

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  $c^{ec}c^{ec}$  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. $\alpha$ -glucosidase

Linkage tests have shown that the gene,  $Aglp$ , determining the electrophoretically detectable variation of  $\alpha$ -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  $\alpha$ -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)