Thu., **23 May**, 4:00pm

Jukhyun Bio Auditorium(RM.121)

Korean

Chemical Proteomics Using Photoaffinity Probes Reveals New Therapeutic Target for Ocular Neovascularization

Speaker | Seung-Yong Seo

Affiliation | Gachon University

Host | Prof. Sung-Gyoo Park

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

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Speaker
Seung-Yong Seo, Ph.D.

Education/Experience

B.S., Seoul National University, Korea
M.S., Seoul National University, Korea
Ph.D., Seoul National University, Korea

2004-2006	Senior Research Scientist, Research Institute of Pharmaceutical Science, Seoul National University, Korea
2006-2008	Postdoctoral Fellowship, Yale University (PI: Craig M. Crews)
2008-2012	Assistant Professor, College of Pharmacy, Woosuk University
2012-2015	Assistant Professor, College of Pharmacy, Gachon University, Korea
2016-2018	Dean, Department of Pharmacy, College of Pharmacy, Gachon University, Korea
2015-present	Associate Professor, College of Pharmacy, Gachon University, Korea

Abstract

The standard-of-care therapeutics for the treatment of ocular neovascular diseases like wet age-related macular degeneration (AMD) are biologics targeting vascular endothelial growth factor signaling. There are currently no FDA approved small molecules for treating these blinding eye diseases. Therefore, therapeutic agents with novel mechanisms are critical to complement or combine with existing approaches. Here, we identified soluble epoxide hydrolase (sEH), a key enzyme for epoxy fatty acid metabolism, as a target of an antiangiogenic homoisoflavonoid, SH-11037. SH-11037 inhibits sEH in vitro and in vivo and docks to the substrate binding cleft in the sEH hydrolase domain. sEH levels and activity are up-regulated in the eyes of a choroidal neovascularization (CNV) mouse model. sEH is overexpressed in human wet AMD eyes, suggesting that sEH is relevant to neovascularization. Known sEH inhibitors delivered intraocularly suppressed CNV. Thus, by dissecting a bioactive compound's mechanism, we identified a new chemotype for sEH inhibition and characterized sEH as a target for blocking the CNV that underlies wet AMD.