CHROMOSOMAL SITES NECESSARY FOR NORMAL LEVELS OF MEIOTIC RECOMBINATION IN *DROSOPHILA MELANOGASTER*.[1] I. EVIDENCE FOR AND MAPPING OF THE SITES

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ABSTRACT

Meiotic exchange was measured in females heterozygous for a normal sequence *X* chromosome and for each of eleven *T(1;4)*s and each of sixteen *T(1;Y)*s. The results indicate that the *X* chromosome can be divided into five intervals, such that heterozygosity for a breakpoint in one interval strongly suppresses exchange within that interval, but has little or no effect on exchange in other intervals. The boundaries between these intervals are identified and mapped to regions 3C4–6/7, 7A-7E, 11A and proximal to 18C on the standard salivary map; each boundary is located at (or within a small region containing) a major constriction (*i.e.*, a block of intercalary heterochromatin).—— Exchange was examined in females heterozygous for translocations broken within the constriction at 11A. The results imply that a boundary occupies only a subregion of the entire constriction and is subdivisible by translocation breakpoints. Several other properties of boundaries have been elucidated. Finally, the relationship of these data to a simple model of meiotic pairing proposed by I. SANDLER (1956) and to the role of intercalary heterochromatin in the meiotic process is discussed.

TRANSLOCATIONS, when heterozygous with normal sequence chromosomes, behave as region-specific dominant suppressors of exchange, with the effect being most pronounced in the region surrounding the breakpoint (DOBZHANSKY 1931; STONE 1934). Considerable evidence suggests that this suppression is due to an abnormality of pairing in the region of the breakpoint, rather than to the elimination of crossover chromatids (reviewed by ROBERTS 1970; also see BURNHAM 1932, 1944, 1968). A systematic study of exchange suppression by translocations led ROBERTS (1972) to suggest that regions highly susceptible to exchange suppression by translocations represent those regions in which synapsis is initiated. The existence of similar regions was also postulated by BURNHAM *et al.* (1972) on the basis of a cytological analysis of compound translocation heterozygotes in maize.

An examination of meiotic exchange in Drosophila females carrying a normal-sequence *X* chromosome and either one of two translocations between the *X*

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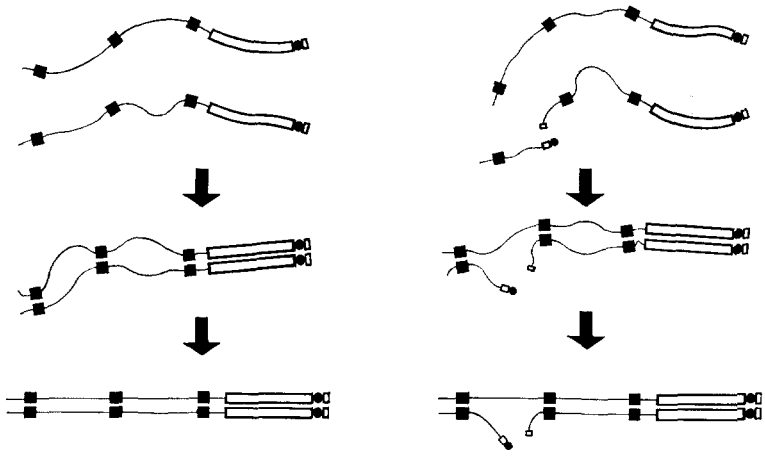

FIGURE 1.—A diagrammatic representation of I. SANDLER's hypothesis displaying the early stages of meiotic pairing between two normal sequence *X* chromosomes (left-hand sequence) and in a heterozygote for a *T(1;4)* (right-hand sequence). The dark squares represent the putative "pairing sites."

and fourth chromosomes led I. SANDLER (1956) to suggest a somewhat different role of specific chromosomal regions or sites in initiating chromosome pairing and thus facilitating normal levels of meiotic exchange. She observed that *T(1;4)8B1* [= *T(1;4)A17*], when heterozygous, almost eliminated exchange in the distal region of the *X* chromosome, but did not affect proximal recombination. On the other hand, exchange in females heterozygous for *T(1;4)16A1* [= *T(1;4)B*^{s}] was reduced some ten-fold in the proximal region, but not reduced at all in the distal region of the *X* chromosome. To explain these results, she proposed that the *X* chromosome was divided into at least two exchange intervals that are bounded by three "pairing sites," one located distally, one medially and one proximally. She proposed that the presence of a discontinuity, owing to heterozygosity for a translocation breakpoint, in an interval between two pairing sites was sufficient to prevent exchange, and presumably meiotic pairing, within the entire interval. A diagrammatic representation of SANDLER's hypothesis is presented in Figure 1. This report presents a series of tests that tend to validate the existence of such specific chromosomal sites.

RESULTS AND DISCUSSION

The basic experiments reported here measure meiotic exchange in females heterozygous for an *X* chromosome translocation—either a *T(1:4)* or a *T(1;Y)*—and for a normal sequence, but multiply marked, *X* chromosome. The translocations are listed by breakpoint in Table 1; detailed descriptions of many of these aberrations and all of the mutants used are found in LINDSLEY and GRELL (1968). In this report, translocations are referred to by breakpoint rather than by "common name" (for example, *T(1;4)B*^{s} is referred to as *T(1;4)16A1*). However, the set of five *T(1;Y)*s broken in region 11A will be distinguished by the stock num-

TABLE 1

A list of the aberrations used in this study

Translocations*	"Common name"	Reference†	Translocations*	"Common name"	Reference†
T(1;4)1A5	A10	STONE (1934)	*T(1;Y)5C*	B36	STEWART and MERRIAM (1973)
T(1;4)3B1	JC43	YOUNG and JUDD (1978)	*T(1;Y)7A*	B123	MERRIAM (unpubl.)
T(1;4)3C3	w^{m5}	GRIFFEN and STONE (1938)	*T(1;Y)7E*	B170	MERRIAM (unpubl.)
			T(1;Y)9B	B167	MERRIAM (unpubl.)
T(1;4)3C6/7	N^{8a}	JUDD (1955)	*T(1;Y)10A*	B105	MERRIAM (unpubl.)
T(1;4)4C3	—	LINDSLEY (unpubl.)	*T(1;Y)10C*	B145	MERRIAM (unpubl.)
T(1;4)5A1	A9	STONE (1934)	*T(1;Y)11A*	B87	MERRIAM (unpubl.)
T(1;4)8B1	A17	PATTERSON, STONE and BEDICHEK (1937)	*T(1;Y)11A*	B45	STEWART and MERRIAM (1973)
T(1;4)9A1	—	—‡	*T(1;Y)11A*	B53	STEWART and MERRIAM (1973)
T(1;4)11A6/7	A8	STONE (1934)			
T(1;4)11C/D	N87	LEFEVRE (unpubl.)	*T(1;Y)11A*	B44	STEWART and MERRIAM (1973)
T(1;4)13B	Sidsky-a	—‡			
T(1;4)13F6	A4	STONE (1934)	*T(1;Y)11A*	D9	MERRIAM (unpubl.)
T(1;4)16A1	B^S	SANDLER (1956)	*T(1;Y)11D*	B88	MERRIAM (unpubl.)
T(1;4)18C5	A13	STONE (1934)	*T(1;Y)12A*	B166	MERRIAM (unpubl.)
T(1;4)A19	—	STONE and GRIFFEN (1940)	*T(1;Y)13A*	B128	MERRIAM (unpubl.)
			T(1;Y)16A	B106	NICOLETTI and LINDSLEY (1960)
T(1;4)A20	—	STONE and GRIFFEN (1940)	*T(1;Y)18A*	B50	STEWART and MERRIAM (1973)
T(1;Y)4C	B163	MERRIAM (unpubl.)			

* The breakpoints listed are those determined by the investigator(s) who isolated the aberration, except for *T(1;Y)7A(B123)*, whose breakpoint was initially placed in 7E.

† Denotes the reference most relevant to the exchange behavior of the aberration. In cases where the exchange behavior has not been studied, the reference reporting the isolation of the aberration is listed.

