

invites to a seminar by

Jung-Shin Lee Ph.D.

Department of Molecular Bioscience, College of Biomedical Sciences, Kangwon National University, Kangwondaehak-gil, Chuncheon-si, Gangwon-do, 24341

Republic of Korea

Transcriptional regulation by methylation of the fourth lysine on histone H3 in yeasts

15th of July 2019 at 12.00 p.m.

Venue: Centre of New Technologies, Banacha 2C, Lecture Hall 0142 (Ground floor)

Host: Prof. Magda Konarska

Abstract:

The methylation of the fourth lysine on histone H3 (H3K4me) is a well-known mark of transcription activation and the ubiquitination of the 123rd lysine of on histone H2B monoubiquitination (H2Bub1) is the prerequisite for H3K4me in Saccharomyces cerevisiae. All three types of H3K4 methylation, including mono, di, and tri-methylation, occur by sole H3K4 methyltransferase, Set1 in S. cerevisiae. We created the strain defective bulk level of H3K4me3 even when H2Bub1 signal is normal without any mutation of proteins. We found that some oxidation-reduction related genes are less-expressed in H3K4me3 defective strain comparing the strains bearing normal level of H3K4me3 by RNA-seq analysis. Also, Candida albicans, which is the most common fungal pathogen in human, has Set1complex as its methyltransferase for the H3K4. It is previously described that Set1 deleted mutant shows attenuated virulence and pathogenesis in C. albicans. However, it is unclear why Set1is important for virulence of C. albicans. In this study, we performed RNA-sequencing of wild-type and Aset1 strain to identify the role of Set1 in C. albicans pathogenesis. In Δset1, the 156 genes are downregulated more than 2-fold. The GO enrichment analysis revealed that the significant number of these genes have oxidoreductase activity. Indeed, the Δset1 strain is more sensitive to hydrogen peroxide or menadione which induces oxidative stress. The survival assay in macrophages indicated that the survival rate of Δset1 in macrophages is less than wild-type strain. These results show that the Set1 is required for the survival in host cells by regulating the expression of genes whose products defend against an oxidative stress.