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Linkage relationships of markers on chromosome 17 of the house mouse

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SUMMARY

Linkage data for the following markers on chromosome 17 of the house mouse were obtained: centromere (marked by translocation *R67*), *Brachyury* (*T*), *tufted* (*tf*), *H-2*, and *thin fur* (*thf*). The markers were found to be arranged in that order in the genetic map and the combined genetic distances between individual markers were found to be as follows: *Rb7*...*T*, 4·5 cM; *T*...*tf*, 5·8 cM; *tf*...*H-2*, 5·0 cM; *H-2*...*thf*, 15·1 cM. The localization of the *thf* locus on the non-centromeric side of the *H-2* complex provides an important marker for this arm of chromosome 17. The map distances in the centromeric portion of chromosome 17 changed drastically in the presence of various *t* factors. These factors strongly reduce the recombination frequency in the *T* . . . *tf* and *tf*...*H-2* intervals and this crossing-over suppression is most likely responsible for the linkage disequilibrium between *t* and *H-2* reported earlier. Recombinants involving a *t* chromosome but occurring to the right of the *H-2* complex do not change the properties of *t* factors suggesting that all determinants responsible for the t phenotype are located in the chromosomal region between *T* and *tf* (*H-2*).

1. INTRODUCTION

Chromosome 17 is the most intensively studied chromosome of the house mouse, *Mus musculus* L. The main reason for the wide interest in this chromosome is the fact that it carries two complex genetic systems, *T* and *H-2*, involved, respectively, in differentiation and in immune mechanisms. In addition to the *T* and *H-2* complexes, several other loci have been identified on chromosome 17: two loci affecting hair growth, *tufted* (*tf*, cf. Lyon, 1956) and *thin fur* (*thf*, cf. Key & Hollander, 1972); at least one locus affecting myelin formation (*quaking*, *qk*, cf. Sidman, Dickie & Appel, 1964); a host of loci affecting tail structure and possibly related to the *T* complex, namely *Fused* (*Fu*, cf. Reed, 1937), *Kinky* (*Ki*, cf. Caspari & David, 1940) and *Hair-pin tail* (*Hp*, cf. Dickie, Griffin & Frazier, 1965); a locus, *Low* (Dunn & Bennett, 1968), affecting segregation of the chromosome 17; a locus controlling the immune response to alloantigens (*Ir-5*, cf. Zaleski & Klein, 1974); and a group of loci expressed as alloantigens on thymus and leu-

kemia cells (*Tla*, cf. Old, Boyse & Stockert, 1963), on lymphoid cells (*Gv-1*, cf. Stockert, Old & Boyse, 1971), on dermal cells (*H-31*, *H-32*, *H-33*, cf. Flaherty & Wachtel, 1974, and Flaherty, 1975), and on erythrocytes (*Ea-2*, cf. Pizarro & Vergara, 1973). At least three translocations involving chromosome 17 have also been identified: *T(9;17)138Ca*, *T(1;17)190Ca* (Carter, Lyon & Phillips, 1956), and *Rb(16;17)7Bnr* (Gropp, Tettenborn & von Lehmann, 1970).

For investigators interested in the *T* and *H-2* complexes, the other loci on chromosome 17 are valuable for two reasons: they serve as markers in various genetic studies, and at least some of them may prove to be functionally related to either of the two complexes. For this reason, it is important to know the exact linkage relationships of the various loci among themselves and with respect to the *H-2* and *T* complexes. In this communication we report on the linkage information obtained in our laboratory over several years in many different crosses. This information defines with better precision the linkage map of chromosome 17.

2. MATERIALS AND METHODS

(i) *Origin of markers used*

The line carrying the translocation *Rb(16;17)7Bnr*, abbreviated *Rb7*, was derived from a hybrid between *Mus musculus* L. and *M. poschiavinus Fatio*, which was kindly supplied to us by Dr S. Ohno, City of Hope Medical Center, Duarte, California. The progeny from this hybrid was repeatedly back-crossed to an inbred strain (B10.D2) and a congenic line carrying the translocated chromosome was established. The translocation is identifiable as a metacentric chromosome which has arisen by centromeric fusion of telocentric chromosomes 16 and 17. The *Brachyury*, *T*, marker was present either in a congenic line B10-*T* obtained from Dr G. D. Snell, The Jackson Laboratory, Bar Harbor, Maine, or in various non-inbred stocks carrying recessive *t* factors. Breeders of balanced lethal lines with *t* factors (T/t^{w1}, T/t^{w5}, and T/t^{12}) were supplied to one of us by the late Professor L. C. Dunn; the T/t^{6} line was purchased from the Jackson Laboratory. The *tufted*, *tf*, marker was available on various backgrounds in the balanced lethal *t* lines and also in the congenic line B10.G obtained from Dr J. H. Stimpfling, McLaughlin Research Institute, Great Falls, Montana. The *H-2* complex and its various haplotypes were available in various inbred strains and congenic lines maintained in our mouse colony. The stock carrying the *thin fur*, *thf*, marker was kindly provided by Dr W. F. Hollander, Department of Genetics, Iowa State University, Ames, Iowa.

