**THE ROLE OF SEMAPHORIN-3C (SEMA3C) IN INTERPLAY BETWEEN RENAL EPITHELIAL-ENDOTHELIAL CELLS DURING CYST DEVELOPMENT IN POLYCYSTIC KIDNEY DISEASE**

**J. Park**, E. Lee

Sookmyung Women's University, Seoul, Republic of Korea

**Objective:** Autosomal polycystic kidney disease is a one of the common genetic renal diseases in which epithelial-lining cysts appear in kidneys. Numerous cysts arise from abnormally proliferating renal tubular epithelial cells are the evident pathology of ADPKD, accompanied by interstitial inflammation, fibrosis and the destruction of renal tubular architectures. In these regards, current PKD studies have focused on the cyst epithelia, and much less is known about vascular dysfunction during the disease progression. Abnormally dilated vessels around the cysts as well as stiffening of the large elastic arteries in patient with ADPKD were recently reported.

**Method:** Herein, we tried to figure out the significance of intact vasculature and interplay between renal epithelia and endothelia via endothelial factors during PKD progression. Semaphorin-3C, a secreted protein which originally identified as a regulator of axonal branching in nervous system, was abnormally increased in ADPKD mice. Sema3C also has known to have roles in cardiovascular development, and recent studies from another group indicated anti-angiogenic function of Sema3C.

**Results:** Therefore, we identified the Sema3C effects on renal endothelial cells and observed the changes of *in vitro* cystogenesis as well as cysts-promoting signaling pathways including cell proliferation. We have further planned to test whether renal epi-endo cells could affect each other via co-culture system.

**Conclusion:** Our attempts to identify the functional link between renal epi-endo cells mediated by Sema3C during PKD progression could newly suggest the significant renovascular role in cyst development, and finally propose novel biomarkers for either diagnosis or therapy.