‡ Obtained from Bowling Green Stock Center.

bers assigned to them by J. MERRIAM and the two Robertsonian fusions of the *X* and fourth chromosomes having breakpoints near the centromere of the *X* chromosome will be referred to as *T(1;4)A19* and *T(1;4)A20*.

All crosses were performed as single pair matings that were transferred to a fresh vial on day 5 and discarded on day 9. No significant differences between the two broods were noticed in any of the crosses reported; the data reported here are the sum of both broods.

Exchange in translocation heterozygotes: Tables 2 and 3 show the results of examining exchange in females heterozygous for each of 11 *T(1;4)*s and for either of two multiply marked normal sequence *X* chromosomes. In both Tables 2 and 3, the control cross measures exchange in females heterozygous for the multiply marked *X* chromosome and for a wild-type, Canton-S, *X* chromosome. The map lengths observed in the control cross for the w^a–*m*, *pn*–*m* and *m*–*f* regions are very close to the standard values (LINDSLEY and GRELL 1968). Although the total *pn*–*m* map length approximates the standard value, the map lengths of 6.7 for *pn*–*cv* and 25.9 for *cv*–*m* differ significantly from the standard

TABLE 2

Results of crossing T(1;4) / y w^a m f *females to* y w^a m f / Y *males*

Type of exchange*	Control	Breakpoint of the *T(1;4)* 3B1	3C3	3C6§	4C3	8B1	9A1	11C/D	13B	16A1	*Xh* A19	A20
NCO	2068	601	421	883	734	1500	1297	2234	2763	1855	646	989
SCO 1	24	0	1	0	0	0	1	14	25	9	10	18
2	1271	224	174	225	164	52	59	403	844	1047	448	670
3	517	171	92	309	175	247	135	59	46	65	131	189
DCO 1,2	3	0	0	0	0	0	0	0	0	0	4	4
1,3	8	0	0	0	0	0	0	0	0	0	1	1
2,3	171	19	21	34	15	15	7	1	8	3	42	49
Total	4068	1015†	709†	1451†	1078†	1814‡	1499‡	2711	3686	2884	1282	1970
Aneuploid ♀ ♀	0	—	—		—	109	40	0	17	30‖	—	—
Map length												
y – w	0.9	0.0	0.0	0.0	0.0	0.0	0.1	0.5	0.7	0.3	1.2	1.2
w – m	35.5	23.9	27.5	17.8	16.6	3.7	4.4	14.9	23.1	36.4	38.5	36.7
m – f	17.1	18.7	15.9	23.6	17.6	14.4	9.5	2.2	1.5	2.3	13.6	12.1

* Regions 1, 2 and 3 represent the *y – w*, *w – m* and *m – f* intervals, respectively.
† Only *y* progeny are included.
§ In this cross, the normal sequence *X* chromosome was also marked with *ct*. Of the SCOs, 201 were between *ct* and *m*, as were 18 of the DCOs.
‡ In both of these cases, *m f* female progeny may arise as a result of either exchange or nondisjunction of the distal piece of the translocation from the *X* chromosome. The number of *m f* females resulting from exchange was estimated from the *y* w^a females. This estimate agreed very well, in both cases, with the number of *m f* males.
‖ Ten hyperploid males were also recovered.

values of 12.4 and 19.7. Since *T(1;4)16A1/y pn cv m f·y*$^+$ heterozygotes, having a control level of *pn–m* exchange, show standard *pn–cv* and *cv–m* map lengths, the control variations may be due to the Canton-S chromosome. Fortunately, the analysis presented below requires only a comparison of the *pn–m* and *m–f* intervals.

Data of STONE (1934) for *T(1;4)1A5*, *T(1;4)5A1*, *T(1;4)11A6/7*, *T(1;4)13F6* and *T(1;4)18C5* are presented in Table 4. These five translocations have all been lost. The multiply marked normal sequence chromosome used for the crosses involving *T(1;4)5A1* and *T(1;4)18C5* did not carry the markers *y* or *m* but did carry *sc* and *v*. In this analysis, the *sc–v* and *v–f* intervals will be equated with the *y–m* and *m–f* intervals, respectively.

TABLE 3

Results of crossing T(1;4)/y pn cv m f·y+ *females to* y pn cv m f·y+/Y *males*

Map length	Control	Breakpoint of the *T(1;4)* 3C3	4C3	8B1	9A1	11C/D	13B	16A1
pn – cv	6.7	3.2	0.8	0.6	1.6	10.1	12.5	12.7
cv – m	25.9	20.8	14.3	1.2	1.3	4.0	13.3	17.9
m – f	13.0	16.7	18.1	8.0	5.7	0.9	1.4	2.2
N	1246	1073	1438	2394	4502	2103	1196	955

TABLE 4

Data of STONE *(1934) regarding exchange in three* T(1;4)/sc ec ct v g f *and two* T(1;4)/y w^a m f *heterozygotes*

Map lengths	Control	Breakpoint 1A5	5A1	11A6/7	13F6	18C5§
sc – ct	22.8	—	1.2	—	—	24.7
ct – v	13.4	—	8.7	—	—	11.2
v – g	11.1	—	9.0	—	—	4.2
g – f	10.8	—	14.5	—	—	1.9
y – w^a	—	0.8	—	1.6	0.2	—
w^a – m	—	28.5	—	2.4	16.4	—
m – f	—	20.6	—	3.4	2.0	5.8
N	2137	1213	2563	637	1388	1405

Because the *T(1;4)*s were obtained from a variety of sources (see Table 1) they are of various genetic backgrounds. To assess the effect of different genetic backgrounds on exchange in translocation heterozygotes, crossing over was measured using two different multiply marked *X* chromosomes (Table 5). It can be seen that, in several paired comparisons, noticeable differences in the total map length between *y* and *f* are observed, which very likely reflect the effect of

TABLE 5

A comparison of the total amount of exchange from y *to* f *and the distribution of exchanges in the proximal and distal half of the chromosome when exchange is measured with two different multiply marked chromosomes*

Breakpoint	Multiply marked chromosome used	Total map length	Fraction of total *y – f* map length *y – m*	*m – f*
Control	*y pn cv m f·y+*	45.6	0.71	0.29
	y w^a m f	52.6	0.67	0.33
3C3	*y pn cv m f·y+*	40.7	0.59	0.41
	y w^a m f	43.4	0.64	0.36
4C3	*y pn cv m f·y+*	32.4	0.44	0.56
	y w^a m f	32.2	0.48	0.52
8B1	*y pn cv m f·y+*	9.8	0.18	0.82
	y w^a m f	18.1	0.20	0.80
9A1	*y pn cv m f·y+*	8.6	0.34	0.66
	y w^a m f	14.0	0.32	0.68
11C/D	*y pn cv m f·y+*	14.9	0.94	0.06
	y w^a m f	17.6	0.88	0.12
13B	*y pn cv m f·y+*	27.2	0.95	0.05
	y w^a m f	25.3	0.94	0.06
16A1	*y pn cv m f·y+*	32.8	0.93	0.07
	y w^a m f	37.8	0.94	0.06

genetic background. However, despite differences in the total amount of *y–f* exchange, the fraction of those exchanges that occur in the *y–m* region is remarkably constant. Accordingly, the effect of each *T(1;4)* on the distribution of those exchanges that do occur will be used as the basis for comparing the effects of the various *T(1;4)*s.

In Figure 2, the fraction of the total *y–f* exchange that occurs in the *y–m* interval is plotted against the breakpoint for each *T(1;4)/y* w^a *m f* heterozygote. Clearly, the *T(1;4)*s may be divided into three groups on the basis of their effects on recombination. The three *T(1;4)*s broken distal to 3C3 and the two translocations with breakpoints in the proximal heterochromatin do not affect the distribution of exchanges between *y* and *f*; those *T(1;4)*s broken between 3C6/7 and 9B suppress exchange in the *y–m* interval; those *T(1;4)*s broken proximal to 11A but distal to the proximal heterochromatin suppress exchange primarily in the *m–f* interval (the behavior of the *T(1,4)* broken at 11A will be discussed below). These observations allow the *X* chromosome to be divided into three intervals such that all *T(1;4)*s broken in a given interval affect exchange in a similar way. The distal interval is defined by the breakpoints of those *T(1;4)*s that do not suppress exchange; the medial interval defined by those *T(1;4)*s that suppress exchange primarily between *y* and *m*; and the proximal interval is defined by the breakpoints of that group of translocations that suppress exchange primarily between *m* and *f*.