(ii) *Detection of markers*

The *Rb7* translocation was detected as a metacentric chromosome in mitotic metaphase or meiotic metaphase II preparations produced as described previously (Hammerberg & Klein, 1975*a*). The *H-2* haplotypes were determined by serological typing using the PVP haemagglutination technique of Stimpfling (1961) with

modifications described elsewhere (Klein, Klein & Shreffler, 1970). The designation *H-2*^{x} in the various crosses means either a haplotype not yet completely defined or the fact that more than one *H-2* haplotype was used in different experiments. Presence of all other markers was established by visual inspection.

(iii) *Design of experiments*

Most of the segregation data were obtained as byproducts of experiments designed for other purposes. The crossing-over suppression experiments were designed for this specific purpose.

3. RESULTS AND DISCUSSION

(i) *Genetic distances between markers of chromosome 17*

Information about genetic distances between the centromere (represented by the *Rb7* translocation), *T*, *tf*, *H-2* and *thf* was obtained in a series of three-point crosses shown in Tables 1 through 4 and summarized in Table 5. This mapping confirms the previously established order of the four markers (*centromere . . . T . . . tf . . . H-2*, for references cf. Klein, 1975) and in addition establishes, for the first

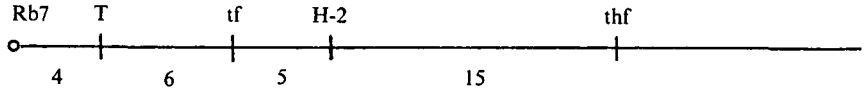

Fig. 1. Genetic map of chromosome 17. Only markers studied in this communication are shown. Numbers indicate combined (for male and females) genetic distances in centi-Morgans.

time, the position of the *thf* locus on the non-centromeric side of *H-2*. The *thf* locus thus finally provides the long sought-after marker for the telomeric end of the *H-2* chromosome. A genetic map based on these results is shown in Fig. 1.

The data in Tables 1–5 are all straightforward and self-explanatory. The segregation of markers was as expected, perhaps with the exception of *thf* which was found to be transmitted to slightly less than one half of the parental type progeny, particularly if the heterozygous parent was a male. This distortion can be explained by incomplete penetrance of the *thf* allele.

(ii) *Crossing-over suppression by* t *factors*

It has been known for some time that normal linkage relationships in chromosome 17 can be distorted by the presence of *t* factors. This suppression was first demonstrated for the interval between *Brachyury* and *Fused* (Dunn & Caspari, 1945), and later also for the interval between *T* and *tf* (Lyon & Phillips, 1959). But the extent of the suppression beyond the *tf* has not been determined. Yet, for the evaluation of the relationship between the *T* and *H-2* complexes, it is particularly

useful to know whether the latter is also in the sphere of the suppressive effect. To determine the extent of crossing-over suppression by various *t* factors, we set up several crosses summarized in Tables 6 and 7, and compared the obtained results with those shown in Tables 1–5. Table 6 shows that in the presence of *t* factors the recombination frequency between *tf* and *H-2* was reduced from its normal value of

Table 1. *Progeny of three-point cross*
TH-2^b + / + H-2^{x}thf × + H-2^{x}thf/ + H-2^{x}thf

Heterozygous parent	*TH-2^b*+	+*H-2^{x}thf*	+*H-2^b*+	*TH-2^{x}thf*	+*H-2^x*+	*TH-2^{b}thf*	+*H-2^{b}thf*	*TH-2^x*+	Total
♀	90	85	18	16	15	11	1	1	237
♂	108	85	12	18	30	11	0	8	272
Total	198	170	30	34	45	22	1	9	509

Recombination (%) ± S.E.

