

standard nomenclature. $M(c^m)$ has no effect on $c^{ch}c^{ch}$.

b. A possible second modifier of c^m which in the homozygote resembles $Mcm-1+$ and which interacts with $Mcm-1$ has been found and tentatively called $Mcm-2$, though allelism with c^m has not been completely excluded. $Mcm-1$ $Mcm-1$ $Mcm-2$ $Mcm-2$ $c^m c^m$ mice are practically black-eyed whites.

c. Another lighter mottled c -allele has also been isolated from the stocks; without ss to concentrate the 2 colours, the mice resemble c^{ec^e} animals, so it is provisionally being called extreme dilution mottled (c^{em}). The possibility of the phenotype being due to yet another modifier, very closely linked to c^m , has not been completely excluded; no crossovers have been found in 35BC and 159 I.C. mice. $c^m c^{em}$ mice are intermediate between $c^m c^m$ and $c^{em} c^{em}$.

d. A dominant gene, which segregates independently of $c^{ch}c^{ch}$ and bb , causes $c^{ch}c^{ch}$ mice to look more brown; it has no effect on $+c^{ch}$ mice. This is provisionally called Mch (modifier of chinchilla). (Phillips)

13. α -glucosidase

Linkage tests have shown that the gene, $Aglp$, determining the electrophoretically detectable variation of α -glucosidase (Peters and Swallow, MNL 60: 46) is on chromosome 17. In the cross (C3H/He x SM/J) F_1 x SM/J no recombinants out of 167 animals tested have been found between $Aglp$ and Apl . Thus these genes are either closely linked or identical. The recombination frequency between $Aglp$ and $Pgk-2$ is $0.9\% \pm 0.9\%$ (one recombinant out of 116 animals).

Incubation of liver homogenates from SM/J with neuraminidase alters the electrophoretic mobility of acid phosphatase and α -glucosidase. By a fluorometric assay method we have found (as Womack and Potier, MNL 61: 64) that liver from SM/J is relatively deficient in neuraminidase compared to C3H/He. The SM/J mouse has 20%, and the (C3H/He x SM/J) F_1 has 60% of the neuraminidase activity of C3H/He. Tests are in progress to find out if a gene on chromosome 17 determines activity levels of neuraminidase. (Peters and D.M. Swallow (MRC Human Biochemical Genetics Unit))

Linkage data

1. Position of Va and ma on Chr. 3

Backcrosses of $Va + ma/+ T(2;3)24H +$ mice to mated homozygotes have produced 14 $Va ma$, 27 $T24H$, 2 $Va T24H$, 4 $T24H ma$, 8 Va , total 55. Thus the order is as shown, with an RF of $3.6 \pm 2.5\%$ between Va and $T24H$ and $21.8 \pm 5.6\%$ between $T24H$ and ma . This result confirms Eicher's assignment of LG XV1 to Chr. 3 (MNL 60: 50) and suggests that $T24H$ has little if any effect on crossing-over round its Chr. 3 breakpoint, since Lane and Eicher (J. Hered. 70: 239) found an RF of $30.2 \pm 2.1\%$ between the Va and ma loci. It also suggests that the Va locus is in or near band 3H1, in which one breakpoint of $T24H$ has been located (Searle et al., Cytogenet. Cell Genet. 23: 255). (Beechey and Searle)

2. Close linkage of T(X;4)37H with spf

Backcrosses of $T37H ++$ spf females to sparse-fur males produced 19 $T37H$, 36 spf, 1 $T37H$ spf; total 56. Thus the $T37H$ breakpoint is very close to the spf locus, with an RF of $1.8 \pm 1.8\%$, agreeing with the G-band location of XA2 (MNL 57: 17). The shortage of $T37H$ offspring was also found in a previous linkage test with Ta (MNL 58: 46) but not with brown and misty (MNL 56: 39). (Beechey and Searle)