CDB SEMINAR

Date:	**Monday, August 4**
Time:	**16:00 ～ 17:30**
Place:	**7th floor Conference Room, CDB**

<16:00 ～ 16:45>

Speaker: Akihira Otoshi (M.D. , Ph.D.)

< Department of Molecular Genetics,
University of Texas, MD Anderson cancer center >

Title: “Genetic regulation of retinal interneuron differentiation”

Summary

We have isolated mouse Vsx1, a paired-like homeobox gene expressed in cone bipolar cells of the retina. Cone bipolar cells are interneurons that >connect visual pathways between photoreceptors and ganglion cells. MouseVsx1 encodes a homeoprotein that contains a CVC domain that was originally identified as a conserved motif among mouse CHX10, goldfish VSX-1 and C. elegans CEH-10. Linkage analysis showed that mouse Vsx1 mapped to the distal region of chromosome 2. To elucidate the function of VSX1, we generated Vsx1-targeted mice and found that Vsx1 mutant mice lack differentiated cone bipolar cells. Electrophysiological studies demonstrated that Vsx1 mutant mice have an impairment in the cone visual pathway while the rod pathway remains intact. Hence, Vsx1 is required for the differentiation and function of cone bipolar cells in the mouse retina. These studies suggest that VSX1 may regulate color vision in vertebrates.

<16:00 ～ 17:30>

Speaker: Ichiko Nishijima (Ph.D.)

**< Department of Molecular and Human Genetics,
Baylor College of Medicine >**

Title: “Manipulating mouse genome from single gene knockout to chromosome engineering”

Summary

Loss of heterozygosity (LOH) for the short arm of human chromosome 1 (1p32-36) has been observed in a wide variety of cancers including leukemia, breast, lung, intestinal, liver and neuroblastoma. In addition to containing genes critical for tumorigenesis, 1p32-36 also harbors many recessive mutant and haploinsufficient genes related to developmental disorders such as 1p36 deletion syndrome (monosomy 1p; developmental delay, growth abnormalities, craniofacial dysmorphism). To pinpoint the genes at 1p32-36 that are altered in these human diseases, we have generated a series of ES cell lines and mice with large deletions in the distal region of mouse chromosome 4 which is a conserved linkage group with human 1p32-36. We believe the knowledge of these genetic changes will help to understand basic developmental biology and to develop therapeutic strategies.

Host Shin-ichi Nisikawa Stem Cell Biology, CDB

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