Topic	New era for AKI : Kidney Fibrosis after Acute Kidney Injury
Authors	Jung Pyo Lee

Most previous research has primarily focused on assessing in-hospital mortality in patients with acute kidney injury (AKI). Only a few reports have assessed long-term outcomes following discharge. We are unable to predict long-term renal outcomes and the mortality risk of early survivors although this information is highly sought after by investigators. Typically previous episode of AKI not featured amongst risk factors. Recent epidemiological and case-controlled studies indicate that AKI and chronic kidney disease (CKD) are interconnected.

Patients who experienced an AKI episode had a greater chance of experiencing worsening renal function than the patients who had normal kidney function and did not experience an AKI episode. In addition, these patients were shown to have a higher risk of progression to end-stage renal disease (ESRD). "3-month CKD progression" among AKI patients is a risk factor for long-term clinical outcomes such as progression to ESRD and long-term mortality. Renal functional assessment at 3 months following AKI may be a useful measure to predict long-term clinical outcomes.

The pathogenesis of CKD following renal ischemic injury is not fully understood and followings are suggested; (1) a loss of renal vasculature, (2) tubular atrophy or dilatation, and (3) alterations in glomeruli. These changes lead to progressive renal fibrosis and permanent renal functional loss. We hypothesized that periostin was involved in the progression of acute kidney injury to kidney fibrosis. Urine periostin measured at the time of the AKI episode were associated with CKD progression. At 6 wk after unilateral ischemia-reperfusion injury (UIRI), interstitial fibrosis/tubular atrophy was significantly alleviated in periostin-null mice compared with wild-type controls. In addition, periostin-null mice had attenuated expression of fibrosis/apoptosis markers and phosphorylated-p38 MAPK compared with wild-type controls. In vitro, hypoxic injury increased the expression of fibrosis markers, periostin, and phosphorylated-p38 MAPK, which was comparable to or substantially greater than their expression levels following treatment with recombinant transforming growth factor-β1 under normoxic conditions. Furthermore, rPeriostin treatment under hypoxic conditions enhanced fibrosis/apoptosis markers and phosphorylated-p38 MAPK. Periostin promotes kidney fibrosis via the p38 MAPK pathway following acute kidney injury triggered by a hypoxic or ischemic insult. Periostin ablation may protect against chronic kidney disease progression. Protective effects of periostin suppression and correlation with the p38 MAPK pathway.