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The tubular circadian clock modulates post-ischemic immune responses and kidney repair in a context-dependent manner

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Objectives : Circadian clock genes regulate metabolic and inflammatory homeostasis, yet their role in acute kidney injury (AKI) and chronic kidney disease (CKD) remains unclear. We investigated whether tubular BMAL1, a core circadian clock transcription factor, regulates post-ischemic immune–epithelial interactions and recovery following ischemia–reperfusion injury (IRI).
Methods : Bulk RNA sequencing was performed in wild-type (WT) kidneys before and 7 days after IRI to identify differentially expressed genes (DEGs) and their overlap with rhythmic genes. Kidney tubule-specific Bmal1 knockout (KO) mice underwent bilateral IRI, and renal function was assessed at days 1, 7, and 28. Single-cell RNA sequencing at day 7 was performed to define cell type–specific transcriptional changes. Adenine-induced CKD was used to evaluate chronic injury responses.
Results : In WT kidneys, a substantial fraction of IRI-induced DEGs overlapped with rhythmic genes, indicating circadian transcriptional reprogramming after IRI. Bmal1 deletion did not affect peak serum creatinine at day 1, but KO mice demonstrated enhanced functional recovery at day 7 after IRI, whereas renal function was largely comparable between groups by day 28. Single-cell analysis at post-IRI day 7 showed that WT proximal tubules upregulated chemokines (Cxcl1, Cxcl2, Ccl2) and stress-response programs after IRI, whereas these inflammatory signals were attenuated in KO tubules. KO kidneys exhibited reduced IL-6 expression and decreased neutrophil infiltration. Monocyte clusters displayed increased expression of antigen presentation–related genes (Ciita, Cd74), suggesting altered immune programming. Despite increased macrophage numbers, the overall transcriptional profile indicated reduced inflammatory amplification and accelerated resolution. In contrast, tubular Bmal1 deficiency worsened renal function in adenine-induced CKD.
Conclusions : Tubular BMAL1 modulates post-ischemic immune and transcriptional responses in a context-dependent manner. Its deletion alters inflammatory signaling and early repair dynamics after IRI, suggesting that circadian regulation is important for kidney injury and repair, but may differentially influence AKI recovery and CKD progression.