CDB SEMINAR

Speaker: Kazuhiro Shimomura

<Center for Functional Genomics Northwestern University>

Title:

"Genetic analysis of circadian behavior in mouse"

Date:	Tuesday, November 9
Time:	15:00 P.M.～ 17:00 P.M.
Place:	1F Auditorium of Building C, CDB

Summary:

Although nine genes have been proposed to be components of core clock mechanism, using complex trait loci analysis we have recently identified at least 13 additional loci, a number of loci affect the circadian behavior through complex epistatic interaction. This suggests not only that there are more clock relevant genes in the mammalian genome, but also that the function of core clock genes may be well conserved among inbred strains of mice. Natural variation of circadian behavior observed in mammals, including humans, may not be the result of rare mutations in core clock genes but rather allelic variants due to ancestral polymorphisms. To identify the loci that can modify a mutant phenotype is a powerful approach to uncover genes that functionally interact with the mutant locus. The fact that the circadian phenotype of *Clock*/+ mice can be significantly suppressed by genetic background provides a unique opportunity to identify loci interacting with *Clock*. Since the protein function of the *Clock* gene product is known, the function of such modifier loci can be more easily delineate. This would be a key advantage of this type of analysis. We have found an allele-specific suppressor of circadian period in *Clock*/+ mice that is highly associated with presence of a BALBc/J genotype on mouse chromosome 1 (MMU1). This locus has been named *Soc-1* (*Suppressor of Clock-1*) and has been isolated in congenic strains. With SNP based haplotype analysis among multiple inbred strains, we have mapped the *Soc-1* locus to a 1.2 Mb interval on MMU1.

Host: Hiroki Ueda <Systems Biology, CDB>

E-mail hiro@cdb.riken.jp Tel: 078-306-3191 (ext:4211)

RIKEN Center for developmental Biology http://www.cdb.riken.go.jp/