	♀	♂	Total
T . . . H-2	15·2 ± 2·3	14·0 ± 2·1	14·5 ± 1·6
H-2 . . . thf	11·8 ± 2·1	18·4 ± 2·3	15·1 ± 1·6
T . . . thf	26·2 ± 2·9	29·0 ± 2·8	27·7 ± 2·0

Table 2. *Progeny of three-point cross* Rb7 + + / + Ttf ♂ × + + tf/ + + tf ♀

Experiment involving*	*Rb7*+ +	+ *Ttf*	*Rb7Ttf*	+ + +	*Rb7*+*tf*	+*T*+	*Rb7T*+	+ +*tf*	Total
t^6	45	29	1	1	3	2	1	0	82
t^{12}	22	30	0	1	1	0	0	1	55
Total	67	59	1	2	4	2	1	1	137

Recombination (%) ± S.E.

Rb7 . . . T	3·6 ± 1·6
T . . . tf	5·8 ± 2·0
Rb7 . . . tf	8·0 ± 2·3

* *Rb7* + + / + *Ttf* males used in this experiment were obtained from the cross + *Ttf*/*t* + + × *Rb7* + + / *Rb7* + +.

Table 3. *Progeny of three-point cross* Rb7 + H-2^x/ + TH-2^b ♀ × + + H-2^x/ + + H-2^x ♂

Rb7+*H-2^x*	+*TH-2^b*	*Rb7TH-2^b*	+ +*H-2^x*	*Rb7*+*H-2^b*	+*TH-2^x*	*Rb7TH-2^x*	Total
39	35	1	3	4	3	1	86

Recombination (%) ± S.E.

Rb7 . . . T	5·8 ± 2·5
T . . . H-2	9·3 ± 3·1
Rb7 . . . H-2	14·0 ± 3·7

7·6% (in males) to about 1% (t^6, t^{12}) or less (t^{w1}, t^{w5}). In all crosses involving *t* factors, we observed no recombination between *T* and *tf*; all recombinations occurred either in the *tf . . . H-2* interval or to the right of *H-2* (see below). These results indicate that the *H-2* complex is definitely included in the sphere of action

of *t* factors as far as the crossing-over suppressive effect is concerned. Apparently, almost the entire chromosomal arm from the centromere to the *H-2* complex is for all practical purposes excluded from the normal process of recombination. This conclusion is in agreement with the data which we have recently obtained by H-2 typing of *t*-bearing strains (Hammerberg, Klein, Artzt & Bennett, submitted for publication;

Table 4. *Progeny of three-point cross* Rb7 + H-2^x/ + tfH-2^a × + tfH-2^q/ + tfH-2^q

Heterozygous parent	*Rb7* + *H-2*^{x}	+ *tfH-2*^{a}	*Rb7tfH-2*^{a}	+ + *H-2*^{x}	*Rb7* + *H-2*^{a}	+ *tfH-2*^{x}	*Rb7tfH-2*^{x}	+ + *H-2*^{a}	Total
♀	75	78	7	9	2	1	1	0	173
♂	33	25	2	1	1	1	0	0	63
Total	108	103	9	10	3	2	1	0	236

Recombination (%) ± S.E.

	♀	♂	Total
Rb7 . . . *tf*	9·8 ± 2·3	4·8 ± 2·7	8·5 ± 1·8
tf . . . *H-2*	2·3 ± 1·1	3·2 ± 2·2	2·5 ± 1·0
Rb7 . . . *H-2*	11·6 ± 2·4	7·9 ± 3·4	10·6 ± 2·0

Table 5. *Map distances between markers in chromosome 17: summary of Tables 1–4*

(Three values are given for most intervals, ♀♀ (top), ♂♂ (middle), and ♀♀ + ♂♂ (bottom).)

	thf	*H-2*	*tf*	*T*
Rb7	—	12·4	9·8	5·8
	—	7·9	7·0	3·6
	—	11·5	8·3	4·5
T	26·2	13·6	—	
	29·0	14·0	5·8	
	27·7	13·8		
tf	—	3·9		
	—	7·6		
	—	5·0		
H-2	11·8			
	18·4			
	15·1			

Hammerberg & Klein, 1975*b*). The typing revealed a strong linkage disequilibrium between *t* and *H-2*. We found several instances in which *t* factors derived from wild mice captured at widely separated geographical areas (e.g. Denmark *v.* United States) carried similar *H-2* haplotypes. Since *t* factors associated with a particular *H-2* haplotype also belonged to the same complementation group, we suggested that *t* factors represented a certain combination of genes in chromosome 17 which remain genetically interlocked in the mouse population for many generations. We postulated that the mechanism responsible for the interlocking of genes was the crossing-over suppression effect. The finding reported in this communica-

tion that recombination is indeed strongly reduced all the way from the centromere to the *H-2* complex supports this speculation.

