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Cellular and Molecular Basis in Chronic Kidney Disease

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Message from the Guest Editor

Chronic kidney disease (CKD) corresponds to a condition involving an alteration in kidney capacity persisting for three months or more with eventual loss of function over time. CKD can be divided into five specific stages, with factors such as glomerular filtration rate (GFR) and albuminuria serving as criteria for categorization. One of the central pathways involved in the pathogenesis of CKD is the renin–angiotensin–aldosterone system (RAAS). When this pathway is activated, it induces contraction of the glomerular afferent arterioles, resulting in worsening renal ischemia which ultimately diminishes glomerular filtration capacity. Collectively, RAAS, TGF-B1, and other factors such as vascular calcification and uremic toxins are known to be involved in CKD, causing worsening renal inflammation and fibrosis, both of which diminish renal capacity and function. This Special Issue on CKD aims to recruit original papers, reviews, and communication that enhance the understanding of cellular and molecular mechanisms involved in the pathophysiology of CKD.

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Cells has become a solid international scientific journal that is now indexed on SCIE and in other databases. We have successfully introduced a special issues format so that these issues serve as mini-forums in specific areas of cell science. *Cells* encourages researchers to suggest new special issues, serve as special issues editors, and volunteer to be reviewers. Our main focus will remain on cell anatomy and physiology, the structure and function of organelles, cell adhesion and motility, and the regulation of intracellular signaling, growth, differentiation, and aging. We are open to both original research papers and reviews.

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