Seminar on

“Targeting transcriptional regulators of ocular angiogenesis in eye diseases”

to be delivered by

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11:30am Feb 9, 2018 (Friday)
Seminar Room 4
G/F, Laboratory Block
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Abstract:
Pathologic blood vessel growth in the eye is a leading cause of blindness in many eye diseases. These include retinopathy of prematurity in children, diabetic retinopathy in adults and age-related macular degeneration in the elderly. Design of new therapies depends on an improved understanding of the disease pathogenesis. In addition to protein- and lipid-based angiogenic factors, our recent work investigated several transcriptional regulators of ocular angiogenesis, including nuclear receptors, transcription factors and non-coding RNAs in experimental models of vascular eye diseases. These factors may be targeted for suppressing pathologic vessel growth. Specifically, we found that: (1) both genetic depletion and pharmacological inhibition of a lipid-sensing nuclear receptor RORα (RAR-related orphan receptor alpha) significantly reduced inflammation-mediated pathologic blood vessel growth in mouse models of ocular angiogenesis; (2) endothelial deletion of a transcription factor Twist1, which is enriched in pathologic retinal vessels, leads to inhibition of pathologic ocular neovascularization via suppression of VEGF receptor; (3) miR-150, a microRNA deficient in pathologically proliferating retinal blood vessels, functions as an intrinsic suppressor of pathologic ocular angiogenesis in vascular eye disease models. Together these findings uncovered multiple transcriptional regulators of ocular angiogenesis, which may be targeted to prevent blindness in children and adults.

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