The boundary between the distal and medial intervals must lie between 3C3 and 3C6/7, an interval that includes a major constriction in the salivary gland chromosome. The boundary between the medial and proximal intervals must lie between 9B and 11C. Moreover, *T(1;4)11A6/7*, which is broken in the constric-

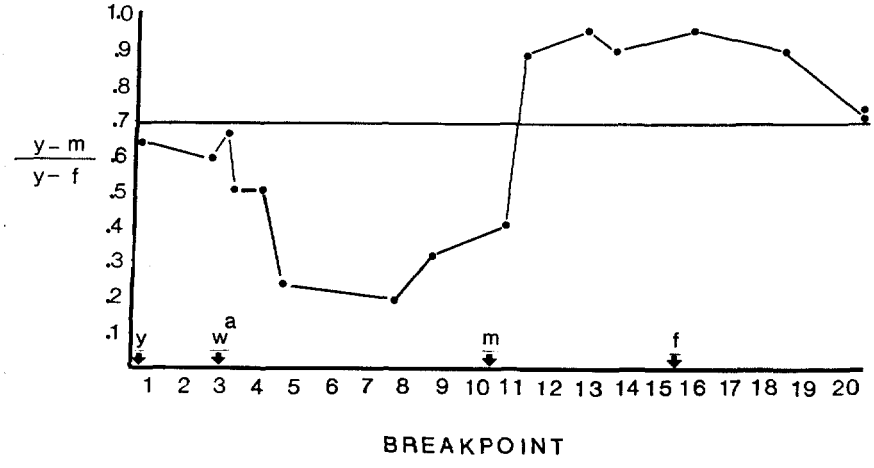

FIGURE 2.—The fraction of *y–f* exchange occurring in the *y–m* region (ordinate) plotted against the breakpoint of the *T(1;4)* (abscissa) for each *T(1;4)* heterozygote. The horizontal line represents the control value. The positions of the markers are indicated by arrows. For *T(1;4)5A1*, *T(1;4)11A6/7* and *T(1;4)18C5*, the value plotted is the fraction of *sc–f* exchange occurring in the *sc–v* interval.

tion in region 11A, is the only translocation to suppress exchange in both the medial and proximal intervals (see Table 4). Thus, its breakpoint is likely at or very near to the boundary between these intervals, placing that boundary in or near 11A, which is the site of another major constriction. That the region between *m* and the centromere must consist of two intervals with a boundary that maps proximally to the breakpoint of *T(1;4)18C5* is demonstrated by the behavior of *T(1;4)A19* and *T(1;4)A20*, which are broken near the centromere of the *X* chromosome but do not affect recombination. This fourth interval, which includes the heterochromatin, has not been studied and therefore will not be discussed further. The region between 11A and 18C5 will be referred to as the proximal interval.

Therefore, while the *X* chromosome is divisible into at least four discrete intervals, it should be noted that *T(1;4)*s broken distally in the *m–f* interval also weakly suppress exchange in the w^a–*m* interval and that *T(1;4)9A1*, broken in the distal interval, weakly suppresses *m–f* exchange. As shown below, these suppressions result from the proximity of these regions to the fourth chromosome centromere or telomere, and are not the result of heterozygosity for a breakpoint. Furthermore, the inequality of exchange suppression in the w^a–*m* interval observed among *T(1;4)*s broken within that interval (see Table 2) is due to a fourth boundary mapping in region 7 that divides the w^a–*m* region into two intervals. This boundary is obscured in Figure 2 by the absence of a marker subdividing the interval and by the effects of the fourth chromosome centromere and telomere.

The centromere effect: BEADLE (1932) demonstrated that the chromosome *4* centromere causes a suppression of exchange that weakens with increasing distance from the centromere (the centromere effect). The argument that the centromere effect is responsible for the slight w^a–*m* exchange suppression caused by *T(1;4)*s broken proximal to *m* is based on a comparative analysis of exchange in *T(1;4)* and *T(1;Y)/y* w^a *m f* heterozygotes with breakpoints proximal to *m*. Because the *Y* chromosome is many times larger than chromosome *4*, there is, in most cases, much more *Y* chromosome material between the *Y* centromere and the breakpoint of a *T(1;Y)* than there is chromosome *4* material between the centromere and a comparable breakpoint of a *T(1;4)*. This should reduce the severity of the w^a–*m* exchange suppression in *T(1;Y)*s as compared with *T(1;4)*s.

The results of measuring recombination in six *T(1;Y)/y* w^a *m f* heterozygotes are presented in Table 6. As illustrated in Figure 3, *T(1;Y)*s broken distally in the *m–f* interval have an effect qualitatively similar to, but (in most cases) much less severe, on w^a–*m* exchange, than do *T(1;4)*s with similar breakpoints, although *T(1;4)*s and *T(1;Y)*s broken between region 11 and region 18 are, in general, equally effective in suppressing exchange between *m* and *f* (Tables 2 and 6). However, given the large size of the *Y* chromosome, some *T(1;Y)*s broken proximally on the *Y* should exert a strong centromere effect, while some broken very distally might be expected to show a very weak effect. Thus, *T(1;Y)12C* is a much stronger suppressor of w^a–*m* exchange than is *T(1;Y)11D*, while *T(1Y;)11A(B53)* shows no suppression of w^a–*m* exchange (Table 6).

TABLE 6

Results of crossing T(1;Y) y·B^{S}, y+/y w^{a} m f *females to* y w^{a} m f/Y *males*

Type of exchange*	Breakpoint of the *T(1;Y)* 11A†	11D	12C	13A	16A†	18A‡	Control‖
SCO 1	1034	1300	860	821	786	624	—
SCO 1	583	454	147	342	523	392	—
2a	0	0	0	3	—	—	—
2b	34	27	18	13	—	—	—
2	1	27	18	16	80	29	—
3	—	—	—	—	—	0	—
DCO 1,2	0	2	1	2	10	1	—
1,2a	0	0	0	0	—	—	—
1,2b	10	2	1	2	—	—	—
2a,2b	10	0	0	0	—	—	—
Total	1661	1783	1026	1181	1399	1046	—
Aneuploid ♀ ♀	415	396	328	323	—	460¶	—
Map length							
$w^{a}-f$	38.3	27.2	16.2	30.6	44.5	40.5	52.6
$w^{a}-m$	37.5	25.6	14.4	29.1	38.1	37.6	35.5
$m-f$	2.6	1.6	1.9	1.5	6.4	2.9	17.1
Fraction of total exchange							
$w^{a}-m$	0.93	0.94	0.88	0.95	0.86	0.93	0.67
$m-f$	0.07	0.06	0.12	0.05	0.14	0.07	0.33

* The intervals $w^{a}-m$, $m-f$ and $f-$18A are designated as intervals 1, 2 and 3, respectively. The designations 2a and 2b are used to represent the $m-$ breakpoint and breakpoint $-f$ intervals respectively.

† This cross involves *T(1;Y)11A(B53)*.

§ *T(1;Y)16A* does not carry y^{+} on the distal element.

‡ *T(1;Y)18A* is broken in Y^{L}.

‖ Data are presented in Table 2.

¶ 197 males hyperploid for *Xp* were also obtained.

An alternative to the centromere effect is that breakpoints involving the *X* and *Y* chromosomes are inherently less able to suppress exchange distally to the breakpoint than are breakpoints involving the *X* and chromosome *4*. However, this explanation is inadequate because *T(1;Y)18A* is as effective at suppressing *m–f* exchange as *T(1;4)18C5* (Table 4).

