THE *T6* TRANSLOCATION IN THE MOUSE: ITS USE IN TRISOMY MAPPING, CENTROMERE LOCALIZATION, AND CYTOLOGICAL IDENTIFICATION OF LINKAGE GROUP III.*

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ARSTRACT

The occurrence of hairless piebald mice trisomic for the chromosome segments of the T6M chromosome has shown that the LG III loci *hr* and *s* are not located on T6M. The *T6* breakpoint in LG III is therefore in the position *hr—s—T6*. T6M must carry the gene *Fkl*, which is located on the far side of the *T6* breakpoint from *hr* in LG III.——*T6* reduces recombination in the *hr—s* region.——Trisomy for the chromosome segments of the T6M chromosome appears to severely reduce viability.——The gene *hr* has been shown to lie between the centromere and the *T6* breakpoint. The order of loci in LG III is therefore: centromere—*hr—s—T6*.——Equations are given for the relation between the frequency of adjacent-2 segregation and the frequency of recovery of complementation zygotes for the case in which the translocation heterozygote can form either quadrivalent or univalent-trivalent configurations at meiosis.——Linkage Group III is carried on chromosome 14. LG VI is the other linkage group involved in *T6*, and is carried on chromosome 15.

THE reciprocal translocation *T(3;?)6Ca (T6)* was discovered in an irradiation experiment (CARTER, LYON and PHILLIPS 1955). One of the translocated chromosomes was that bearing Linkage Group III (LG III), as shown by close linkage to piebald (*s*). Subsequent work did not lead to identification of the other linkage group (CARTER, LYON and PHILLIPS 1956).

From a study of first meiotic metaphase chromosomes of males doubly heterozygous for *T6* and *T264 (T(14:17)264Ca)*, SLIZYNSKI (1957) concluded that *T6* and *T264* shared a common chromosome. *T264* is known to be an exchange between the chromosomes bearing LG XVII (CARTER, LYON and PHILLIPS 1955, 1956) and LG XIV (PHILLIPS 1961). The linkage group common to the two translocations therefore was expected to be either XIV or XVII.

This paper is dedicated to the memory of Dr. ALLEN B. GRIFFEN, one of the first cytologists to try to identify mouse pachytene chromosomes carrying specific linkage groups.

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The *T6* translocation is an unequal reciprocal exchange between the largest chromosome that commonly displays a negative heteropycnotic staining region (NHR) just distal to its centromere region, and a medium-sized chromosome (FORD 1966; FORD *et al.* 1956). The result is a small marker chromosome with the NHR, the *T6* marker (hereafter T6M), and another reciprocal product that cannot be identified by conventional staining procedures. However, with the recent application of quinacrine fluorescent staining to mouse chromosomes, the other normal chromosome contributing to *T6* and the reciprocal translocation product were identified (MILLER *et al.* 1971; NESBITT and FRANCKE 1971). The large chromosome with the NHR (numbered 14 by FORD) is number 15, and the other chromosome is number 14 (COMMITTEE 1972).

In this paper we describe: (1) trisomy of the chromosomal region contained in T6M and its use in mapping the breakpoint of *T6* with respect to *s* and hairless (*hr*) in LG III, (2) localization of the centromere in LG III, (3) identification of the chromosome carrying LG III, and (4) attempts to identify the other linkage group involved in *T6*.

MATERIALS AND METHODS

The *T6* translocation was very kindly sent to us by Dr. M. F. LYON, Harwell. It had been crossed for 13 generations into the CBA/H inbred strain and was subsequently made homozygous and maintained by brother–sister matings. In the various crosses reported here it was recognized in one of three different ways:

1. Breeding tests for semisterility: About half the gametes produced by translocation heterozygotes are unbalanced, and, when united with normal gametes, produce zygotes that die *in utero*. We tested mice for semisterility by crossing them to unrelated normal mice, killing the female when 11 or more days pregnant, and counting the live and dead embryos. We classified the mouse as fertile or semisterile in accordance with the criteria of CARTER *et al.* (1955).

2. Cytologically: The T6M chromosome is smaller than any normal chromosome and easily recognized cytologically. Chromosome preparations were made from bone marrow cells using standard air-dried procedures. The presence or absence of T6M and the total number of chromosomes were determined for at least three well-spread metaphase plates from each animal. Meiotic chromosome preparations from male mice were made according to EICHER (1966).