Experiments shown in Table 7 were designed to test whether the crossing-over suppression effects extends beyond the *H-2* complex. Unfortunately, the results of these experiments are not easy to interpret because of the variability of recombination frequencies in the *H-2 . . . thf* interval depending on the genetic background. Compared to the average recombination frequency in males (Table 5) the presence

Table 6. *Progeny of three-point cross* TtfH-2^x/t + H-2^t ♂ × + tfH-2^x/ + tfH-2^x ♀

t factor	*TtfH-2^x*	*t + H-2^t*	*TtfH-2^t*	*t + H-2^x*	*T + H-2^t*	*ttfH-2^x*	Total
t^6	21	79	1	0	0	0	101
t^{12}	18	57	1	0	0	0	76
t^{w1}	19	92	0	0	0	0	111
t^{w5}	22	59	0	0	0	0	81

Recombination (%) S.E.

	t^6	t^{12}	t^{w1}	t^{w5}
tf . . . H-2	1·0 ± 1·0	1·3 ± 1·3	0·0	0·0

Table 7. *Recombination between* T, H-2, *and* thf *in the presence of* t

TH-2^{b}thf/tH-2^t + ♂ × + *H-2^{x}thf/* + *H-2^{x}thf* ♀

t factor	*TH-2^{b}thf*	*tH-2^t* +	*TH-2^b* +	*tH-2^{t}thf*	*TH-2^t* +	*tH-2^{b}thf*	*TH-2^{t}thf*	*tH-2^b* +	Total
t^6	32	39	14	8	0	0	1	0	94
t^{w1}	31	109	11	18	1	0	0	2	172
t^{12}	75	90	30	3	1	0	0	0	199
t^{w5}	1	42	1	7	0	0	0	0	51

TH-2^b + / + *H-2^{x}thf* ♂ × + *H-2^{x}thf/* + *H-2^{x}thf* ♀

	TH-2^b +	+ *H-2^{x}thf*	*TH-2^{b}thf*	+ *H-2^x* +	*TH-2^{x}thf*	+ *H-2^b* +	*TH-2^x* +	+ *H-2^{b}thf*	
T	40	27	4	21	6	4	8	0	110

Recombination (%) ± S.E.

	t^6	t^{w1}	t^{12}	t^{w5}	Total
T . . . H-2	1·1 ± 1·1	1·7 ± 1·0	0·5 ± 0·5	0·0	16·4 ± 3·5
H-2 . . . thf	24·5 ± 4·4	18·0 ± 2·9	16·6 ± 2·6	15·7 ± 5·1	30·0 ± 4·4
T . . . thf	24·5 ± 4·4	18·6 ± 3·0	17·1 ± 2·7	15·7 ± 5·1	39·1 ± 4·7

of *t* factors does not reduce (t^6, t^{w1}) or reduces only slightly (t^{12}, t^{w5}) recombination between *H-2* and *thf*. However, compared to a control mating on the same genetic background as the *t* but involving *T* instead, the recombination frequency is reduced almost by one half (Table 7). These differences in recombination frequencies may be due to either variation in penetrance of *thf* in the different crosses, or differences in the strength of crossing-over suppression for various *t* factors. If the *t* factors are arranged according to the strength of their suppression of recombination in the *H-2-thf* interval, one gets the following order: t^6, t^{w1}, t^{12}, and t^{w5}, with t^6 covering the shortest and t^{w5} the longest segment of chromosome 17,

possibly extending beyond the *H-2* complex. This distribution of *t* factors along the chromosome closely parallels the hypothetical arrangement of *t* factors by Lyon & Meredith (1968*b*). However, the plausibility of the above interpretation is somewhat limited by the small sample sizes involved and the possible variability in the penetrance of *thf* in the different crosses.

