S3-1

Genetic characterization of the behavioral diversity using a series of wild derived mouse strains

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Many aspects of mouse behavior have been studied using only a relatively small sample of available laboratory strains. An inherent problem in analyzing mouse behavior is that genetic diversity is limited among currently available strains. In this respect, the use of strains that are derived from a variety of wild mice should provide a means to identifying novel behavioral phenotypes. We conducted multi-phenotype behavioral characterizations using a series of mouse strains derived from wild mice of different subspecies, PGN2, BFM/2, NJL, BLG2, HMI, CAST/Ei, CHD, KJR, SWN and MSM, a strain derived from fancy mice JF1, and three laboratory strains C57BL/6, BALB/cAnN and DBA/1. In all the behavioral tests studied, we found a great diversity of the phenotype. In order to identify the loci involved in the behavioral diversity, we are currently conducting the genetic analyses followed by candidate screening to understand the molecular mechanism underlying the behavior. Particularly, we are focusing on two types of behavior, pain sensitivity and spontaneous activity. Recent progress of the genetic analyses on these behavioral phenotypes will be presented.

S3-2

Identification of the New Recessive Cataract Gene (lr2) on Mouse Chromosome 14 by Positional Cloning

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A mouse mutant is an excellent model system in dissecting human disorders. A recessive cataractous mouse, CXSD (D), showed the first clinical symptom of cataract at 5 weeks old and the penetrance was complete. In order to identify the responsible gene, lens rupture 2 (lr2) by positional cloning, we mapped the locus genetically and constructed the physical map spanning the critical region. Using total of 586 intersubspecific F2 mice between cataract D mutant mice and Mus musculus molossinus (MSM), linkage analysis, homozygosity mapping and additive mating experiments revealed the commonly inherited region at D14Mit28 and D14Mit87. The construction of STS-content physical map resulted in isolation of the genomic DNA clones encompassing the critical region and determination of the physical order of 63 STSs that were dispersed among 20 YAC clones and 75 BAC clones. The markers included 11 genetic, 39 clone-end, 13 gene specific EST markers which map to the human chromosome 8p21-22. The sequence analysis of the genomic clones revealed a putative cDNA in the critical region that harbored 27 bp deletion in CXSD mouse genome, indicating this gene was the cataract-causing gene. Understanding the function of the this gene will provide a new insight into cataractogenesis.This work was supported by the grants (M1-0016-00-0040 and 2000-N-NL-01-C-207) from MOST in Korea.