Thus, those w^{a}–*m* exchange suppressions observed in translocations broken proximal to *m* are most readily accounted for as a result of the centromere effect. However, the centromere effect accounts neither for the fact that translocations broken in region 11 suppress exchange proximal to their breakpoint nor for the similarity in the extent of exchange suppression observed in the *m–f* interval for all translocations broken proximal to *m*.

Evidence for a telomere effect: To determine whether or not the *m–f* exchange suppression observed in *T(1;4)9A1* heterozygotes is a general property of translocations broken proximally in the w^{a}–*m* interval, exchange was measured in females heterozygous for one of seven *T(1;Y)*s with breakpoints distal to *m* and

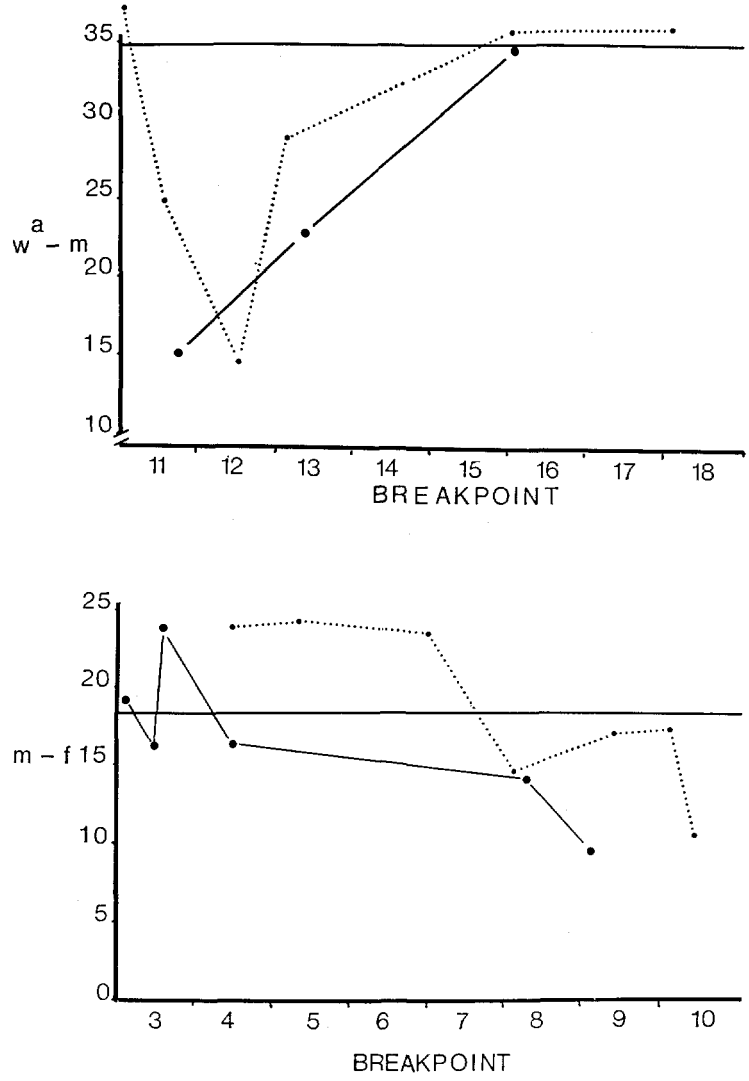

FIGURE 3.—Effects on *T(1;Y)*s (.....) and *T(1;4)*s (——) on exchange in the adjoining interval. (Top panel) The map length of the w^a–*m* interval (ordinate) plotted against the breakpoint (abscissa) of those translocations broken proximal to 11A. (Lower panel) The map length of the *m*–*f* interval (ordinate) plotted against the breakpoint (abscissa) of those translocations broken distal to 11A. The horizontal line indicates the control value. Data of STONE are not included.

for the *y* w^a *m f* chromosome (Table 7). Figure 3 presents a comparison of the effects of *T(1;4)*s and *T(1;Y)*s with breakpoints distal to *m* on exchange between *m* and *f*. (The effects of these translocations on w^a–*m* exchange will be considered in the following section.)

It may be seen that *T(1;Y)*s with breakpoints distal to *m* allow more *m*–*f* exchange than do *T(1;4)*s. Furthermore, although *T(1;4)9A1* reduces *m*–*f*

TABLE 7

Results of crossing T(1;Y) y·B^{S}, y+/y w^{a} m f *females to* y w^{a} m f/Y *males where the breakpoint was distal to* m

Type of exchange*	Breakpoint of the *T(1;Y)* 4C	5C	7A	7E	9B	10A	10C	Control§
NCO	918	950	1307	1580	996	943	1451	—
SCO 1a	21	5	28	82	408	318	438	—
1b	427	226	188	85	17	9	0	—
1	448	231	216	167	425	327	438	—
2	370	375	435	288	219	214	181	—
DCO 1,2	43	6	25	13	67	34	32	—
1a,2	3	0	14	11	67	33	32	—
1b,2	40	6	9	3	0	1	0	—
1a,1b	2	0	1	0	0	0	0	—
Total	1781	1562	1983	2049	1707	1468	2102	—
Aneuploid ♀ ♀	353	295	277	160	113	172	403	—
Map length								
$w^a - f$	51.0	39.6	35.5	23.5	45.6	41.5	32.5	52.6
$w^a - m$	27.8	15.2	12.2	8.8	28.8	24.6	22.4	35.5
$m - f$	23.2	24.4	23.3	14.7	16.8	16.9	10.1	17.1
Fraction of total								
$w^a - m$	0.54	0.38	0.35	0.37	0.63	0.59	0.69	0.66
$m - f$	0.46	0.62	0.65	0.63	0.37	0.41	0.31	0.33

* The regions 1a, 1b and 2 correspond to the w^a – breakpoint, breakpoint – m, and $m - f$ regions, respectively.
† These aneuploid female progeny are reported in detail in Table 11.
§ Data are presented in Table 2.

exchange, *T(1;Y)9B* and *T(1;Y)10A* allow near normal levels of *m–f* exchange. Only *T(1;Y)10C* reduces *m–f* exchange to a level similar to that of *T(1;4)9A1*. Thus, the ability of *X* chromosome translocations that have breakpoints distal to *m* to suppress *m–f* exchange is not a consequence of the location of the breakpoint, but of the other chromosome (*Y* or *4*) involved in the translocation. This suggests that such suppressions are not the result of the breakpoint, but of a specific entity on the *Y* and chromosome *4* that suppresses exchange. Since the effect is weaker in *T(1;Y)*s than in *T(1;4)*s, and since either arm of the *Y* is large relative to chromosome *4*, it seems reasonable to suggest that the *m–f* exchange suppressions observed in translocations broken just distal to *m* are caused by an effect generated by the telomere. Such a telomere effect has been previously suggested by LEFEVRE (1971).

Evidence for a boundary in region 7: It is necessary to consider the differences among those translocation broken in each interval with respect to the effect they have on recombination within the interval they define. The low level of exchange between *y* and w^a and proximal to *f* precludes any meaningful analysis of the effects on exchange in these regions of translocations broken in those intervals. Moreover, Tables 2, 6 and 7 show that all translocations broken proximal to *m* have similar effects on *m–f* exchange. However, marked dissimilarities in the

amount of exchange suppression occurring between w^a and *m* are observed for those translocations broken in that interval. *T(1;4)8B1* and *T(1;4)9A1* suppress exchange throughout the interval, while *T(1;4)3C6/7*, *T(1;4)4C3* and *T(1;4)5A1* suppress exchange primarily in the distal portion (Tables 2, 3 and 4). For both *T(1;4)3C6/7* and *T(1;4)5A1*, exchange is strongly reduced from *y–ct*, but reaches near normal levels in the *ct–m* interval (Table 4; footnote to Table 2). These data suggest that the medial interval is composed of two intervals with a boundary near *ct* in region 7. Those translocations broken distal to this new boundary allow normal levels of exchange in the interval proximal to the boundary, while those broken proximal to the boundary suppress exchange both distal and proximal to the boundary, because of the centromere effect.

