Cell Innovation – OIST Joint Meeting

January 12, 2013 at OIST（**B250 Lecture Hall, Center Building, Level B**）

Program

9:30 **Opening Remarks**

Yoshiyuki SAKAKI (Toyohashi University of Technology)

Chairperson: Kazuho IKEO (National Institute of Genetics)

9:35 **Evolution of Chordates**

Noriyuki SATOH (Marine Genomics Unit, OIST)

More than 530 million years ago, chordates originated from a common ancestor shared with nonchordate deuterostomes by developing a novel type of larva (fish-like or tadpole-like larva). In this presentation, I discuss an evolutionary scenario of deuterostomes and molecular mechanisms involved in the origin of chordates.

10:05 **DNA methylation patterns and genome evolution**

Wei QU (Graduate School of Frontier Science, The University of Tokyo)

We identified an unexpected genome-wide role of the CpG methylation state as a major determinant of proximal natural genetic variation: the SNP rate significantly increased by ~50% if the neighboring CpG sites are methylated in human genome. We reconfirmed this finding and uncovered detailed relations between DNA methylation patterns and genome evolution by analyzing six single-base resolution medaka methylomes.

10:35 **Epigenetic control of genes and transposable elements in plants**

Hidetoshi SAZE (Plant Epigenetics Unit, OIST)

Genomes of higher eukaryotes contain many transposable elements (TEs), which are silenced by repressive epigenetic modifications such as DNA methylation and histone H3 Lysine9 methylation. In contrast, these modifications are generally excluded from actively transcribed genes. In *Arabidopsis*, we discovered novel mechanisms that negatively regulate repressive epigenetic marks in genic regions.

11:05 **Break (10 min.)**

Chairperson: Miho OHSUGI (The University of Tokyo)

11:15 **Cohesin loader is a key factor of transcriptional network**

Katsuhiko SHIRAHIGE (Research Center for Epigenetic Disease, The University of Tokyo)

Cornelia de Lange Syndrome (CdLS) is a dominantly inherited congenital malformation. Mutations in nearly 60% of CdLS patients have been identified in NIPBL, which encodes a regulator of the sister chromatid cohesion complex. Through biochemical and genomic studies, we identified NIPBL/Mau2 complex as a key regulator of transcription. The detail mechanisms will be discussed.

11:45 **Control of Cellular Quiescence**

Mitsuhiro YANAGIDA (G0 Cell Unit, OIST)

The fission yeast *Schizosaccharomyces pombe* is an excellent organism to study the molecular mechanism for the entry into, the maintenance of, and the exit from the cellular quiescence. Two principal quiescent states induced by nutritional starvation will be introduced, and some essential genes required for such quiescence will be described. These genes are conserved in human.

12:15 **Closing Remarks**

Tadashi YAMAMOTO (Cell Signal Unit, OIST)

Organized by Innovative Cell Biology by Innovative Technology (Cell Innovation) and Okinawa Institute of Science and Technology Graduate University (OIST)