3. Closely linked marker gene: Piebald (*s*) and hairless (*hr*) are both closely linked with the *T6* breakpoint in LG III (see below). *T6* was tagged with either of these loci and linkage with either of them in *T6* heterozygotes was taken to indicate linkage with the *T6* breakpoint.

The notation *T6* is used to indicate either the two reciprocally interchanged chromosomes or the breakpoint in one or both chromosomes. The notation T6M is used to indicate specifically the small marker chromosome (the smaller of the two reciprocally interchanged chromosomes). The symbol + in the notation *T6*/+ or T6M/+/+ indicates the two normally arranged chromosomes from which *T6* was derived.

RESULTS AND DISCUSSION

Trisomy and the position of the T6 breakpoint in Linkage Group III: The position of the *T6* breakpoint in relation to *hr* and *s* was determined by means of a cross between + + *T6*/*hr s* + females and *hr s* +/*hr s* + males. The offspring were classified visually for *hr* and *s* and then for *T6* by analyzing chromosome preparations from bone marrow cells.

The results of the cross are given in Table 1. Of 335 mice, 22 showed a re-

TABLE 1

Offspring of the cross ♀ + + T6/hr s + × ♂ hr s +/hr s +

+ + *T6*	*hr s* +	*hr* + *T6*	+ *s* +	+ + +	*hr s T6*	Total
173	136	14	8	0	4*	335

* All had 41 chromosomes including T6M.

combination between *hr* and *s*. Recombination between *hr* and *s* is significantly lower than normal in *T6* heterozygotes, 6.57 ± 1.35% in comparison with 13.13 ± 0.82% (222/1691) calculated from the data of SNELL (1931), KIDWELL, GOWEN and STADLER (1966), and unpublished data of P. W. LANE, K. P. HUMMEL and G. D. SNELL of The Jackson Laboratory. *T6* therefore probably reduces recombination in regions adjacent to the breakpoint.

Four mice were classified as *hr s T6* and at first sight appeared to be recombinants between *s* and *T6*. However, all of these had 41 chromosomes including T6M and must have been of the genotype *hr s* +/*hr s* +/T6M. Since all of the mice with 41 chromosomes were hairless and piebald, the *hr* and *s* loci (hr^+ and s^+ alleles) cannot be located on the T6M chromosome, assuming that the + +/*hr s*/*hr s* genotype produces a wild-type mouse. This means that the *T6* breakpoint must be outside the *hr*–*s* interval and, since the breakpoint is closer to *s* (0/335 recombinants) than to *hr* (22/335 recombinants), the order in LG III is *hr*—*s*—*T6*.

LYON and GLENISTER (1970) reported that the gene freckled (*Fkl*) is located 18 cM from *s* in LG III, the order being *hr*—*s*—*Fkl*. If our conclusions are correct, the *Fkl* locus is on the other side of the *T6* breakpoint from *hr* and *s* and therefore located on the T6M chromosome.

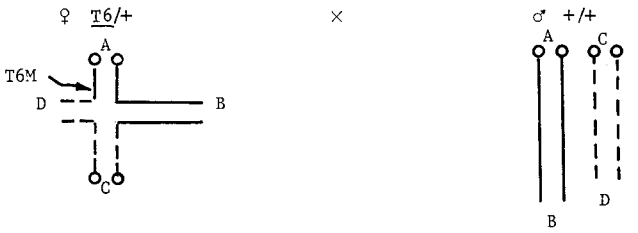
The four *hr s* +/*hr s* +/T6M mice with 41 chromosomes were all runts. It is, possible that the number of such mice born was considerably higher than the number surviving to be fully classified. Since hairless mice (*hr*/*hr*) lose their hair between 2 and 3 weeks of age, all mice were classified for *s* before 2 weeks, marked by toe-clipping, and classified a week or two later for *hr* and *T6*. Twenty of those originally classified as piebald and only one classified as nonpiebald were missing by the time of the second classification. Some of the loss of piebald animals may have been caused by the effects of the extra T6M chromosome, although some of this loss may also have been due to the deleterious effects of *hr* and *s*, particularly *s* which causes megacolon in some homozygotes (BIELSCHOWSKY and SCHOFIELD 1962).

