw* region ($X_1^2 = 14.0$, $P < 0.001$), whereas no significant difference was observed for male recombination in the *dp-b* region ($P = 0.77$).

A detailed analysis of the recombinant progeny of these crosses, however, leads us to suspect that this increase is mainly due to the recovery of large presumptive clusters of male recombinants for the *b-cn* and *cn-bw* regions in the *OK1/dp b cn bw; In(3LR)Ubx*^{130}*/ve* males, but not in the *OK1/dp b cn bw; ve/ve* male sibs. These presumptive clusters are evidenced by an analaysis of the distribution of male recombinants in each bottle. From the *OK1/dp b cn bw; Ubx*^{130}*/ve* males there appear to be large clusters in bottle 3 (35/1172 = 0.0299) and in bottle 4 (41/1652 = 0.0248 and 35/1653 = 0.0212).

Effect of third-chromosome inversion heterozygosity on segregation of second chromosomes in the OK1 and T-007 lines

Hiraizumi (1971) and Hiraizumi *et al* (1973) have reported that, in addition to male recombination, male lines bearing *T-007* show distortion of segregation. The amount of distortion is measured by the value k, where k is the proportion of non-recombinant $+/$*marker* progeny from crosses of $+/$*marker* males to *marker marker* females (with no segregation distortion, the Mendelian expectation $k = 0.5$). The total k values for each experiment in this study are shown in tables 2, 3, and 4. In experiments B and C, the second-chromosome k values for the inversion-containing males were significantly higher than in their inversion-free sibs (experiment B, $F_{(1,7)} = 11.96$, $P < 0.01$; experiment C, $F_{(1,8)} = 5.71$, $P < 0.05$), whereas in experiment A the k value in the inversion-bearing males is significantly lower than in their inversion-free brothers ($F_{(1,35)} = 10.92$, $P < 0.01$). This suggests that third-chromosome inversion heterozygosity may affect the segretation of second chromosomes in these male recombination lines, but that this effect is probably geneotype dependent.

DISCUSSION

The results of experiments A and B show that there is no apparent classical interchromosomal effect on spontaneous male recombination. In addition, we believe that after an analysis of possible clusters of male recombinants, the data from experiment C do not contradict this conclusion. Since there is an interchromosomal effect on meiotic and mitotic recombination in females (Lucchesi 1976, Ronen 1964), these results suggest that a large fraction of spontaneous male recombination is occurring by a mechanism other than classical crossing over. What, then, is the mechanism for spontaneous male recombination, and why is this mechanism not influenced by an interchromosomal effect?

Hiraizumi *et al* (1973), Thompson and Woodruff (1978a), Voelker (1974), Woodruff and Thompson (1977), and Yamaguchi (1976) have suggested that male recombination is caused by chromosome breakage and reunion. We believe that their suggestion may be correct, and that chromosome breakage could also cause the other events often associated with male recombination, *i.e.*, sterility, segre-

gation distortion, and mutator activity. Furthermore, structural heterozygosity for one chromosome pair would not, *a priori*, be expected to increase the frequency of breaks on other nonhomologous chromosomes. In this respect, Ronen (1964) has observed that X-ray induced X-chromosome somatic recombination in females (largely heterochromatic chromosomal breakage) was not increased in the presence of autosomal structural heterozygosity. Yet, Lucchesi (1966) has observed an increase in X-ray induced X–Y recombination in females in the presence of autosomal inversions. There appears to be no increase in recombination in irradiated males in the presence of nonhomologous inversions (Ramel 1962, Whittinghill 1955). Thus, in both spontaneous (this report) and induced male recombination (a large part of which seems to be premeiotic in origin), there appears to be a lack of a classical interchromosomal effect.

