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Unraveling Cellular and Molecular Changes in AKI-to-CKD Transition Using High-Resolution Spatial Transcriptomics

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Objectives : Acute kidney injury (AKI) is a major risk factor for chronic kidney disease (CKD) , but its spatiotemporal dynamics have not been fully elucidated. This study employs high-resolution single-cell spatial transcriptomics to characterize renal cell population changes during the AKI-to-CKD transition in a unilateral ischemia-reperfusion injury (uIRI) model.
Methods : Mouse kidney samples were collected at six time points post-uIRI (0 hours, 4 hours, 12 hours, 48 hours, 7 days, and 4 weeks), with two samples per time point. Using 10X Genomics' in situ Xenium platform and associated Mouse Tissue Atlassing gene panel, Cell type annotation was performed using known kidney cell type markers overlapping with, and thus detectable by, the Xenium gene panel. Further, the CRAWDAD method was applied to analyze intercellular interactions and identify key cellular transitions contributing to fibrosis and functional decline.
此外，使用 crawdad 方法来分析细胞间相互作用并确定有助于纤维化和功能下降的关键细胞过渡。
Results : The analysis revealed dynamic changes in renal cell composition following uIRI, which progressed through three distinct phases. In the early injury phase (4–12 hours), there was a significant loss of proximal tubular cells, marking the onset of acute kidney damage. Concurrently, there was an upregulation of inflammatory markers, indicating early immune activation. By the intermediate phase (48 hours–7 days), myofibroblast and macrophage populations had expanded, suggesting the initiation of fibrosis and persistent immune activation. During this period, endothelial cell integrity was compromised, and tubular cell loss continued, further impairing renal function. In the chronic phase (4 weeks), fibroblast-like cells became the predominant population, while the number of repair-associated proximal tubular cells was significantly reduced. This shift reflected sustained fibrosis, loss of regenerative capacity, and progressive CKD development.
这种转变反映了持续的纤维化，再生能力的丧失和进行性 CKD 的发展。
Conclusions : This study provides a detailed temporal map of renal cell dynamics during the AKI-to-CKD transition, highlighting key cellular contributors to fibrosis. The findings offer insights into potential biomarkers and therapeutic targets for mitigating CKD progression following AKI.