Further evidence on the deleterious effect of the extra T6M chromosome can be obtained by calculating the expected frequency of this condition and comparing it with our observed frequency of 1.2%.

E. P. EVANS (personal communication from C. E. FORD, Harwell) examined meiotic figures from *T6*/+ males and found that 371 out of 999 first metaphases (37%) showed a univalent-trivalent configuration, the univalent being the T6M chromosome. The remaining 628 first metaphases (63%) showed a ring or chain

TABLE 2

Kinds of gametes produced by segregation in quadrivalent and univalent–trivalent configurations in T6/+* *animals and the corresponding outcross offspring*



Pairing in T6/+		Segregation in T6/+	Gametes of T6/+	Offspring	
Type	Freq.			Type	Freq.
Quadrivalent chain or ring	0.63	Alternate	AD.CB	T6/+	0.315
			AB.CD	+/+	0.315
		Adjacent-1	AD.CD	-	0
			AB.CB	-	0
Univalent-trivalent AD and AB.CB.CD	0.37	Alternate	AD.CB	T6/+	0.123
			AB.CD	+/+	0.123
			AB.CD.AD	T6M/+/+	0.123
			CB	-	0
		Adjacent-1	AD.CD	-	0
			AB.CB	-	0
			AB.CB.AD	-	0
			CD	-	0

* No crossing-over is shown since crossing-over gives the same final results. Adjacent-2 segregation does not produce viable offspring and is not included.

quadrivalent. If we allow the following assumptions, we can predict the frequency of the aneuploid 40-plus-T6M chromosome constitution: (1) the frequency of univalent-trivalent formation in *T6/+* females equals that in *T6/+* males, (2) the univalent segregates independently of the trivalent and is not lost during meiosis, (3) a gamete with the normal complement plus T6M functions normally in fertilization, and (4) viability of the 40-plus-T6M mice is normal up to the time of classification.

The possible kinds of gametes produced by univalent-trivalent segregation are

outlined in Table 2. Only alternate segregation is expected to result in viable offspring. The AB.CD.AD gamete has a 20-plus-T6M (T6M/+) constitution, producing a 40-plus-T6M (T6M/+/+) aneuploid offspring, while AB.CD and AD.CB result in +/+ and *T6*/+ offspring, respectively.

Of the expected 63% of meiotic products resulting from segregation in a quadrivalent and 37% resulting from segregation in a univalent–trivalent, 43.8% (31.5 + 12.3) will give rise to *T6*/+ young, 43.8% (31.5 + 12.3) will give rise to +/+ young and 12.3% will give rise to T6M/+/+ (40-plus-T6M) young.

We obtained 55.3% *T6*/+, 43.5% +/+, and 1.2% T6M/+/+. Since the observed frequency of the T6M/+/+ class is significantly lower than expected ($P < .001$), one or more of the original assumptions must be false. We do not know the extent to which assumptions 1 to 3 are true, but number 4, normal viability of the aneuploid condition, is almost certainly false. In our cross, viability was also reduced by homozygosity for *hr* and *s*. Since both +/+ and T6M/+/+ are mostly *hr s*/*hr s*, we can make allowance for this effect and calculate the expected number of T6M/+/+ as (12.3/43.8) × 144 (total number of +/+) = 40.4. The observed number of 4 is about 10% of the expected number.

The 40-plus-T6M condition has also been reported by CATTANACH (1967), who found an overall frequency of 5.8% (7/121). CATTANACH also reported that EVANS and MEREDITH had found 6.5% of 9-day embryos from *T6*/+ × +/+ matings with a 40-plus-T6M chromosome constitution. These values are close to half of the expected frequency of 12.3%, and much larger than our value of 1.2%. Thus it appears that T6M interacts with *hr* and *s* to reduce viability more than expected.

CATTANACH noted that the 40-plus-T6M condition "seemed to be associated with a peculiar head shaking behavior and jerky nervous movement." Our four mice that proved to be 40-plus-T6M were in such poor condition when killed that any such behavior may have passed unnoticed.

