

Targeting Dendritic Cells in Acute Kidney Injury

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Ischemia-reperfusion injury (IRI) is one of the major causes of acute kidney injury (AKI) and evidence supporting the involvement of both innate and adaptive immunity in renal IRI has accumulated in recent years. In addition to leukocytes, kidney endothelial cells promote inflammation after IRI by increasing adhesion molecule expression and vascular permeability. Kidney tubular epithelial cells increase complement binding and upregulate toll-like receptors, both of which lead to cytokine/chemokine production in IRI. Activation of kidney resident dendritic cells appears to play an important critical event in the early immune response to AKI. This presentation will summarize recent studies in my laboratory on the role of dendritic cells in AKI.