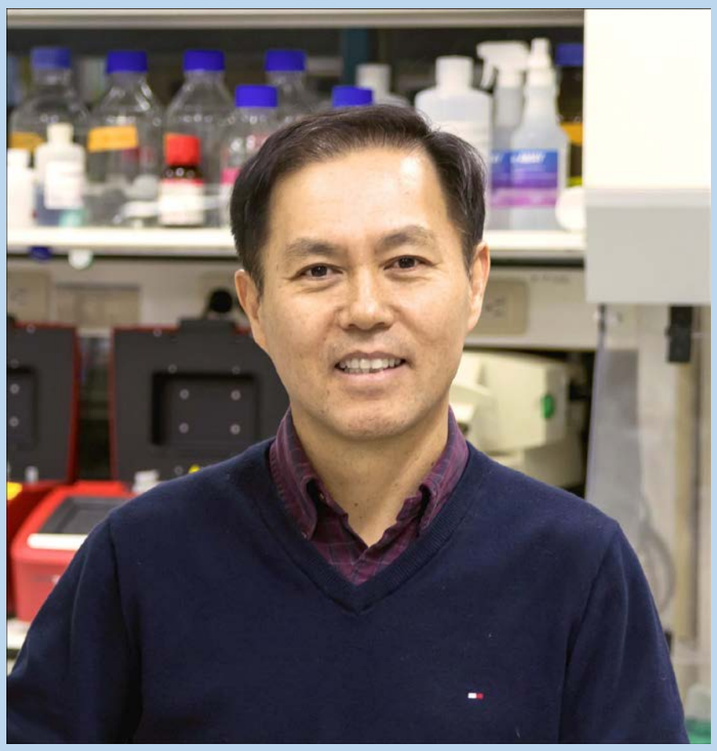
No. 2022-24

School of Life Sciences
Seminar Series

Tuesday
4:00 PM
Oct 18

This seminar will be held face-to-face.

VENUE: S3 Life Sciences Bldg. Jukhyun Bio Hall(#121)



Conventional and unconventional pathways of retinal regeneration

연사 김진우 교수

소속 KAIST

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언어: English

학력

1993 B.S., Department of Life Science, KAIST
1995 M.S., Department of Life Science, KAIST
1999 Ph.D., Department of Biological Sciences, KAIST

경력

1999 Post-doc., Dept. of Biological Sciences, KAIST
1999 - 2002 Post-doc., National Creative Initiatives Research Center for Cell Death, Korea University
2002 - 2006 Research Associate, The Salk Institute for Biological Studies, La Jolla, U.S.A.
2013 - 2014 Visiting Scientist, Friedrich Miescher Institute (FMI), Basel, Switzerland
2009 - 2018 Head of Global Research Laboratory for Neural Fate Programming, KAIST & College de France
2006 - 2018 Assistant and Associate Professor, Department of Biological Sciences, KAIST
2018 - present Professor, Dept. of Biological Sciences, KAIST
2019 - present Director, Laboratory Animal Resource Center, KAIST

Abstract

Retinal neurons are generated during development and degenerate during lifetime. Degenerated cells can be replaced by new cells in many animal tissues that contain stem cells. However, given the absence of residual retinal stem cells, degenerated retinal neurons cannot be regenerated in the retina. However, retinal neurons can be regenerated from Müller glia (MG) in cold-blooded vertebrates upon the injury but not in in mammalian retina, highlighting the incompetency of mammalian MG for retinal regeneration. Previous studies have proposed the factors, which induce mammalian MG to proliferate after the retinal injury. Those include ß-catenin, Yes-associated protein (Yap), and achaete-scute homolog 1 (Ascl1), which however have limited capability to boost MG proliferation. We found that prospero-related homeobox (Prox1) accumulated in MG and suppresses injury-induced MG reprograming and proliferation in the injured mouse retina, but not in the regenerating zebrafish retina. Consistent with this, we succeeded in inducing proliferation of MG in the injured mouse retina by blocking the accumulation of Prox1. Conversely, we could suppress MG proliferation in the injured zebrafish retina by providing Prox1 externally. Collectively, our study suggests that MG proliferation and subsequent retinal neurogenesis in the injured mammalian retina can be recovered by eliminating PROX1 in MG by various therapeutic strategies.