Position of the centromere in LG III: SEARLE, FORD and BEECHEY (1971) have devised a method for determining the position of the centromere that makes use of the frequency of particular kinds of viable offspring resulting from union of complementary unbalanced gametes produced by translocation heterozygotes. Using this method we crossed *hr T6*/*hr* + males with + *T6*/++ females and recorded the number of normal and hairless offspring. There were 14 *hr*/*hr* in a total of 285 mice (4.91 ± 1.28%).

If *hr* is on the noncentromere side of the breakpoint, *hr*/*hr* gametes will result from passage of the centromeres of adjacent nonhomologous chromosomes to the same pole at first meiotic metaphase (adjacent-1 segregation). This usually occurs with about the same frequency as their passage to opposite poles (alternate segregation). Under these circumstances SEARLE *et al.* (1971) have shown that union of complementary unbalanced gametes leads to an expected recovery of noncentromeric markers in 1/6 of the offspring or 16.7%.

If *hr* is on the centromere side of the breakpoint, gametes carrying *hr*/*hr* will result from passage of homologous centromeres to the same pole (adjacent-2 segregation); adjacent-2 segregation is rare. SEARLE *et al.* (1971) and SEARLE and

BEECHEY (1971) have reported the recovery of presumed centromeric markers in crosses like the above for several different translocations to be between 1.3 and 4.9%.

The frequency of *hr/hr* offspring in our experiment (4.9%) is considerably lower than 16.7%, and in good agreement with the range of frequencies for centromeric markers reported by SEARLE and co-workers. There is no evidence for inviability of *hr/hr* severe enough to cause such a marked deficiency of a noncentromeric marker. We therefore conclude that *hr* is on the centromere side of the breakpoint and that the order of genes in LG III is: centromere—*hr*—*s*—*T6*.

The frequency of adjacent-2 segregation in a translocation heterozygote influences both the frequency of recovery of centromeric markers and the relative viability of the offspring. CARTER *et al.* (1955) found that the relative viability of offspring from outcrosses of *T6*/+ was 0.37. By use of calculations similar to those of SEARLE *et al* (1971) we can determine whether the frequency of adjacent-2 segregation calculated from our observed frequency of *hr/hr* offspring is in accord with that calculated from the relative viability found by CARTER *et al.* (1955).

The calculations of SEARLE *et al.* (1971) were based on the assumption that translocation heterozygotes will form only quadrivalents at meiosis with chiasmata in either one or both interstitial arms. As noted above EVANS found only 63% quadrivalent and 37% univalent-trivalent formation in *T6*/+ males. We have incorporated this information into the analysis of the relation between the frequency of recovery of centromeric markers and the frequency of adjacent-2 segregation.

Figure 1 shows the frequency of gametes and the viable zygotes resulting from alternate, adjacent-1, and adjacent-2 segregation in all combinations of quadrivalent and univalent-trivalent configurations in matings between *T6* heterozygotes. It is assumed that the AD strand is the T6M univalent. If p is the frequency of adjacent-2 segregation, and q is the frequency of quadrivalents, the following relationships can be deduced from Figure 1:

$$\begin{aligned}
32\,P(A) &= p^2 \cdot (1+q)(2+q) + (1-p)^2 \cdot 2(1-q)^2 \\
32\,P(B) &= p^2 \cdot q(1+q) + (1-p)^2 \cdot 2(1+q^2) \\
32\,P(C) &= p^2 \cdot (4-q+3q^2) \\
32\,P(D) &= p^2 \cdot (2-3q+3q^2) + (1-p)^2 \cdot 2(1+q^2) \\
32\,P(V) &= p^2 \cdot (4-q+3q^2) + (1-p)^2 \cdot (5+2q+5q^2)
\end{aligned}$$

where P(V) is the expected proportion of viable zygotes, and P(A), P(B), P(C), and P(D) are the expected proportions of complementation zygotes homozygous for arms A, B, C, and D, respectively. The proportion of each of the four kinds of complementation zygotes among all viable zygotes is

$$P(E/V) = P(E)/P(V)$$

where E = A, B, C, or D. Since animals with trisomy for the T6M region (T6M/*T6*/+ and T6M/+/+) have severely reduced viability, they have not been included as viable zygotes in the calculations.

