Kidney and Vascular Pathogy

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Major Scientific Interests of the Group

Understand the common mechanism of kidney disease progression, particularly cell to cell interaction among glomerular resident cells. Podocyte pathobiology and its response are the main focus to understand segmental nature of glomerulosclerosis.

Projects for Regular Students in Doctoral or Master's Programs

1) Mechanism of podocyte loss.
2) Molecular dynamics and the roles of cytokine/chemokine-receptors determining parietal cell migration.
3) Establishment of one nephrotic two kidney model.

Study Programs for Short Stay Students (one week – one trimester)

1) Renal biopsy diagnosis.
2) Observation of glomerular structures by Low-vacuum scanning electron microscopy.

Selected Publications

1) Podocyte Injury and Its Consequences Kidney Int. 2016, Jun;89(6):1221-30.
2) Podocyte injury-driven intracapillary PAI-1 accelerates podocyte loss via uPAR mediated beta 1 integrin endocytosis. Am J Physiol Renal Physiol. 2015 Mar 15;308(6):F614-26
3) Podocyte injury-driven lipid peroxidation accelerates the infiltration of glomerular foam cells in focal segmental glomerulosclerosis. Am J Pathol. 2015 Aug;185(8):2118-31.
4) Genetic podocyte lineage reveals progressive podocytopenia with parietal cell hyperplasia in a murine model of focal segmental glomerulosclerosis. Am J Pathol. 2009May;174(5):1675-82.
5) Abberant Notchi-1-dependent migration and dedifferentiation of parietal epithelal cells promote collapsing focal segmental glomerulosclerosis with progressive podocyte loss. Kidney Int. 2013; 83,1065-1075.