

Preface

Broad derangements of oxygen metabolism, such as oxidative stress and hypoxia, have been implicated in the genesis of kidney disease, independently of hemodynamic and metabolic abnormalities. They further impact various biological reactions linked to oxygen metabolism, such as nitrosative stress, advanced glycation, carbonyl stress, and endoplasmic reticulum stress. This causal role of impaired oxygen metabolism in kidney disease has implications for our understanding of current therapeutic benefits accruing from antihypertensive agents, the control of hyperglycemia/hyperinsulinemia or of hyperlipidemia, and the dietary correction of obesity. The defense mechanisms against oxidative stress (e.g., the Nrf2-Keap1 system) and hypoxia (e.g., the HIF-PHD system) have been recently explored in various cells, including kidney cells, and they include intracellular sensors for oxidative stress and hypoxia. Novel approaches targeting these sensors may offer clinical benefits in several disorders in which oxidative stress or hypoxia is a final, common pathway. Leading basic researchers and clinical scientists have contributed to this book and provide up-to-date, cutting-edge reviews on recent advances in the pathobiology of oxygen metabolism in kidney disease, especially oxidative stress and hypoxia.

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Toshio Miyata, MD, PhD
Kai-Uwe Eckardt, MD
Masaomi Nangaku, MD, PhD

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