EVANS found that 98% of the quadrivalents in *T6* heterozygotes were in the

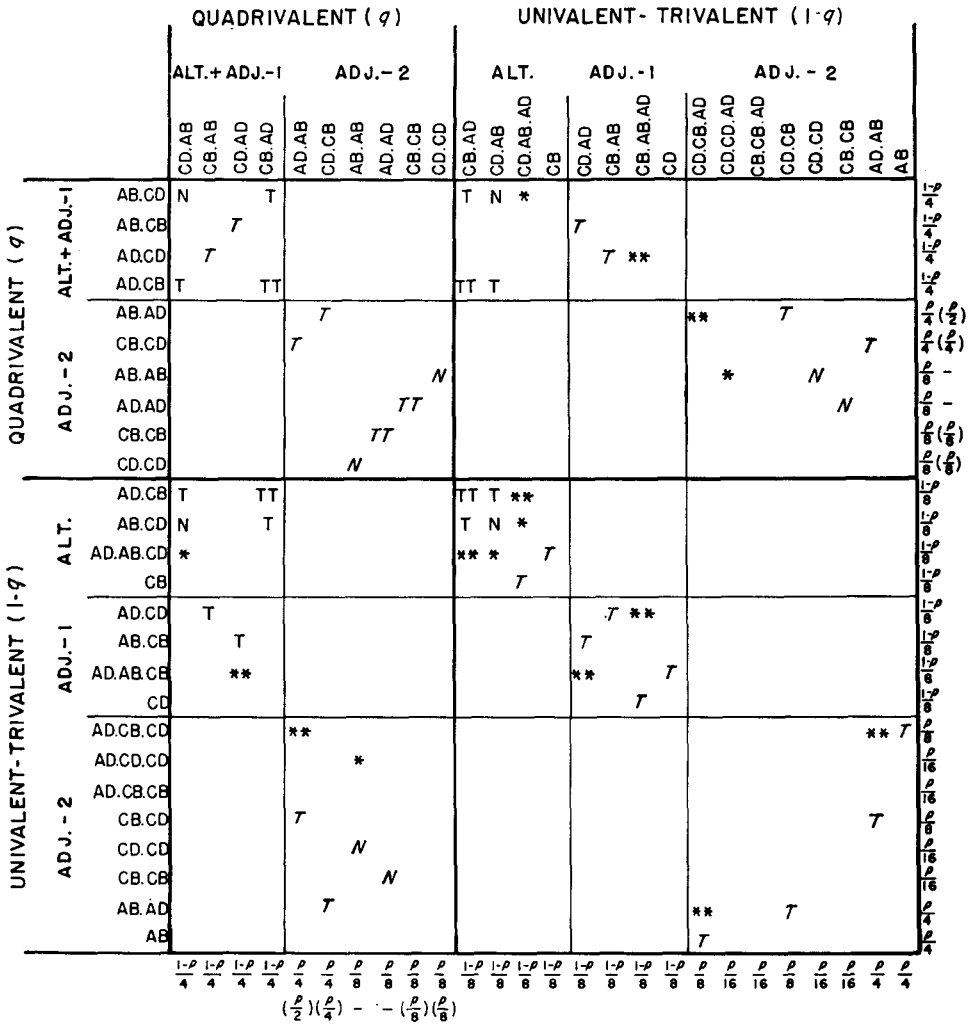


FIGURE 1.—Frequency of gametes and the expected viable zygotes produced by a cross between *T6* heterozygotes. See Table 2 for identification of chromosome arms A, B, C, and D. Unbalanced inviable zygotes are not shown. q is the frequency of quadrivalent formation. p is the frequency of adjacent-2 segregation. The gametic probabilities in parentheses apply to gametes produced when there is no chiasma in A. The symbols N, T and TT represent zygotes that are normal, heterozygous for T6 respectively. These symbols are italicized when the zygote results from the union of two unbalanced gametes. * = T6M/+/+ zyotes. ** = TNM/*T6*/+ zygotes.

form of chains lacking a chiasma in one arm, almost certainly A or D. The above equations apply when all quadrivalents are rings or chains lacking a chiasma in arm D. If the chain results from lack of a chiasma in arm A, the following equations apply:

$$32\,P(C') = p^2 \cdot 4(1+q^2)$$
$$32\,P(V') = p^2 \cdot 4(1+q^2) + (1-p)^2 \cdot (5+2q+5q^2)$$