A demonstration and further mapping of this boundary is provided by the behavior of *T(1;Y)*s broken between 4C and 10C (see Table 7). Those *T(1;Y)*s broken at, or distal to, 7A behave as did the *T(1;4)*s broken at 3C6/7 and 5A1, in that the majority of w^a–*m* exchanges occur proximal to the breakpoint. This is clearest in the case of *T(1;Y)7A*, whose breakpoint bisects the w^a–*m* interval. However, a translocation broken nearby, *T(1;Y)7E*, behaves as do the *T(1;4)*s broken at 8B1 and 9A1 in suppressing exchange throughout the interval. Furthermore, as may be seen in Table 8, *T(1;Y)10C* and *T(1;Y)9B*, when heterozygous with a *y* w^a *ct m f* chromosome, suppress exchange only in the *ct–m* interval and not in the w^a–*ct* interval. Conversely, *T(1;Y)5C* suppresses exchange strongly in the w^a–*ct* interval, but allows normal levels of *ct–m* exchange. Thus, the boundary between these two putative intervals must lie near *ct*, between 7A and 7E.

The meiotic behavior of *T(1;Y)9B* and *T(1;Y)10C*, which suppress exchange only in the *ct–m* interval, further suggests that the ability of *T(1;Y)7E*, *T(1;4)8B1* and *T(1;4)9B1* to suppress exchange throughout the w^a–*m* region is a consequence of the centromere effect. Thus, *T(1;Y)*s broken most proximally in the w^a–*m* region prove to be weak exchange suppressors, as would be expected if the ability of proximally broken *T(1;4)*s to suppress exchange throughout the interval were largely a consequence of the centromere effect.

The data described above are presented graphically in Figure 4, which shows the effects of *T(1;4)s* and *T(1;Y)*s that are broken distal to 11A on w^a–*ct* and *ct–m* exchange. It is clear from Figure 4 that not only is the w^a–*m* region com-

TABLE 8

Results of crossing T(1;Y)y·B^S, y^+/y w^a ct m f *females to* y w^a ct m f/Y *males*

Breakpoint	NCO	SCO 1	SCO 2	SCO 3	DCO 1,2	DCO 1,3	DCO 2,3	Total	w^a – *ct*	*ct* – *m*	*m* – *f*
Control	701	290	162	144	4	35	11	1347	24.4	13.1	14.1
5C	649	12	190	273	0	0	9	1133	1.1	17.6	24.9
9B	703	214	44	145	0	19	2	1127	20.7	4.1	14.7
10C	704	233	50	91	1	11	1	1091	22.5	4.8	9.4

Aneuploid female progeny are not included.

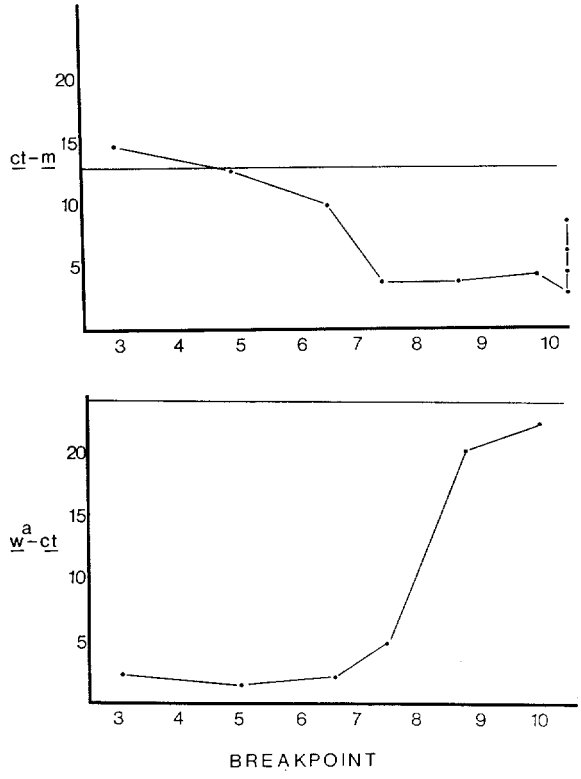

FIGURE 4.—The effects of *T(1;Y)*s and *T(1;4)*s with breakpoints between w^a and *m* on *ct–m* (top panel) and w^a–*ct* (lower panel) exchange. The values plotted for *T(1;Y)7A*, are, in fact, measures of the breakpoint–*m* and w^a–breakpoint intervals, respectively. The values corresponding to the *T(1;Y)*s broken at 11A are computed from data presented in Table 10B.

posed of two intervals with a boundary near *ct*, but that those translocations broken within each interval have similar effects on exchange within that interval. This was also true for those translocations broken proximally to *m*.

Are the exchange suppressions observed merely combinations of the centromere and telomere effect? Both centromere and telomere effects, which result in polar exchange reductions, emanate from the breakpoint of the translocations used in the present analysis. Thus, it might be that the effects observed here are entirely the result of such suppressions, and for that reason translocations broken between *m* and *f* primarily suppress *m–f* exchange and translocations broken in the w^a–*m* interval primarily suppress w^a–*m* exchange. Furthermore, translocations broken near *m* should suppress exchange in both intervals. In fact, the behavior of *T(1;4)11A6/7*, which suppresses exchange from *m–f* and from w^a–*m*, supports exactly such an interpretation.

That such a model is, however, insufficient is indicated by two lines of evidence. First, under such a model, *T(1;Y)*s should be less effective than *T(1;4)*s in suppressing exchange within, as well as outside of, an interval owing to the large

size of the *Y* compared to chromosome *4*. This is clearly not the case. In general, both *T(1;Y)*s and *T(1;4)*s suppress exchange in each interval to approximately the same extent and, where differences occur, no consistent pattern of difference may be discerned.

Second, the exchange behavior of *T(1;Y)*s broken in the w^a–*ct* interval is mimicked by exchange females hyperploid for the distalmost boundary at 3C5. A duplication for the distal element of *T(1;4)3C6/7* carries the euchromatin from *y* to w^a, but carries almost no material from the w^a–*ct* interval. As a control, exchange was also measured in females hyperploid for the distal element of *T(1;4)3C3*. Exchange data from females carrying two normal sequence *X* chromosomes and one of these two duplications are presented in Table 9. In females hyperploid for the distal element of *T(1;4)3C6/7*, exchange is strongly reduced from w^a–*ct*, but is normal or enhanced from *ct* to *m* and from *m* to *f*. The effect of the duplication is very similar to that of *T(1;Y)5C*, but in the case of the dupliation, these effects cannot be due to the centromere effect or the telomere effect. Nor can this suppression be ascribed to the simple model of "competitive pairing" proposed by DOBZHANSKY (1931), as the duplication does not carry any significant fraction of the material in the w^a–*ct* interval. That this suppression is due to hyperploidy for the constriction at 3C5, and not to the hyperploidy of more distal material, is demonstrated by the failure of the distal piece of *T(1;4)3C3* to suppress exchange when carried as a duplication.

It seems reasonable to ascribe the reduction in exchange observed in females hyperploid for the constriction at 3C5 to interference in the pairing of the distalmost boundary or site at 3C5 and thus of the interval of 3C5 to the next boundary in region 7. While a demonstration of this inference must await the analysis of more duplications, it may, at this stage, be noted that at least one other change in the structure or amount of the *X* chromosome causes exchange reductions

TABLE 9

The effect of free duplications of the distal region of the X *chromosome on exchange between two normal sequence* X *chromosomes*

Genotype of females	NCO*	SCO 1	SCO 2	SCO 3	DCO 1,2	DCO 1,3	DCO 2,3	Total	Map lengths w^a – *ct*	Map lengths *ct* – *m*	Map lengths *m* – *f*
y/*y* w^a *ct m f*	701	290	162	144	4	11	35	1347	24.4	13.1	14.1
y/*y* w^a *ct m f*/ *Dp(1;4)3C3*	651	223	190	201	3	12	34	1314	19.8	15.6	18.8
y/*y* w^a *ct m f*/ *Dp(1;4)3C6/7*†	691	24	143	188	0	4	24	1073	2.6	15.5	20.0
y w^a *ct m f*/ *T(1;Y)5C*	649	12	190	273	0	0	9	1133	1.1	17.6	24.9

Only euploid progeny are included. Data for *T(1;Y)5C* are included for reference.

