Special Issue

Redox Dysregulation and Mitochondrial Adaptation in Kidney Disease

Message from the Guest Editor

The kidneys perform important functions and can experience either acute kidney injury (AKI) or chronic kidney disease (CKD). AKI can be induced by renal ischemia-reperfusion injury, by drugs such as cisplatin, and by heavy metals such as cadmium and arsenic. CKD arises from drugs, heavy metals, hypertension, diabetes, and cancer. Importantly, nearly all kidney disorders involve redox imbalance that initially begins with NADH reductive stress and then transitions to oxidative stress. This pathological process usually involves mitochondrial adaptation and mitochondrial abnormalities such as mitochondrial stress response, impaired mitochondrial homeostasis, and disturbed mitochondrial unfolded protein response. Understanding these redox-related dysregulated pathways may give us novel insights into how to design innovative approaches to fighting kidney disease. This Special Issue will cover all topics related to AKI and CKD and will especially welcome submissions of manuscripts on redox mechanisms and the pathophysiology underlying AKI, CKD, and diabetic nephropathy or diabetic kidney disease (DKD). This Special Issue will consider both review articles and original research articles.

Guest Editor

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Biomolecules is a multidisciplinary open-access journal that reports on all aspects of research related to biogenic substances, from small molecules to complex polymers. We invite manuscripts of high scientific quality that pertain to the diverse aspects relevant to organic molecules, irrespective of the biological question or methodology. We aim for a competent, fair peer review and rapid publication. Please look at some of the exciting work that has been published in Biomolecules so far. We would be delighted to welcome you as one of our authors.

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