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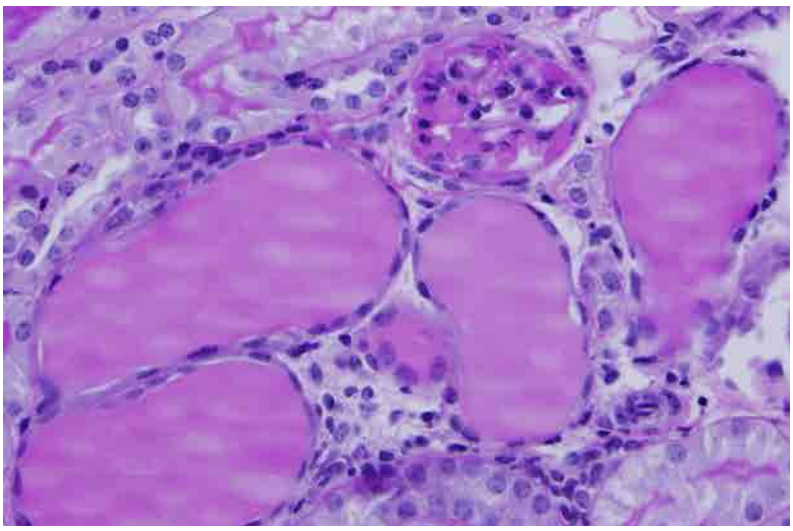
TGF-β1/Smad is the most powerful pro-fibrotic factor discovered to date and operates by activating Smad transcription factors. We recently identified the pivotal role of Smad3/Smad4/CDK9 complex formation in the development and progression of renal fibrosis and the co-administration of Smad3 inhibitor and CDK9 inhibitor achieved better reduction in renal fibrosis compared with administration of single inhibitor in mouse model of renal interstitial fibrosis. This project will utilise our imaging expertise and an array of different models to explore how Smad3/Smad4 complex formation drives organ fibrosis. Smad3/Smad4 complex may be a novel therapeutic target for organ fibrosis.

Research Project:

1. **The role of Smad3/4 complex in driving organ fibrosis**



A severely damaged mouse kidney.

Selected significant publications:

1. Qu X, Jiang M, Sun YB, Jiang X, Dai L, Ren Y, Fu P, Wang D, Caruana G, Bertram JF, Nikolic-Paterson DJ, **Li J**. 2015. The Smad3/Smad4/CDK9 complex promotes renal fibrosis in mice with unilateral ureteral obstruction. *Kidney Int.* [Epub ahead of print].
2. Sun YB, Qu X, Howard V, Dai L, Jiang X, Ren Y, Fu P, Puelles VG, Nikolic-Paterson DJ, Caruana G, Bertram JF, Sleeman MW, **Li J**. 2015. Smad3 deficiency protects mice from obesity-induced podocyte injury that precedes insulin resistance. *Kidney Int.* [Epub ahead of print].
3. Qu X, Zhang X, Yao J, Song J, Nikolic-Paterson DJ, **Li J**. 2012. Resolvins E1 and D1 inhibit interstitial fibrosis in the obstructed kidney via inhibition of local fibroblast proliferation. *J Pathol* 228(4):506-19.
4. **Li J**, Qu X, Yao J, Caruana G, Ricardo SD, Yamamoto Y, Yamamoto H, Bertram JF. 2010. Blockade of endothelial-mesenchymal transition by a Smad3 inhibitor delays the early development of streptozotocin- induced diabetic nephropathy. *Diabetes* 59(10):2612-24.
5. **Li J**, Qu X, Bertram JF. 2009. Endothelial-myofibroblast transition contributes to the early development of diabetic renal interstitial fibrosis in streptozotocin-induced diabetic mice. *Am J Pathol* 175(4):1380-8.