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Renal Fibrosis: Mechanisms and Therapies

Springer

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Preface

The high prevalence of chronic kidney disease (CKD) is a tremendous global burden. In developed countries, it is estimated that more than 10% of adults present with various degrees of CKD. Irrespective of the initial cause, renal fibrosis is the hallmark of most progressive CKD, which is characterized by the excessive accumulation of fibroblasts and extracellular matrix (ECM). This is accompanied by glomerulosclerosis, tubular atrophy, tubulointerstitial inflammation, and irreversible loss of parenchymal cells. These pathological changes cause progressive deterioration of kidney function, and ultimately lead to end-stage renal disease (ESRD). Unfortunately, around 1–2% of the CKD patients will eventually succumb to the need of renal replacement therapy. Therefore, strategies for slowing or even preventing CKD to ESRD progression are of utmost importance.

In light of this, mechanistic studies of kidney fibrosis have been the focus of intensive research, and it is generally accepted that the critical steps for renal fibrogenesis are as follows: (1) activation of inflammatory response and inflammatory cell infiltration after renal injury; (2) release of profibrotic factors, including cytokines, growth factors, and chemokines; (3) excessive accumulation of fibroblasts and ECM in the interstitial compartment, due to the imbalance in ECM synthesis and degradation; (4) phenotypic change and irreversible loss of parenchymal cells; and (5) reduction in renal microvasculature. Among the above processes, ECM accumulation is the most critical step, as it causes renal scarring that leads to irreversible renal injury both structurally and functionally.

Recently, there are many elegant studies shedding light on the pathogenesis of renal fibrosis, arranging from the role of intrinsic kidney cells to the infiltrating inflammatory cells. New findings also uncover new molecular mechanisms mediating renal fibrogenesis. The development of reliable non-invasive biomarkers has also provided a new potential for diagnosis of CKD, and recent and ongoing advances in basic science research have also provided the necessary platform for new drugs development and novel therapies for renal fibrosis, which may alter the unfortunate fate of CKD patients down the road.

Currently, there are very limited books that systemically reviewed the research progress of renal fibrosis. In view of this, we have invited a group of experts in this field to compile a book that aims to systemically introduce the state-of-the-art research on renal fibrosis. The possible mechanisms, biomarkers, and strategies for prevention and treatment of renal fibrosis are to be elaborated in detail. It is hoped that this book will help readers to have a comprehensive understanding of the renal fibrosis. Finally, we would like to express our gratitude to the financial support of the Key National R&D Project of China Science and Technology Ministry (2018YFC1314000) and the Key International Cooperation Program of National Natural Science Foundation (81720108007).

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