Thu., **25 April**, 4:00pm

Jukhyun Bio Auditorium(RM.121)

Korean/English

Regulation of Hippo-YAP/TAZ Pathway in Cancer Biology

Speaker | Hyun Woo Park, Ph.D.

Affiliation | Yonsei University

Host | Prof. Yong-Chul Kim

광주과학기술원 생명과학부
Gwangju Institute of Science and Technology School of Life Sciences

School of Life Sciences
Seminar Series
No. 2019-08

Thursday, **25 April** 2019, 4:00 pm

Jukhyun Bio Auditorium(RM.121)

Speaker
Hyun Woo Park, Ph.D.

Education/Experience

2000.03-2006.08	B.S., Department of Pharmacology, Yonsei University College of Medicine, Seoul
2006.09-2010.08	Ph.D., Department of Biology, Yonsei University, Seoul
2010.09-2011.12	Postdoctoral Fellow, Dept. of Pharmacology, Yonsei University College of Medicine, Seoul
2012.02-2016.07	Postdoctoral Fellow, Dept. of Pharmacology & Moores Cancer Center, University of California San Diego
2016.09-present	Assistant Professor, Dept. of Biochemistry, Yonsei University, Seoul

Abstract

The Hippo-YAP/TAZ Signaling Pathway have emerged as key regulator of organ size and tissue homeostasis, and their dysregulation contributes to human cancer. Here I will present an overview of various upstream regulators of Hippo-YAP/TAZ and TEAD including Mechanotransduction, GPCR ligands, Cell Polarity, and Energy stress as potential targets for anticancer therapies. I will also focus on our recent findings on YAP/TAZ as bona fide effectors of the alternative Wnt signaling pathway. Wnt5a/b and Wnt3a induce YAP/TAZ activation via the alternative Wnt pathway that is independent of Wnt/b-catenin signaling. In addition, we recently identified TAZ expression in leukemia and macrophage cells in several pathologic contexts, which suggest a novel function of Hippo signaling in leukemogenesis and immunity. Unlike YAP/TAZ, the transcription factor TEAD are constantly nuclear. However, our recent findings revealed that specific stress promotes cytoplasmic translocation of TEAD via protein-protein interaction with p38 MAPK. Importantly, inhibition of TEAD by p38-induced cytoplasmic translocation suppresses YAP/TAZ-driven cancer cell growth. Therefore, the Hippo pathway is an attractive target for anticancer drug development.