ECRONICON OPEN ACCESS

EC PHARMACOLOGY AND TOXICOLOGY
Editorial

A Short Introduction of Chronic Kidney Disease and Acute Kidney Injury

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Received: October 29, 2021; **Published:** March 28, 2022

Chronic kidney disease (CKD) is characterized by a gradual decline in kidney function lasting months to years, usually caused by long-term diseases such as diabetes or hypertension, and also directly caused by renal dysfunction. Acute kidney injury (AKI) is defined as the sudden cessation of normal work of the kidney, from slight loss of renal function to complete renal failure, which can occur within hours or days, is a rapid decline in kidney function with high morbidity and mortality worldwide. Human epidemiological and experimental animal studies have shown that AKI is not only an acute renal syndrome but also plays an important role in the development and progression of CKD, and AKI can be developed on the basis of CKD. Indeed, AKI and CKD may not be distinct entities but rather are closely interconnected, AKI and CKD can transform and promote each other under certain conditions [1,2].

The pathology of AKI is characterized by the injury and necrosis of renal tubular epithelial cells [3]. After the initial injury, surviving tubular epithelial cells undergo dedifferentiation for proliferation and kidney repair. In this process, normal repair can restore tubular epithelial integrity and function; however, in cases of severe or episodic AKI, incomplete or maladaptive repair usually result in renal interstitial fibrosis of the kidney and the development of CKD [4,5]. Studies from animal experiments have found that ischemia-reperfusion (IRI) was one of the leadings of AKI which usually occurred in clinical situations, and rodent IRI models are commonly used for AKI-CKD transition research [6]. Not only AKI promotes the development of CKD, CKD could predisposes patients to AKI and make the patients more sensitive [7], the survival rate and renal function outcome of patients after AKI are affected by potential CKD, AKI survivors with complete recovery of renal function still have a high risk of new CKD, which may affect long-term survival [8]. Complete repair of the tissue structure is not achieved after normalization of renal function, usually leaving focal fibrosis and inflammatory responses, and these changes show a progressive increase over time. Importantly, these studies provide direct evidences for a causal relationship between AKI and CKD [9].

The cellular and molecular mechanism of AKI-CKD transition is very important for drug development and new treatment strategy of AKI-CKD. Multiple studies had demonstrated that Endoplasmic reticulum (ER) stress played a role in the development of CKD and CKD after AKI. ER stress inhibited further post-ischemic renal tubular epithelial cell apoptosis, inflammation and autophagy, which may represented a potential therapeutic strategy to impede AKI-CKD transition [10] and there is growing evidences showing that epigenetic regulation also plays an important role in AKI and kidney repair, including histone modification, DNA methylation, and gene regulation of various non-coding RNAs. For example, increased levels of histone acetylation appear to protect the kidney from AKI injury and promote kidney repair, and some studies suggested that microRNAs have the potential to become new diagnostic biomarkers for AKI. Further studies of these epigenetic mechanisms will not only yield new insights into the mechanisms of AKI and renal repair, but may also lead to new strategies for the diagnosis and treatment of the disease [11].

Thus, it is essential to understanding the relationship of AKI and CKD and the cellular and molecular mechanism of AKI-CKD transition for prevention and treatment of AKI-CKD.

Conflict of Interest

There is no conflict of interest.

Bibliography

1. Chawla LS., *et al*. "Acute kidney injury and chronic kidney disease as interconnected syndromes". *New England Journal of Medicine* 371.1 (2014): 58-66.

2. He L., *et al*. "AKI on CKD: Heightened injury, suppressed repair, and the underlying mechanisms". *Kidney International* 92.5 (2017): 1071-1083.

3. Linkermann A., *et al*. "Regulated cell death in AKI". *Journal of the American Society of Nephrology* 25.12 (2014): 2689-2701.

4. Zuk A and Bonventre JV. "Acute kidney injury". *Annual Review of Medicine* 67 (2016): 293-307.

5. Venkatachalam MA., *et al*. "Failed tubule recovery, AKI-CKD transition, and kidney disease progression". *Journal of the American Society of Nephrology* 26.8 (2015): 1765-1776.

6. Fu Y., *et al*. "Rodent models of AKI-CKD transition". *American Journal of Physiology-Renal Physiology* 315.4 (2018): F1098-F1106.

7. Matthew T James., *et al*. "A Meta-analysis of the association of estimated GFR, albuminuria, Diabetes Mellitus, and Hypertension with acute kidney injury". *American Journal of Kidney Diseases* 66.4 (2015): 602-612.

8. Pannu Neesh. "Bidirectional relationships between acute kidney injury and chronic kidney disease". *Current Opinion in Nephrology and Hypertension* 22.3 (2013): 351-356.

9. Humphreys BD. "Mechanisms of Renal Fibrosis". *Annual Review of Physiology* 80 (2018): 309-326.

10. Shu S., *et al*. "Endoplasmic reticulum stress is activated in post-ischemic kidneys to promote chronic kidney disease". *EBioMedicine* 37 (2018): 269-280.

11. Guo C., *et al*. "Epigenetic regulation in AKI and kidney repair: mechanisms and therapeutic implications". *Nature Reviews Nephrology* 15.4 (2019): 220-239.

Volume 10 Issue 4 April 2022