* The intervals 1, 2 and 3 correspond to the w^a – *ct*, *ct* – *m* and *m* – *f* intervals, respectively.

† Of the 31 such females examined, then with respect to euploid offspring, 19 produced primarily w^a-bearing offspring, nine produced primarily w^a+-bearing offspring, while three produced both classes. In all cases, at least one offspring of each type was produced. This phenomenon is under investigation.

identical to those observed in translocation heterozygotes, and that that reduction is not the result of the centromere or the telomere effect.

These data, however, do not rule out the possibility that those exchange suppressions that occur within an interval are the consequence of some combination of (1) an effect that is bounded by specific chromosomal sites, (2) the centromere effect and (3) the telomere effect. However, as diagrammed in Figure 4, none of the *T(1;Y)*s broken in the w^a–*ct* interval strongly suppress *ct–m* exchange, and those *T(1;Y)*s broken in the *ct–m* interval appear to have little or no effect on w^a–*ct* exchange. Thus, most of these translocations exhibit no observable telomere effect or centromere effect on the adjoining interval. *T(1;Y)7E* is an exception; it shows only five map units of exchange from w^a to the breakpoint, implying the possibility of a centromere effect. Since telomere and centromere effects cross boundaries, it seems unlikely that the strong exchange suppression generated by these translocations within the intervals they define are, in any large part, the result of the centromere or telomere effects.

In summary, it appears that the primary cause of exchange suppression in translocation heterozygotes is not the centromere effect or the telomere effect, but rather a third type of exchange suppression onto which the centromere effect and telomere effect are superimposed to create the observed reductions. This third class of exchange suppression is similar for both *T(1;Y)*s and *T(1;4)*s

Finally, we consider the behavior of *T(1;4)11A6/7*, which suppresses exchange on both sides of 11A. If, as seems reasonable, this translocation is broken near or at the 11A boundary, it might be expected to suppress exchange on both sides of that boundary and, thus, from *ct–f*. The exchange suppression observed in the w^a–*ct* interval would then, as was shown to be the case for *T(1;4)8B1* and *T(1;4)9A1*, be the result of the centromere effect. Unfortunately, *T(1;4)11A6/7* is not available for further study.

It may be noted (Figure 3) that four of the five translocations broken between regions 3C and 7 result in an increase in exchange in the *m–f* interval to above 20 map units. On the other hand, those translocations broken proximal to region 7 show normal or slightly reduced levels of *m–f* exchange. We do not, at present, understand these effects of translocation breakpoints, but it is worth recalling that similar increases have been observed by RHOADES (1968) in a bivalent heterozygous for an insertion in maize; exchange was reduced around the insertion and a corresponding increase was observed elsewhere on the bivalent. It may be, therefore, that there exists a general mechanism that causes compensatory increases.

A precise mapping of the medial boundary: The data to this point demonstrate that the medial boundary lies between the breakpoints of *T(1;Y)10C* and *T(1;Y)-11A(B53)*. The behavior of *T(1;4)11A6/7*, which strongly suppresses exchange in both the *m–f* and w^a–*m* intervals (see Table 4), suggests that its breakpoint is near or at the boundary. To test this possibility, exchange was measured in females heterozygous for the *y* w^a *m f* chromosome and for each of five *T(1;Y)*s having breakpoints in the constriction at 11A. The results are presented in Table 10A.

TABLE 10

Exchange in females heterozygous for a T(1;Y) *broken in 11A and (A) for the* y w^a m f *chromosome, crossed to* y w^a m f/Y *males, or (B) for the* y w^a ct m f *chromosome, crossed to* y w^a ct m f/Y *males*

	Type of exchange*						Map lengths		Fraction of total map lengths	
	NCO	SCO1	SCO2	DCO	Total	Aneuploid ♀♀	$w^a - m$	$m - f$	$w^a - m$	$m - f$
(A)										
T(1;Y)11D	1300	454	27	2	1783	396	25.6	1.6	0.94	0.06
T(1;Y)B53	1034	583	34	10	1661	415	35.7	2.6	0.93	0.07
T(1;Y)D9	1851	660	104	16	2631	453	25.7	4.6	0.85	0.15
T(1;Y)B44	1490	605	108	19	2222	374	28.1	5.7	0.86	0.14
T(1;Y)B87	1786	499	133	5	2423	479	20.8	5.7	0.78	0.22
T(1;Y)B45	1269	332	98	23	1722	180	20.6	7.0	0.75	0.25
T(1;Y)10C	1451	438	181	32	2102	403	22.4	10.1	0.69	0.31

	Type of exchange†								Map lengths		
	NCO	SCO1	SCO2	SCO3	DCO1,2	DCO1,3	DCO2,3	Total	$w^a - ct$	$ct - m$	$m - f$
(B)											
T(1;Y)B53	1131	326	147	51	0	7	0	1662	20.0	8.8	3.5
T(1;Y)B87	703	213	31	74	0	5	2	1028	21.2	3.2	7.9
T(1;Y)B44	1026	391	121	78	0	19	3	1638	25.0	7.6	6.1
T(1;Y)D9	889	204	47	61	0	3	1	1205	17.2	4.0	5.4
Control	701	290	162	144	4	11	35	1347	24.4	13.1	14.1

Data for *T(1;Y)11D* and *T(1;Y)10C* are included for reference.
* Regions 1 and 2 correspond to the $w^a - m$ and $m - f$ intervals, respectively.
† Regions 1, 2 and 3 correspond to the $w^a - ct$, $ct - m$ and $m - f$ regions, respectively. Only euploid progeny are recorded.

T(1;Y)11A(D9) and *T(1;Y)11A(B44)* behave as do other *T(1;Y)*s broken proximal to the medial boundary; thus, their breakpoints may also be proximal to this boundary. On the other hand, the behavior of *T(1;Y)11A(B45)* and *T(1;Y)11A(B87)* is similar to that of *T(1;Y)10C*. This suggests that these two translocations are broken distally to the boundary. Four of these *T(1;Y)*s were also examined by measuring exchange in *T(1;Y)/y w^a ct m f* heterozygotes; the results are presented in Table 10B. It may be seen that *T(;Y)11A(B53)* and *T(1;Y)11A(B87)* have reciprocal effects on *m–f* and *ct–m* exchange; *T(1;Y)-11A(B53)* suppresses exchange primarily in the *m–f* interval, while *T(1;Y)11A-(B87)* suppresses exchange primarily in the *ct–m* interval. These observations confirm the mapping of these breakpoints to opposite sides of boundary. Since both of these *T(;Y)*s are broken within the constriction in 11A, then the boundary likely corresponds to a subregion of the constriction.

T(1;Y)11A(D9) and *T(1;Y)11A(B44)* reduce exchange in both the *ct–m* and *m–f* intervals to intermediate levels. As these *T(1;Y)*s cannot be mapped to either side of the boundary, it seems reasonable to suggest that the boundary has length and that these two translocations are broken within it. The intermediate exchange levels observed on both sides of the constriction may be due to a reduction of the

ability of the boundary to function or to centromere and telomere effects. The data presented do not allow us to determine which of these two hypotheses is correct.

Are boundaries chromosomal sites? Evidence has been presented that the *X* chromosome is divisible into five intervals separated by boundaries in regions 3C4–6/7, 7A–7E, 11A and a point proximal to 18C. The question arises whether these putative boundaries correspond to specific sequences of DNA or, alternately, to some extra-chromosomal feature of the meiotic bivalent (for example, some feature of the synaptonemal complex that is independent of chromosomal sequence). To examine this question, the effect of heterozygosity for a translocation breakpoint was measured in females homozygous for an inversion that inverts a region in which two boundaries have been mapped. The inversion used is *In(1)dl-49*, whose breakpoints are at 4D and 11D. The rationale of this experiment is to determine whether moving a chromosome region to which a boundary has been mapped also moves the boundary itself.