Chromosome breakage does occur in male recombination lines. This has been shown by salivary gland chromosome analysis (Voelker 1974; Yamaguchi 1976, Yamaguchi and Mukai 1974) and by the observation that frequent chromosome breakage occurs in spermatocytes of outcorssed *OK1* males (but not in inbred *OK1/OK1* males) (Henderson *et al* 1978). In addition, more direct evidence that male recombination occurs by chromosome breakage comes from the analysis of breakage in spermatocytes derived from reciprocal crosses. It has been shown that there is a reciprocal-cross effect associated with male recombination (Hiraizumi and Meador, in preparation; Kidwell and Kidwell 1975a; Sved 1976; Woodruff and Thompson 1977). Heterozygous males from a cross of *marker/marker* females with males from a newly-derived natural population line show recombinant progeny. Yet, a large fraction of heterozygous males from a cross of *marker/marker* males with females from a natural population line show no male recombination. Henderson *et al* (1978) have observed that in the former cross, which shows male recombination, there is chromosome breakage in spermatocytes, but there is no chromosome breakage in spermatocytes from males of the reciprocal cross. Furthermore, when the latter cross is carried one more generation (by backcrossing heterozygous males to *marker* females), recombination and chromosome breakage is again observed in males. These observations give strong support to the suggestion that male recombination, and probably its associated events, are caused by chromosome breakage.

What is causing the chromosome breakage in the outcrossed male recombination lines? It has been suggested that male recombination (and we believe, therefore, chromosome breakage) may be due to a defective repair system (Yamaguchi 1976), a failure of attachment of chromosomes to the nuclear membrane (Sved 1976) or to an episome, virus, or other microorganism (Roberts 1976, Sochacka and Woodruff 1976, Voelker 1974, Waddle and Oster 1974, Woodruff and Thompson 1977, Yamaguchi 1976). The observation that male recombination can be induced by injection of extracts from flies that show male recombination (Sochacka and Woodruff 1976) and can also be induced by rearing flies in media supplemented with these extracts (Hellack *et al* 1978) tends to rule out the first two of these alternatives. It is known, however, that viruses can induce chromosome breakage in *Drosophila* (Gershenson *et al* 1971). It could be conjectured, thereore, that natural populations of *Drosophila* contain viruses or other such microorganisms and, upon outcrossing the flies with laboratory strains, these microorganisms cause chromosome breakage leading to male recombination and related events. If true, these microorganisms may play a role in the evolution of *Drosophila*, as has been suggested by other workers (L'Heritier 1970, Reanney 1974).

Acknowledgments. We wish to express our gratitude to Drs. M. Ashburner, J. Holden, Y. Hiraizumi, and J. Hellack, and to Kathy Matthews for helpful discussions concerning this report. B. Slatko was supported by research grant NIH GM-19770 to Dr. Y. Hiraizumi. Part of this work was done at the University of Cambrdige while R. C. Woodruff was supported by an M.R.C. grant to Dr. M. Ashburner and Professor B. Clarke. J. N. Thompson, Jr. and R. C. Woodruff were supported by DHEW-NIEHS grant 1-R01-ES01439-01.

LITERATURE CITED

Cardellino, R. A. and T. Mukai 1975 Mutation factors and genetic variance components of viability in *Drosophila melanogaster*. Genetics 80: 567–583.

Gershenson, S., Y. N. Alexandrov and S. S. Maliuta 1971 Production of recessive lethals in *Drosophila* by viruses noninfectious for the host. Mutation Res. 11: 163–173.

Hellack, J. J., J. N. Thompson, Jr., R. C. Woodruff and B. N. Hisey 1978 Male recombination and mosaics induced in *Drosophila melanogaster* by feeding. Experientia. 34: 337.

Henderson, S. A., R. C. Woodruff and J. N. Thompson, Jr. 1978 Spontaneous chromosome breakage at male meiosis associated with male recombination in *Drosophila melanogaster*. Genetics 88: 93–107.

Hiraizumi, Y. 1971 Spontaneous recombination in *Drosophila melanogaster* males. Proc. Natl. Acad. Sci. 68: 268–270.

———, B. Slatka, C. Langley and A Nill 1973 Recombination in *Drosophila melanogaster* male. Genetics 73: 439–444.

Kale, P. G. 1969 The meiotic origin of spontaneous crossovers in *Drosophila ananassae* males. Genetics 62: 123–133.

Kidwell, M. G. and J. F. Kidwell 1975a Cytoplasm-chromosome interactions in *Drosophila melanogaster*. Nature Lond. 253: 755–756.

——— 1975b Spontaneous male recombination and mutation in isogenic-derived chromosomes of *Drosophila melanogaster*. Proc. Natl. Acad. Sci. 38: 953–961.

——— 1976 Selection for male recombination in *Drosophila melanogaster*. Genetics 84: 333–351.