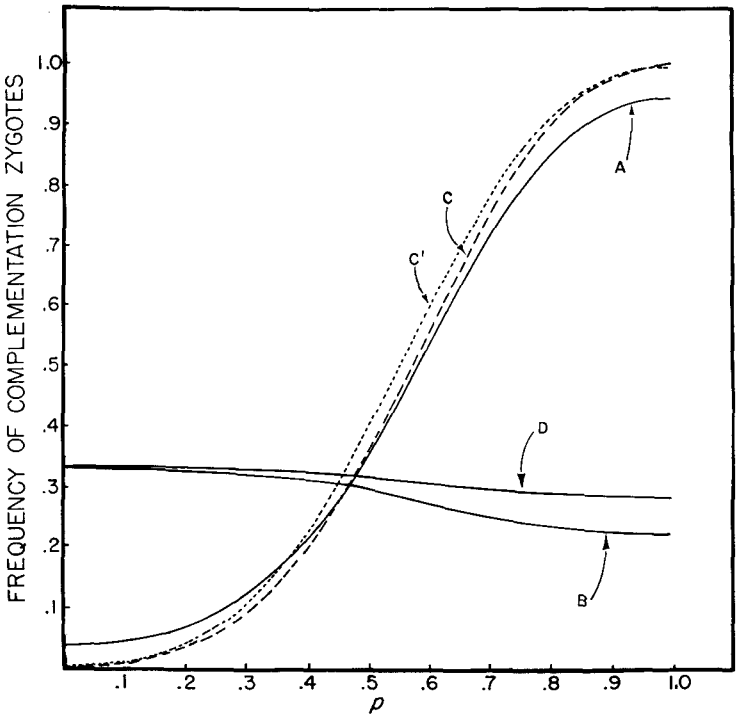


FIGURE 2.—Frequency of complementation zygotes in arms A, B, C, D (see Table 2 for identification of arms) resulting from various frequencies of adjacent-2 segregation (p) in matings between heterozygotes for *T6* (*T6*/+). Quadrivalents and univalent-trivalents are assumed to occur in the proportions 0.63 and 0.37 respectively. All quadrivalents are assumed to be rings or chains of 4 lacking a chiasma in arm D. C′ shows the frequency of homozygotes for arm C, if the chain results from lack of a chiasma in arm A.

The relationship of frequency of complementation zygotes homozygous for each of the four arms and frequency of adjacent-2 segregation is plotted in Figure 2. Since we have shown that the *hr* locus is not on the T6M chromosome (AD) and that it is in the centromeric arm (C) of the other chromosome (CB), one-half the frequency shown for C gives the expected frequency of recovery of *hr*/*hr* offspring (of the complementation zygotes, one-half are *hr*/*hr* and one-half are +/+).

Assuming that the frequencies of quadrivalents (q) is 0.63 and of univalent-trivalents ($1-q$) is 0.37, and that the frequency of adjacent-2 segregation is the same in both, we can use twice the observed frequency of *hr*/*hr* offspring ($0.049 \times 2 = 0.098$) to calculate that the frequency of adjacent-2 segregation (p) is about 0.29. Given the same proportion of quadrivalents (0.63) and univalent-trivalents (0.37), the relative viability on outcrossing of 0.37 found by CARTER *et al.* (1955) corresponds to a value for p of 0.09 (relative viability = $q(1-p)/2 + (1-q)(1-p)/4$, the frequency of balanced gametes, AB.CD and AD.CB, produced by *T6*+). This is lower than the value of 0.29 calculated from the frequency of recovery of *hr*/*hr* offspring. However, SEARLE *et al.* (1971)

found that estimates of p in three different translocations were higher when calculated from the frequency of recovery of centromeric markers (0.13 to 0.20) than from relative viability on outcrossing (0.05 to 0.12). Although these calculations do not lend firm support to the conclusion that *hr* lies between the centromere of LG III and the *T6* breakpoint, they are probably not inconsistent with such a conclusion.