Figure 5 compares the location of the boundaries of a normal sequence chromosome with their predicted locations on the *In(1)dl-49*-bearing chromosome. If *In(1)dl-49* has this new arrangement of boundaries, then a translocation broken between *g* and *f* will suppress exchange in the region from *ct* to *f*. On the other hand, if the boundary in region 11A is not moved by the inversion, then that boundary will lie just distal to *g* allowing normal levels of recombination in the *ct–g* interval. In order to minimize the centromere effect, a translocation broken very proximally was chosen for this experiment. This translocation, *T(1;4)16A1* suppresses exchange throughout the *m–f* interval, but shows no exchange suppression in the w^a–*m* interval when tested against a normal sequence *y* w^a *m f* chromosome (Table 2). A chromosome carrying both this translocation and *In(1)dl-49*, and marked with *y sc v g* and B^s, was isolated from among the progeny of *T(1;4)16A1*, B^s/*In(1)dl-49*, *v sc v g f* females and was then used to construct females of the genotype *T(1;4)16A1*, *In(1)dl-49*, *y sc v g* B^s/*In(1)dl-49*, *ct*. The results of measuring crossing over in these females, as well as in *In(1)dl-49*,

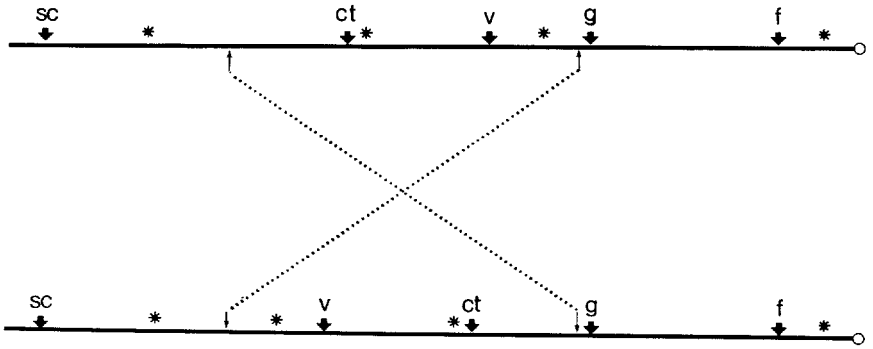

FIGURE 5.—The locations of the four boundaries (asterisks) on a normal sequence *X* chromosome (upper line) and on a chromosome bearing *In(1)dl-49* (lower line). The locations of the breakpoints of the inversion are indicated by the thin arrows.

TABLE 11

Exchange in females homozygous for In(1)dl-49 *and heterozygous for* T(1;4)16A1 *crossed to* In(1)dl-49, y sc v g f/Y *males*

Type of exchange*	*T(1;4)16A1, In(1)dl-49, y sc v g B^{s}/In(1)dl-49, ct*	*In(1)dl-49, y sc v g f/ In(1)dl-49, ct*
NCO	547	437
SCO 1	210	170
2	90	63
3	11	55
4	4	80
DCO 1,2	2	3
1,3	0	8
1,4	4	30
2,3	3	1
2,4	0	0
3,4	2	2
Total	873†§	861†
Map length		
sc – v	24.3	24.5
v – ct	10.9	7.8
ct – g	1.8	7.7
g – B	1.1	13.0

* Regions 1, 2, 3 and 4 correspond to the *sc – v*, *v – ct*, *ct – g* and *g – B* intervals, respectively. In the case of the control cross, the *g – f* interval is considered to be equivalent to the *g – B* interval.

† Only male progeny are included. However, equal map lengths for the *sc – v*, *v – g* and *g – f* intervals are obtained when female progeny are considered (data not shown).

§ 18 hyperploid males were also recovered.

y sc v g f/In(1)dl-49, ct control females, are given in Table 11. Control and experimental females were produced in the same cross [*T(1;4)16A7, In(1)dl-49, y sc v g/In(1)dl-49, y sc v g f* females crossed to *In(1)dl-49, ct/Y* males] and, thus, have similar genetic backgrounds.

As would be expected whether or not the boundaries were moved, exchange is not reduced in the *sc–v* and the *v–ct* regions, but is strongly reduced in the *g–B* region. However, in the critical *ct–g* region, exchange is suppressed by more than four-fold. Thus, it seems reasonable to conclude that the boundaries at regions 11A and 7A-E were moved as a result of the inversion.

It should also be noted that these data also imply that the boundary at region 7A-E, which has been moved more proximally and been inverted with respect to the centromere, does not suffer any loss of function as evidenced by normal exchange in the *cv–v* interval.

The relationship between boundaries and constrictions: Of the four boundaries on the *X* chromosome, two have been mapped precisely to major constrictions (at 3C and 11A) and the other two have been mapped in the vicinity of the constrictions at 7B and 19E. Thus, of the five constrictions on the *X* chromosome, four contain boundaries. There is also a constriction at 12D-E that does not

function as a boundary, as evidenced by the fact that *T(1;Y)*s broken in regions 11 and 12 drastically suppress exchange between *m* and *f*.

No biological function has yet been identified for these constrictions, and considerable evidence suggests that they correspond to regions of intercalary heterochromatin (HANNAH 1951). They are rich in repetitive DNA sequences (RUDKIN and TARTOF 1973), and, because constrictions appear as large targets for X-ray-induced breaks, they likely correspond to large segments of the meiotic chromosomes. Thus, if the distribution of X-ray breaks along a chromosome is proportional to physical length, then, using the data of KAUFMANN (1946), the constriction at 11A represents at least 5% of the euchromatic length of the *X* chromosome. The appearance of such large regions as short constrictions in the polytene chromosome is likely the consequence of under-replication, but might also be the result of very tight packing of the DNA (see LEFEVRE 1976 for discussion).

However, given that the conspicuous constriction at 12D-E does not function as a boundary, it seems unlikely that a boundary is merely the consequence of a large distance between adjacent exchange regions. Furthermore, were boundaries simply large segments of DNA, it is difficult to understand why a duplication for the distalmost boundary should suppress exchange throughout the distal interval (see above). For these reasons it seems that boundaries are more easily thought of as a set of DNA sequences within a constriction.

On the cause of exchange suppression in translocation heterozygotes: Evidence presented here has been interpreted according to the hypothesis that the cause of translocation-induced exchange suppression is chromosomal discontinuity. We may now examine several alternative views. However, it must first be noted that translocations with similar *X*-chromosome breakpoints have similar effects on meiotic exchange, irrespective of the other chromosome involved, implying that exchange suppression is not the result of the particular chromosome involved in the translocation. Thus, a comparison of the fraction of total w^a–*m* interval for *T(1;4)*s and *T(1;Y)*s (Figure 6) reveals that *T(1;4)* and *T(1;Y)* heterozygotes behave in a very similar manner. The dissimilar behavior of *T(1;4)*s and *T(1;Y)*s broken in regions 10 and 11 has been considered above and argued to be the result of the centromere effect. That *T(1;3)*s behave similarly is illustrated by the behavior of *T(1;3)3*, whose *X*-chromosome breakpoint is at 4 D/E, which was studied by DOBZHANSKY (1934), and behaves like *T(1;4)5A1*, *T(1;Y)5C* and *T(1;Y)7A*. Furthermore, although *T(1;3)10A,v/y* w^a *m f* heterozygotes show a low *y*–*f* map length (18 map units), the fraction of *y*–*f* exchange occurring in the *y*–*m* interval is 0.72, which is very similar to the values obtained for *T(1;Y)*s with similar breakpoints (data not shown). Thus, provided that one accounts for the centromere and telomere effects, the effect of an *X*-chromosome translocation on exchange appears to depend only on the location of the *X*-chromosome breakpoint and not on the other chromosome involved in the translocation.

