

Kidney Research Center(KRC)

Major research goals

Kidney disease is a global public health problem, because it leads to end-stage renal disease in many people and is associated with an increased occurrence of cardiovascular disease, and also is associated with high morbidity and mortality.

To overcome kidney disease, we have established a Kidney Research Center(KRC). Our research center aims to carry out the basic science, translational research and clinical study to provide comprehensive update on the pathomechanism and new therapeutic strategies of kidney diseases.

Major research topics

KRC focuses identify the renal physiology, pathophysiology of acute renal injury or chronic kidney disease, genetic consideration of various renal disease, and discovering the reno-protective materials through basic experiments. Also, KRC is performing the many clinical research including large scale clinical trials about various kidney-associated disease and the development of an artificial kidney.

1. Development the technology of cell regenerative

- therapy for kidney disease.
2. Investigation for the control of oxidative stress in kidney disease.
3. Development the target therapy to alleviate the kidney fibrosis.
4. Examination potential biomarkers of kidney disease using the urinary exosomal micro RNA.
5. Establishment of the purification of dialysate and the development of an artificial kidney.

Major achievements

1. Activation of GPR40 attenuates cisplatin-induced apoptosis by inhibiting the generation of ROS, the activation of the Src/EGFR/ERK signaling pathway and the nuclear activation of NF- κ B and pro-apoptotic factors.
2. Peroxiredoxin 5 Protects TGF- β Induced Fibrosis by Inhibiting Stat3 Activation in Rat Kidney Interstitial Fibroblast Cells.
3. Farnesoid X receptor ligand prevents cisplatin-induced kidney injury by enhancing small heterodimer partner.
4. Angiotensin-(1-7) prevents obstructive nephropathy



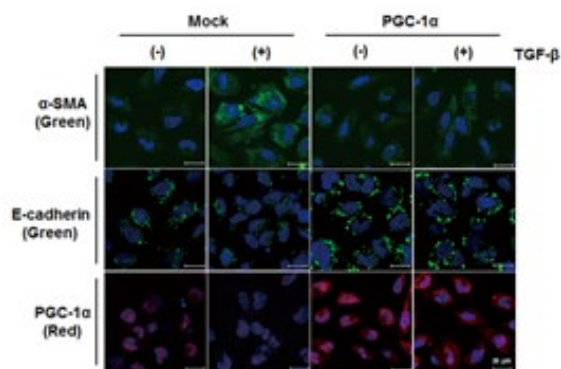
Director
Prof. **Soo Wan Kim**, M.D., Ph.D.

by suppressing renal apoptosis and fibrosis, possibly by regulating TGF- β 1/Smad signaling and cell cycle arrest via suppression of AT1R expression.

5. Our research center has received 19 national research funding of Korea government.

Representative figures of major achievements

Morphological changes of EMT markers in TGF- β treated Mock or PGC-1 α HK-2 cells.



Major relevant publications

1. Choi HI, Ma SK, Bae EH, Lee J, Kim SW. Peroxiredoxin 5 Protects TGF- β Induced Fibrosis by Inhibiting Stat3 Activation in Rat Kidney Interstitial Fibroblast Cells. *PLoS One*. 2016 Feb 12;11(2):e0149266
2. Kim CS, Kim IJ, Bae EH, Ma SK, Lee JU, Kim SW: Angiotensin-(1-7) attenuates kidney injury due to obstructive nephropathy in rats. *PLoS One* 10(11):e0142664, 2015
3. Bae EH, Choi HS, Joo SY, Kim IJ, Kim CS, Choi JS, Ma SK, Lee JU, Kim SW: Farnesoid X receptor ligand prevents cisplatin-induced kidney injury by enhancing small heterodimer partner. *PLoS One* 9(1):e000, 2014
4. Ma SK, Joo SY, Choi HI, Bae EH, Nam KI, Lee JU, Kim SW: Activation of G-protein-coupled receptor 40 attenuates cisplatin-induced apoptosis in the human renal proximal tubular epithelial cells. *Int J Mol Med* 34(4):1117-1123, 2014

5. Park JW, Bae EH, Kim IJ, Ma SK, Choi C, Lee J, Kim SW. Paricalcitol attenuates cyclosporine-induced kidney injury in rats. *Kidney Int*. 2010 Jun;77(12):1076-85.

Research networks

KRC is conducting an MOU with medical school of Aarhus University in Denmark, as well as prosecuting international academic exchanges with Osaka City Medical School, University of Toronto and Vanderbilt University, etc. We closely collaborate with leading research groups in the field of kidney disease.

