**“How to Have Good DREAMs in Lung Cancer”**

**Moon Jong Kim**

Department of Life Science/Lee Gil Ya Cancer and Diabetes Institute, Gachon University, Korea

In this seminar, I would like to share my recent research accomplishment on the bypassing mechanism of cell quiescence during lung tumorigenesis discussed below.

: The somatic cells have limited cell division and remain quiescent upon terminal differentiation. Deregulation of this crucial process induces cell quiescence exit and hyperproliferation, which is highly implicated in cancer. In a recent study, I unexpectedly found that PAF (PCLAF/KIAA0101) drives cell quiescence exit to promote lung tumorigenesis by remodeling the DREAM complex, a master coordinator of cell quiescence. PAF is highly expressed in lung adenocarcinoma (LUAD) and is closely associated with the poor prognosis of patients. Intriguingly, Paf knockout markedly suppressed LUAD development in mouse models. PAF depletion induced LUAD cell quiescence and growth arrest. PAF is required for the global expression of cell-cycle genes controlled by the repressive DREAM complex. Mechanistically, PAF inhibits DREAM complex formation by binding to RBBP4, a core DREAM subunit, leading to transactivation of DREAM target genes. Furthermore, pharmacological mimicking of PAF-depleted transcriptomes inhibited LUAD tumor growth. These results unveil one of the fundamental mechanisms of how the cancer cell bypasses cell quiescence during tumorigenesis and suggest that attuning cancer cell quiescence could be a therapeutic intervention for cancer treatment.