SHORT PAPER

\textit{t}^M, a new viable \textit{t}-mutant in the mouse

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ABSTRACT

A new \textit{t}-mutant, \textit{t}^M, was isolated from our laboratory mouse stock. The new \textit{t}-haplotype is a recombinant recovered as \textit{t}^M \textit{tf} in \textit{T tf}^{t\textit{v15}+\textit{tf}} \times \textit{T tf}^{t\textit{v16}+\textit{tf}} matings. \textit{t}^M is viable under homozygous conditions. The transmission ratio of \textit{t}^M was calculated to be 0.30. Not all \textit{t}^M/\textit{t}^M males are sterile, although the frequency of sterile males may be higher compared to the wild-type.

1. INTRODUCTION

The \textit{T}/\textit{t} locus of a mouse is a chromosomal region identified by sets of dominant and recessive mutations. Some of these affect embryonic development, sperm production, function and genetic recombination in this chromosome region (for reviews, Bennett 1975; Sherman and Wudl 1977).

Mutations at the \textit{T}/\textit{t} locus may be divided into two classes, dominant and recessive. All dominant mutations cause shortening of the tail under heterozygous conditions (\textit{T}/+\textit{t}) and lethality in homozygotes (\textit{T}/\textit{T}). Recessive mutations produce normal tailed heterozygotes (\textit{t}/+\textit{t}). When combined with \textit{T} (\textit{T}/\textit{t}), however, they yield tailless phenotype. Some of the recessive \textit{t} mutations are lethal in homozygotes (\textit{t}/\textit{t}). These lethal \textit{t}-haplotypes contain a factor responsible for the interaction with \textit{T} to produce taillessness and a recessive lethal factor which maps some 10 cM apart from the tail-interaction factor (Lyon and Meredith 1964).

Recombination in \textit{t}/+\textit{t} heterozygotes produce reciprocal cross-over products quite different from the parental. Recombinants retaining the centromeric end of the parent \textit{t}-haplotype continue to interact with \textit{T} to produce taillessness, but are no longer lethal under homozygous conditions. When the recombinants retain the distal end of the original \textit{t}-haplotype, they are lethal when homozygotes but no longer interact with \textit{T} to produce tailless phenotype (Meredith and Lyon 1964).

In this paper, we report a new \textit{t}-haplotype which was found in our mouse stock (\textit{T tf}/\textit{t}^{\textit{v16}+\textit{tf}} \times \textit{T tf}/\textit{t}^{\textit{v16}+\textit{tf}}) as a recombinant using \textit{tf} as a genetic marker.
2. MATERIALS AND METHODS

*T tf/twl8+tf* mice were originally supplied by Dr. K. Artzt and Dr. D. Bennett of the Sloan-Kettering Institute for Cancer Research. They were kept as a balanced lethal stock in our laboratory. Tufted (*tf*) is a recessive marker; the homozygotes show repeated waves of hair loss and regrowth. This marker lies approximately 8–12 cM on the side of *T* distal to the centromere on chromosome 17. Most of *t*-haplotypes block recombination in the *t-tf* region. Haplotypes belonging to the *t*™-complementation group, however, do not suppress recombination.

Male fertility was tested as follows. One male was caged with 4 fertile females. The male was recorded as fertile if any one of females in its cage became pregnant within one month of observation period.

3. RESULTS AND DISCUSSION

The *t*M (M stands for Mitsubishi-Kasei Institute of Life Sciences) haplotype was isolated in *T tf/twl8+tf* intercrosses as a female tailless mouse with *tf*-phenotype. When mated with a *T tf/+ tf* male, this female produced three types of progenies: 5 normal-tailed, 7 tailless and 4 short-tailed progenies out of a total of 16 offspring. When the female was mated with *T tf/t*M male, 6 tailless and 7 normal-tailed offspring were produced. These results indicate that *t*M originated as a result of a recombination between *T tf-* and *twl8+tf*-haplotypes and that *t*M is not identical to the original *twl8*-haplotype. To date, three viable recombinants *tw29*, *tw60*, and *tw70* have been reported to be originated from *twl8* haplotypes (Dunn, L.C. et al. 1962; Bennett, D. et al. 1976).

When the tailless *F1* were intercrossed, they produced 56 tailless and 11 normal-tailed but no short-tailed offspring, as expected from the viable *t*-haplotype (Table 1).

The transmission ratio of the *t*M-haplotype was calculated by examining the progenies of *+/+ (♀) X T/t*M (♂). The transmission ratio of *t*M was calculated to be 0.30 (Table 2). The transmission ratio of *T*-haplotype is comparable to that of the wild-type. Taking the transmission ratio into account, the frequency of *t*M/*t*M offspring is within the limit of the theoretical value calculated, assuming the homozygotes to be fully viable (*P > 0.10*).

| Table 1. Progenies of intercross matings with *T tf*M |
|---------------------------------|-------------------|
| Parents                        | Phenotype of progeny   |
| Female *T*M                    | Male *T*M            |
| Normal tailed                  | Tailless            |
| 56 (51.6)*                     | 11 (15.4)           |

* The bracketed values correspond to the theoretical expectations on the fully viability of both haplotypes. (The transmission ratio of *t*M was calculated as 0.30.)
The fertility of \( t^M/t^M \) males was tested by mating with fertile females. Four out of six homozygous males were found to be fertile after one month. Six \( T^+/+ \) males were all found to be fertile under similar conditions. All \( t^M/t^M \) females were also found to be fertile.

It is concluded that \( t^M \) retained the tail-interaction factor but lost the lethal factor as a result of recombination.

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REFERENCES


