Yamate Evening Seminar (KU&YU-NIPS Workshop)

March 15th,2018,16:30-17:15

Large meeting room, 2nd floor,Yamate 3rd Bldg.



Dynamic Coordination of Mitochondrial Structure and Function

Prof.Woong Sun (Department of Anatomy, Korea University College of Medicine)

Mitochondria have evolutionarily, functionally and structurally distinct outer— (OMM) and inner—membranes (IMM). Thus, mitochondrial morphology is controlled by independent but coordinated activity of fission and fusion of the OMM and IMM. Although mitochondrial physiology, including oxidative phosphorylation, is important for efficient mitochondrial division, morphological alterations of IMM is less understood. Recently we found that spontaneous and repetitive constriction of mitochondrial inner compartment (CoMIC) which is a priming event for efficient mitochondrial division. We also found that mitochondrial fission machinery controls the mitochondrial membrane potential via direct activation of mitochondrial ion transporter, which is a strategy of cells to coordinate structure and function of mitochondria to control the quality of mitochondria.

Contact: Motohiro Nishida(#5560)

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Chemokine signaling regulates eye morphogenesis in *Xenopus*

Prof. Hosung Jung (Department of Anatomy, Yonsei University College of Medicine)

Collective cell migration is a key mechanism underlying organogenesis, but extracellular signals that regulate this type of cell movement are largely unknown. During vertebrate eye morphogenesis, the continuous lateral migration of the neuroepithelial sheet from the ventral midline places the prospective ventral retina in its proper position. Here we show that the chemokine BRAK controls this process in *Xenopus*. BRAK, an orphan chemokine, is expressed dorsally in the developing retina, and it attracts retinal progenitors from the ventral optic stalk. We show that BRAK antagonizes SDF1 by inhibiting SDF1 effectors, such as MAPK and Wnt/ β -catenin. Thus, the two most evolutionarily conserved chemokines, BRAK and SDF1, regulate neuroepithelial sheet migration by antagonistic actions on shared molecular effectors.

Contact: Motohiro Nishida(#5560)