——— and P. T. Ives 1977 Spontaneous nonreciprocal mutation and sterility in strain crosses of *Drosophila melanogaster*. Mutation Res. 42: 89–98.

Lewis, E. B. 1952 The psuedoallelism of *white* and *apricot* in *Drosophila melanogaster*. Proc. Natl. Acad. Sci. 38: 953–961.

L'Heritier, Ph. 1970 *Drosophila* viruses and their role as evolutionary factors. Evolutionary Biol. 4: 185–209.

Lindsley, D. L. and E. H. Grell 1968 Genetic variations of *Drosophila melanogaster*. Carnegie Instit. Washington, Publ. No. 672.

Lucchesi, J. C. 1966 The effect of heterozygous autosomal inversions on induced exchanges between the attached-X and Y chromosomes in *Drosophila melanogaster* females. Genetics 54: 1013–1018.

——— 1976 Inter-chromosomal effects. *In:* M. Ashburner and E. Novitski (eds.) The Genetics and Biology of *Drosophila*, Volume 1A. Academic Press, New York.

——— and D. T. Suzuki 1968 The interchromosomal control of recombination. *In:* H. L. Roman (ed.) Annual Review of Genetics, Volume 2. Annual Reviews, Palo Alto, CA.

Morgan, T. H. 1912 Complete lingage in the second chromosome of the male *Drosophila*. Science 36: 718–720.

——— 1914 No crossing over in the male of *Drosophila* genes in the second and third pairs of chromosomes. Biol. Bull. 26: 195–204.

Ramel, C. 1962 Interchromosomal effect of inversions in *Drosophila melanogaster*. I. Crossing over. Hereditas 48: 1–58.

Reanney, D. C. 1974 Viruses and evolution. Int. Rev Cytol 37: 21–44.

Roberts, P. A. 1976 The genetics of chromosome aberration. *In:* M. Ashburner and E. Novitski (eds.) The Genetics and Biology of *Drosophila*, Volume 1A. Academic Press, New York.

Ronen, A. 1964 Interchromosomal effects on somatic recombination in *Drosophila melanogaster*. Genetics 50: 649–658.

Slatko, B. 1976 Mutator elements in natural populations of *Drosophila melanogaster:* The *high* mutator gene revisited. Mutation Res. 36: 387–390.

——— and Y. Hiraizumi 1973 Mutation induction in the male recombination strains of *Drosophila melanogaster*. Genetics 75: 643–649.

——— 1975 Elements causing male crossing over in *Drosophila melanogaster*. Genetics 81: 313–324.

Sochacka, J. H. M. and R. C. Woodruff 1976 Induction of male recombination in *Drosophila melanogaster* by injection of extracts of flies showing male recombination. Nature Lond. 262: 287–289.

Sved, J. A. 1976 Hybrid dygenesis in *Drosophila melanogaster:* A possible explanation in terms of spatial organization of chromosomes. Australian J. Biol. Sci 29: 375–388.

Thompson, J. N., Jr. and R. C. Woodruff 1978a Chromosome breakage: A possible mechanism for diverse genetic events in outbred populations. Heredity 40: 153–157.

——— 1978b Mutator genes: Pacemakers of evolution. Nature Lond. 274: 317–321.

Voelker, R. A. 1974 The genetics and cytology of a mutator factor in *Drosophila melanogaster*. Mutation Res. 22: 265–276.

Waddle, F. R. and I. I. Oster 1974 Autosomal recombination in males of *Drosophila melanogaster* caused by a transmissible factor. J. Genet. 61: 177–183.

Whittinghill, M. 1955 Crossover variability and induced crossing over. J. Cell Comp. Physiol. 45 Suppl. 2: 189–220.

Woodruff, R. C. and J. N. Thompson, Jr. 1977 An analysis of spontaneous recombination in *Drosophila melanogaster* males: Isolation and characterization of male recombination lines. Heredity 38: 291–307.

Yamaguchi, O. 1976 Spontaneous chromosome mutation and screening of mutator factors in *Drosophila melanogaster*. Mutation Res. 34: 389–406.

——— and T. Mukai 1974 Variation of spontaneous occurrence of chromosomal aberrations in the second chromosomes of *Drosophila melanogaster*. Genetics 78: 1209–1221.