It should be pointed out that the assumption made in our calculations and those of SEARLE *et al.* (1971), that the frequency of adjacent-2 segregation (p) is the same in both ring and chain quadrivalents and in univalent-trivalents, may be false. We do not know whether p is affected by these various conditions.

Twelve of the 14 hairless mice produced from the *hr T6/hr* + × + *T6*/+ + cross were tested for fertility. Three were sterile and the remaining nine were semisterile. Chromosome spreads from bone marrow of one of the sterile and five of the semisterile mice all showed T6M and 39 normal chromosomes, indicating that the mice were *T6*/+ in genotype. If *T6* were a typical translocation with chiasmata in all four arms and with the marker on the centromere side of the break, one-third of the *hr/hr* animals should be *T6/T6* or +/+ and therefore fully fertile (SEARLE *et al.* 1971). When there is no chiasma in arm A or none in both A and D (producing a univalent-trivalent), none of the resulting *hr/hr* offspring should be *T6/T6* or +/+ (see Figure 1). The fact that none of our *hr/hr* mice were found to be fully fertile or to be *T6/T6* or +/+ is in accord with the relatively low frequency of all quadrivalents and particularly of ring quadrivalents in *T6* heterozygotes.

Crosses attempting to identify the second linkage group of T6: Table 3 gives the results of crosses to test for linkage of *T6* with markers in Linkage Groups IV, VII, XIV, XVI, XVII, and XVIII. No linkage was evident.

TABLE 3

Tests for linkage of T6 *or* T6 *tagged with a closely linked marker*
All crosses were double backcrosses, with *T6* and the mutant under test in repulsion

Linkage group	Locus	*T6* tag	Number of recombinants	Number	Percentage of recombination	± S.E.
XIV	*f*	*T6**	63	142	44.4	4.2
XIV	*cr*	*T6**	15	31	48.4	9.0
XIV	*fs*	*s*+	107	194	55.2	3.6
XIV	*bg*	*s*+	105	195	53.8	3.6
XVII	*bf*	*s*+	96	195	49.2	3.6
XVII	*Hm*	*s*+	96	195	49.2	3.6
IV	*Sl*	*hr*	40	65	61.5	6.0
VII	*Re*	*hr*	40	79	50.6	5.6
XVI	*Va*	*hr*	51	79	64.6	5.4
XVIII	E^{so}	*hr*	13	23	56.5	10.3
XVIII	*Os*	*hr*	87	171	50.9	3.8
XVII	W^{v}*(T264)*	*T6*†	11	29‡	37.9	9.0

* *T6* classified by semisterility.
† *T6* classified cytologically.
‡ One ♀ had 41 chromosomes including two T6M chromosomes and is not included in the data.

The absence of linkage of *T6* with markers in LG XIV and LG XVII led us to question SLIZYNSKI's evidence that *T6* and *T264* share a common chromosome, since *T264* shows linkage with both these groups. We therefore crossed *T6/T6* females with *T264* $W^v/+$ + males and analyzed the meiotic chromosomes of three sons showing the W^v marker. W^v (viable dominant spotting) is 4 cM from *T264*. In diakinesis and first metaphase plates two separate translocations were always present. Thus, there was no evidence that *T6* and *T264* shared a common chromosome.

As a further test, we crossed F_1 mice bearing the two translocations to normal mice and classified the offspring visually for W^v as a tag for *T264* and then cytologically for *T6* (Table 3). There was no linkage between *T6* and W^v, confirming the conclusion that *T6* and *T264* do not have a chromosome in common.

An extensive search has failed to find the unknown autosomal linkage group in the translocation *T(X;?)16H* (LYON *et al.* 1964; and LYON personal communication). It seemed possible that the unknown linkage group might be the same as that in *T6*. We therefore crossed a *T16* +/+ *Ta* female to a *T6/T6* male. The F_1 females lacking *Ta*-mosaicism (therefore *T6* + +/+ *T16* +) were crossed to normal *Ta* males (*Ta*/Y) and the offspring classified for *T16* and cytologically for *T6*. (The *T16* +/+ *Ta* females show no *Ta*-mosaicism while the + +/+ *Ta* females do show *Ta*-mosaicism. The *T16* males are sterile and have small testes.) As can be seen from the data presented in Table 4, *T6* and *T16* do not share a common chromosome.