(iii) *Properties of recombinant* t *chromosomes*

There is every reason to believe that the *t* complex consists of more than one locus and that the various properties of the *t* factors (lethality effect, segregation distortion, tail-enhancing effect on *T*, crossing-over suppression) are controlled

Table 8. *Properties of recombinants derived from the mating* TH-2^{b}thf/tH-2^{t} + ♂ × + H-2^{x}thf/ + H-2^{x}thf ♀

Recombinant derived from	Genotype of recombinant chromosome	Lethality factor present	Tail-modifying factor present	*T-H-2* recombination frequency (and fraction)	Transmission ratio	χ^{2}*
t^{w1}	$t^{w1a}H$-2^{t}*thf*	Yes	Yes	N.T.†	0·93	11.2
t^{w1}	$t^{w1b}H$-2^{t}*thf*	Yes	Yes	N.T.	0·64	1·64
t^{w1}	$T^{c}H$-2^{b}+	No	N.T.	22·2 ± 13·9 (2/9)	N.T.	—
t^{w1}	$t^{w1d}H$-2^{t}*thf*	N.T.	Yes	N.T.	0·86	3·6
t^{6}	$t^{6e}H$-2^{t}*thf*	Yes	Yes	—† (0/51)	0·63	5·58
t^{6}	$t^{6f}H$-2^{t}*thf*	N.T.	N.T.	1·9 ± 1·9 (1/53)	0·49	0·01
t^{6}	$T^{g}H$-2^{b}+	No	N.T.	N.T.	0·52	0·04
t^{12}	$T^{h}H$-2^{b}+	No	N.T.	N.T.	N.T.	—
t^{12}	$T^{i}H$-2^{b}+	No	N.T.	18·2 ± 11·6 (2/11)	N.T.	—
t^{12}	$T^{j}H$-2^{b}+	No	N.T.	11·1 ± 7·4 (2/18)	N.T.	—
t^{w5}	$t^{w5k}H$-2^{t}*thf*	Yes	Yes	N.T.	0·63	0·5
t^{w5}	$T^{l}H$-2^{b}+	No	N.T.	N.T.	N.T.	—
t^{w5}	$t^{w5m}H$-2^{t}*thf*	N.T.	Yes	N.T.	1·0	7·0
t^{w5}	$t^{w5n}H$-2^{t}*thf*	N.T.	Yes	N.T.	0·91	7·32

Superscript small letters, in association with *t* symbols, indicate independent recombinants. Presence or absence of the lethality factor was determined by the ability of the recombinant chromosome to complement the *t* factor from which it was derived. Presence of the tail-modifying factor was determined by the production of tailless offspring from the matings of mice carrying the recombinant chromosome to mice with the *Brachyury* mutation.

* Observed frequency of recombinant chromosome compared to expected frequency (50%).

† N.T.: not tested.

‡ No recombination occurred in the sample tested.

by different regions of chromosome 17. A direct evidence supporting the latter hypothesis was obtained by Lyon & Meredith (1964*a*, *b*, *c*) who recovered t^{6} crossovers in which the various regions of *t* recombined. To determine whether similar segregation of *t* regions occurred in some of the crosses in Tables 6 and 7, the recombinants were further mated to appropriate strains and their properties were analysed. The following properties were tested: the capacity to enhance the tail-shortening effect of *T* (the presence of the tail-enhancing factor), the ability to produce *t*/*t* homozygotes (the presence of the lethality factor), the ability to dis-

tort segregation of chromosome 17, and the capacity to suppress recombination. The actual crosses are not shown, instead, only the conclusions drawn from them are summarized in Table 8. (Only recombinants derived from the crossing over in the *H-2* . . . *thf* interval are described; recombinants from the crossing over in the *tf* . . . *H-2* interval unfortunately failed to produce progeny.) In these experiments, in contrast to those of Lyon and Meredith (1964*a*, *b*, *c*), there was no indication that in any of the recombinant chromosomes, determinants responsible for the various properties of *t* factors were separated by crossing over. In all cases, the *t* region was inherited intact. Since in the experiments of Lyon and Meredith the crossing over occurred in the *T* . . . *tf* interval, whereas in our experiments it occurred in the *tf* . . . *thf* interval (but only *H-2* . . . *thf* crossovers could be tested), one can speculate that all the genetic determinants responsible for the *t* phenotype are located on the centromeric side of *H-2*. Even the determinant responsible for crossing-over suppression must be located in that interval, although its effect may extend beyond the *H-2* complex. The reason why we did not find any recombinants in the *T* . . . *tf* interval was probably the relatively small test sample. (The reported frequency of crossing over between *T* and *tf* in the presence of *t* is approximately 0·2%, cf. Lyon & Phillips, 1959.) At any rate, instances in which the interlocked combinations of determinants constituting the *t* complex are disturbed are extremely rare. Most likely, *t* factors found in natural mouse populations are very old combinations of genes conserved in these populations for a very long time.

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