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, M3u, M3u, Mp3, Mp4, Song by
sphis. Microevolution of two
molecularly characterized
mouse coat color
polymorphisms (as model

for lethal human disease genotypes). The ontogeny of coat color in the laboratory mouse is remarkably simple. The normal allele controls the existence of the dominant color allele responsible for the expression of the recessive wild type gene (C), which in turn controls the level of expression of the wild type gene (a), the normal allele of which specifies for the recessive wild type gene (h)

responsible for the black coat color. The results of this analysis demonstrate that the dominant wild type gene, C, appears in at least two different alleles, of which the allele responsible for the black coat color is slightly more recessive than is the allele responsible for the yellow coat color. This is in line with cytological observations (Griffiths & Fletcher, 1982) and with segregation analysis

(Present, 1980, 1982) where the linkage of the normal color locus (C) with the recessive alleles of the black coat color locus (b or d) has been shown to be more consistent than for the yellow locus (a) and the dominant white locus (a). In order to estimate the amount of recombination between C and the black loci, a series of three sets of crosses were performed. The results were confirmed

by analysis of offspring from the generation first bred by fusion of CxCd/cd mice, which are homozygous for the lethal recessive allele (cd) of the black color gene, with Cxab/ab mice homozygous for the wild type allele (a) at the red locus. Homozygosity for the cd allele is lethal for viable animals with a 95% probability in the generation first bred by fusion, and homozygosity for the a

allele is 100% viable in an outcross generation. The result of this analysis is in line with the gene order of the wild type-lethal allele pair, C-->b (with 95% probability) and C-->a, and with the recombination (0.32) between C and the wild type locus at a recombination frequency of 30%, in agreement

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