Since the centromere of T6M is derived from chromosome 15, we conclude that the as-yet-unidentified linkage group, not LG III, is carried on chromosome 15. LG III must therefore be on chromosome 14.

There still remains the problem of identification of the other linkage group associated with *T6*. A summary of the tests for linkage with *T6* is given in Table 5. It can be seen that the only linkage groups which could still involve *T6* and therefore chromosome 15 are IV, VI, XV, XVI, and XIX.

Translocation *T(11;?)1Ald (T1Ald)* has been reported by LYON and HAWKES (1969) to have a chromosome in common with *T6*. MILLER *et al.* (1971) have shown that the common chromosome is number 15. When the unknown linkage group in *T1Ald* or *T6* is found, it will be known for both translocations, and will be known to be located on chromosome 15.

TABLE 4

Recombination between T6 *and* T16 *tagged with* Ta. T6 *was classified cytologically*

Parents		Offspring						Percentage of recombination	± S.E.
♀	♂	Sex	*T6*+	+*T16*	*T6 T16*	++	Total		
T6 + + / + *T16 Ta*	× + + *Ta* / Y	♀	8*	1	3	4	16	51.8	9.6
		♂†	1	3(1)	5(3)	2(2)	11‡		

* One female had 41 chromosomes including two T6M chromosomes.

† Parentheses indicate number tested for fertility; otherwise males were classified by testis size. *T16* males are sterile and have small testes.

‡ One sterile male had 41 chromosomes including one T6M. He is not included in the data.

TABLE 5

Summary of autosomal marker genes and translocations shown to be not linked to T6

Linkage group	Chromosomes*	Genes	References†	Translocations‡	References†
I	7	*c^{ch}*	1	*T8*	3
II	9	*d, se*	1	*T138, T163*	3, 6
IV		*Sl*	4	.....	
V	2	*a*	1	*T1§, T2, T5, T7, T83, TSn, T26*	3, 5, 7
VI		*Ca*	1	.....	
VII		*Re, wa-2*	2, 4	*T8*	3
VIII	4	*b*	1	*TSn, T1§*	3, 5
IX	17	*T*	1	*T138, T190*	3
X	10	*v*	2	.....	
XI	6	*Mi^{wh}*	1	*T7, T281, T1Ald*	3, 5
XII	19	*ru*	2	*T163*	6, 8
XIII	1	*fz, ln*	2	*T5, T83, T190*	3
XIV	13	*f, cr, fs, bg, Xt, pe*	4, 5	*T264*	4
XV		*Tw*	5	.....	
XVI		*Va*	4	.....	
XVII	5	*bf, Hm, W^v*	4, 5	*T264*	4
XVIII	8	*E^{so}, Os*	4	*T26*	7
?		*je*	2	.....	
XX	X	sex	1	*T16*	4

* COMMITTEE 1972

† 1. CARTER *et al.* 1955
2. CARTER *et al.* 1956
3. SLIZYNSKI 1957
4. This publication
5. LYON, M. F. Pers. comm.
6. EVANS *et al.* 1967
7. SEARLE, A. G., Pers. comm.
8. EICHER 1971

‡ *TSn* = *T(5;8)Sn*; *T1* = *T(5;8)1Ca*; *T2* = *T(5;?)2Ca*; *T5* = *T(5;13)5Ca*; *T7* = *T(5;11)7Ca*; *T8* = *T(1;7)8Ca*; *T83* = *T(5;13)83Ca*; *T138* = *T(2;9)138Ca*; *T190* = *T(9;13)190Ca*; *T264* = *T(14;17)264Ca*; *T281* = *T(11;?)281Ca*; *T16* = *T(X;?)16H*; *T26* = *T(5;18)26H*; *T163* = *T(2;12)163H*; *T1Ald* = *T(11;?)1Ald*

§ *T1* and *T1Ald* have no chromosome in common. Since *T1Ald* and *T6* share a chromosome, *T6* cannot involve either of the chromosomes of *T1*.

Note added in proof: We have found close linkage between *T6* and underwhite (*uw*) in LGVI. LGVI is therefore carried on chromosome 15, and the centromere is at the *uw* end.

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