Renal fibrosis advances in energy metabolism.

Yue Du*

Department of Nephrology, The First Hospital of Jilin University, China

Introduction

Renal fibrosis is a common precursor to Chronic Kidney Disease (CKD) and the main pathophysiological cause of end-stage renal disease, therefore preventing these conditions is crucial to lowering the morbidity and death that follow from them. There is growing evidence linking metabolic reprogramming to diseases with many aetiology, including disorders caused by anomalies in energy metabolism pathways like glycolysis, the tricarboxylic acid cycle, and lipid metabolism. The degree of fibrosis may be dramatically reduced by cytokine treatments in damaged metabolic pathways, revealing potential therapeutic targets for renal fibrosis medication development. Here, we explore the interaction between glycolysis, lipid metabolism, mitochondrial dysfunction, and renal fibrosis and suggest that the next generation of medicines for renal fibrosis will likely involve targeted therapies for particular metabolic pathways [1].

Multiple different cell types working together in concert are necessary for normal kidney function. These interactions must be precise and sophisticated. This delicate balance requires precise coordination between energy supply and demand since it involves energy-intensive activities, particularly salt transport. When this equilibrium is thrown off by different metabolic, hemodynamics, toxic, or immunological insults, the ensuing disturbance typically takes on a clinical pattern marked by decreased glomerular filtration, thrown off salt and water balance, and loss of endocrine functions. Short-term stresses can be overcome by the kidney, but persistent injury can result in Chronic Kidney Disease (CKD), which affects 8–16% of the world's population and is an irreversible and frequently progressing condition. Additionally, the high expense of renal replacement therapy for people with end-stage kidney failure constitutes a considerable financial burden on both the public and private sectors [2].

The most common pathological feature of CKD, regardless of the underlying aetiology, is the fibrosis that replaces normal structures in the glomeruli and, more significantly from the perspective of prognosis, the tubule-interstitial space. A deeper comprehension of this process may hold the key to advancements in treatment because tubule-interstitial fibrosis represents a significant final common pathway in the development of CKD. Unraveling the processes necessary for the Epithelial-Mesenchyme Transition (EMT), in which functioning epithelial cells change into mesenchymal cells that produce scars, like fibroblasts and myofibroblasts, has been a crucial component of this endeavor. TIF is known to be significantly influenced by inflammation, which occurs when immune cells like macrophages invade the kidney and release cytokines that cause tissue damage [3].

Defective energy metabolism has recently been identified as another factor in the aetiology of renal fibrosis, with studies showing a correlation between chronic kidney disease and poor renal Fatty Acid Oxidation (FAO) and glucose metabolism. Therefore, an important question is how altered renal energy metabolism may be related to the emergence of fibrosis and CKD. A work by colleagues that was published in the Journal of Pathology sheds light on this relationship by showing how the cellular energy sensor AMP-activated protein kinase inhibits the growth of renal fibrosis by having an impact on both EMT and inflammation [4].

The study's novel discovery that the catalytic subunit of AMPK interacts to the -subunit of Casein Kinase 2 (CK2) is an intriguing aspect. Yeast two-hybrid screening was used to find this interaction, and immunoprecipitationk tests later verified it. Although little is known about the pleiotropic kinase casein kinase 2 and its -subunit's function in the kidney, it has been demonstrated that they interact with Liver Kinase B1 (LKB1) to control cell polarity and the epithelial-mesenchymal transition. Because liver kinase B1 directly phosphorylates and activates the crucial T-loop residue of AMPK at Thr172 during energy stress, this connection seems to be significant. Furthermore, liver kinase B1 suppresses tumour growth and is crucial for preserving cellular polarity; some studies also suggest that AMPK participates in this process. However, casein kinase boosted the activity of AMPK by a mechanism requiring increased phosphorylation at Thr172, most likely by liver kinase B1. It is interesting to note that AMPK does not appear to affect casein kinase 2 activities [5].

Conclusion

There are no proven treatments for renal fibrosis, despite the fact that it is a prevalent side effect of many chronic conditions. The survival, reproduction, differentiation, and functionality of cells and organisms depend on the regulation of cellular energy metabolism, and the kidneys use a significant quantity of cellular energy. Strong evidence currently exists that metabolic reprogramming takes place during and contributes to the renal fibrosis process. Drugs that can lessen fibrosis have been identified through studies using animal models and

***Correspondence to:** Yue Du. Department of Nephrology, The First Hospital of Jilin University, China, E-mail: duyeue@jlu.edu.cn

***Received:** 26-Dec-2022, Manuscript No. AACNT-23-85377; **Editor assigned:** 29-Dec-2022, PreQC No. AACNT-23-85377(PQ); **Reviewed:** 12-Jan-2023, QC No AACNT-23-85377; **Revised:** 17-Jan-2023, Manuscript No. AACNT-23-85377(R); **Published:** 24-Jan-2023, DOI:10.35841/aacnt-7.1.131*

***Citation:** Du Y. Renal fibrosis advances in energy metabolism. J Clin Nephrol Ther. 2023;7(1):131*

in vitro research. The procedures and medications known to restore normal metabolic pathways *in vitro* and *in vivo* are plausible candidates for the therapy of fibrosis of the kidneys and other organs in humans, even though none of these medications are currently used for the therapeutic treatment of fibrosis.

References

1. Srivastava SP, Kanasaki K, Goodwin JE. Loss of mitochondrial control impacts renal health. Front Pharmacol. 2020;11:543973.
2. Bikbov B, Purcell CA, Levey AS, et al. Global, regional, and national burden of chronic kidney disease, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet. 2020;395(10225):709-33.
3. Meng XM, Nikolic-Paterson DJ, Lan HY. TGF-β: the master regulator of fibrosis. Nat Rev Nephrol. 2016;12(6):325-38.
4. Huang S, Susztak K. Epithelial plasticity versus EMT in kidney fibrosis. Trends Mol Med. 2016;22(1):4-6.
5. Mount PF, Power DA. Balancing the energy equation for healthy kidneys. J Pathol. 2015;237(4):407-10.

Citation: *Du Y. Renal fibrosis advances in energy metabolism. J Clin Nephrol Ther. 2023;7(1):131*