There are three observations that argue that the suppression is not a consequence of interactions between the elements of the translocation. First, the coefficients of coincidence for exchanges proximal to the breakpoint and distal to

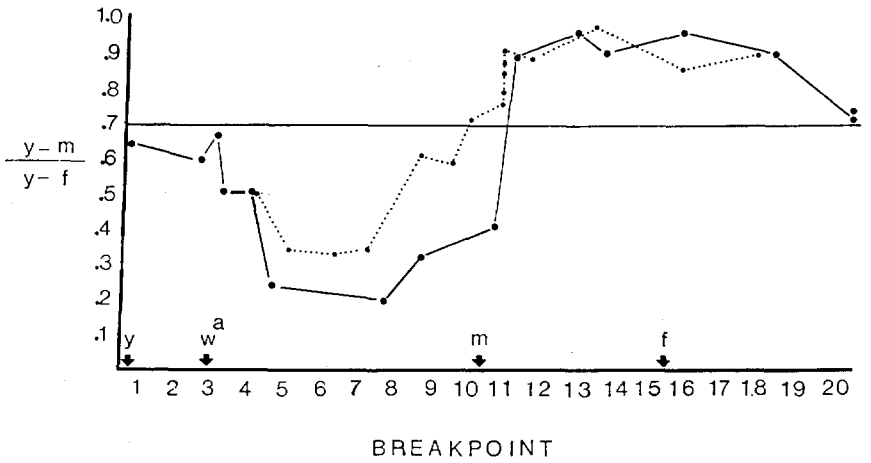

FIGURE 6.—A comparison of the effects of *T(1;Y)*s (.....) and *T(1;4)*s (——) on the fraction of total *y–f* exchange occurring in the *y–m* interval (ordinate) plotted against the breakpoint (abscissa). For the *T(1;Y)*s, the value plotted is actually the fraction of w^a–*f* exchange occurring in the w^a–*m* interval.

the breakpoint of a translocation, considering only those cases where more than ten double exchanges occurred, are 1.14, 0.75, 1.54, 0.53, 0.64, 0.51 and 1.35 for *T(1;Y)7A*, *T(1;Y)9B*, *T(1;Y)10A*, *T(1;Y)11A(B44)*, *T(1;Y)11A(B53)*, *T(1;Y)11A(D9)* and *T(1;Y)11A(B45)*, respectively (Tables 6 and 7). That is, exchange involving one element of a translocation does not influence the likelihood of an exchange occurring in the other element. Thus, we may conclude that whether or not one element of the translocation undergoes pairing and exchange is not greatly influenced by whether or not the other element undergoes pairing and exchange.

Second, one might suggest that the reduction in exchange is due to a tendency of the two elements of the translocation to pair with each other rather than with the normal sequence *X* chromosome. This alternative can be tested by looking at those cases where the two elements of the translocation have gone to opposite poles at meiosis I; that is to say, by looking at the aneuploid female offspring. The various hyperploid progeny obtained from each *T(1;Y)* heterozygote are presented in Table 12. Map lengths derived from hyperploid progeny of *T(1;Y)* heterozygotes are very similar to those obtained using euploid progeny. It can be seen that exchange between one element of the translocation and the normal sequence chromosome is also not affected by whether both elements of the translocation go to the same or opposite poles.

Finally, if the exchange reduction observed in a translocation heterozygote were due to any interaction between the two elements of the translocation, then removal of one element should alleviate the lesion and allow normal levels of exchange. To test this hypothesis, exchange was measured in females deficient for the proximal element of *T(1;*Y^L*)18A* (Table 13). It is clear that exchange in the distal element of the translocation is independent of the presence or

TABLE 12

Aneuploid females resulting from crossing T(1;Y)·B^S,y$^+$/y w^a m f *females to* w^a m f/Y *Males*

								Breakpoint 11A								
Phenotype	4C	5C	7A	7E	9B	10A	10B	B87	B45	B44	D9	B53	11D	12C	13A	18A
Total	353	295	277	160	113	172	403	479	180	374	453	415	396	328	323	460
y m f B^S	189	206	201	115	62	31	62	0	0	0	0	0	0	0	0	107
y m B^S	60	46	54	38	6	11	7	79	0	99	83	0	0	0	0	0
y B^S	100	43	22	5	2	0	0	0	3	1	0	0	0	0	0	0
y w^a f B^S	0	0	0	0	0	0	0	0	1	0	0	0	0	0	0	0
y f B^S	4	0	4	1	1	0	0	0	25	0	0	7	0	0	0	7
y w^a m f B^S	0	0	0	0	0	0	0	0	0	0	0	0	0	0	0	209
w^a	0	0	0	0	29	88	249	4	0	0	6	0	2	0	0	0
w^a m	0	0	0	0	0	0	0	0	100	0	0	271	272	295	210	0
m	0	0	0	0	0	0	0	0	51	0	0	143	122	33	113	0
wild-type	0	0	0	0	14	42	85	2	0	3	3	1	0	0	0	137
y w^a m B^S	0	0	0	0	0	0	0	340	0	225	296	0	0	0	0	0
f	0	0	0	0	0	0	0	50	0	45	62	0	0	0	0	0
w^a f	0	0	0	0	0	0	0	0	0	1	1	0	0	0	0	0
Map length *w^a – m*																
euploid	—	—	—	—	—	24.6	22.4	20.8	20.6	28.1	25.7	35.7	25.6	14.4	29.1	37.6
aneuploid	—	—	—	—	—	32.5	25.4	18.8	33.8	30.5	21.8	34.5	31.3	10.2	35.0	33.1
m – f																
euploid	23.2	24.4	23.3	14.7	16.8	16.9	10.1	5.7	7.0	5.7	4.6	—	—	—	—	—
aneuploid	18.1	15.5	20.9	24.3	9.9	17.0	10.7	10.7	10.3	6.1	12.5	—	—	—	—	—

absence of the proximal element. Similar results were obtained by BEADLE (1933), who demonstrated that exchange in *T(3;4)* heterozygotes was independent of whether the female carried 0, 1 or 2 free fourth chromosomes. This result was confirmed in the case of *T(1;4)*s by SANDLER (1956). In general, therefore, there is no evidence that exchange between any two homologous elements in a translocation heterozygote is affected by presence or absence of other elements of the interchange complex.

In summary, the lesion that causes the observed exchange suppression must be a feature of the *X* chromosome breakpoint. This lesion cannot be a connection of *X*-chromosome material to *Y*-chromosome or autosome material, since *X*-chromosome exchange appears to depend only on the location of the *X*-chromo-

TABLE 13

A comparison of exchange in females heterozygous for T(1;Y)18A *and for the* y w^a m f *chromosome in the presence (top row) and absence (lower row) of the proximal element*

Genotype of female	NCO	SCO 1	SCO 2	SCO 3	DCO 1,2	DCO 1,3	DCO 2,3	Total	Map length *w^a – m*	Map length *m – f*	Map length *f – Bkpt*
T(1;Y)18A/y w^a m f	624	392	29	1	1	0	0	1047	37.5	2.9	<0.1
T(1;Y)18Ad/y w^a m f	584	268	20	0	4	0	0	876	31.1	2.7	0

some breakpoint and not on the other chromosome invoved in the translocation. The most likely candidate for this lesion would appear to be the discontinuity in the *X* chromosome implied by a breakpoint.

The manner by which a discontinuity might cause an exchange suppression in translocation heterozygotes is suggested by the work of BURNHAM (1932) on translocation heterozygotes in maize. He noted that the regions shown genetically to be subject to exchange suppression are frequency asynapsed or involved in nonhomologous pairing. He further observed that the cross formed by the interchange complex had varying arm lengths, as though nonhomologous pairing allowed the center of the cross to slip back and forth along the chromosome. Similar observations have been made by MCCLINTOCK (1933) and by RHOADES (1968), who noted that, in insertion heterozygotes in maize, the "buckle" or insertion loop migrated over the region of the bivalent in which exchange was suppressed.

Based on these observations, the data presented above may be interpreted to mean that the chromosomal sites that function as barriers to exchange suppressions are sites where normal homologous pairing is established or reestablished. It may indeed be that the model presented in Figure 1 is not merely mnemonic, but that the boundaries mapped here are, in fact, "pairing sites." Furthermore, all of these boundaries map to very small regions that contain constrictions. It might be further suggested that one function of constrictions, and thus of intercalary heterochromatin, is the establishment of proper chromosomal associations for meiotic recombination.

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