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Forum Editorial

Role of Reactive Oxygen Species in Renal Function and Diseases

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In the last years, our concept of the reactive oxygen species (ROS) in normal renal function and renal disease has changed drastically. This change has been based not only on the increase in the knowledge on the initial mechanisms of ROS production and ROS action, but on the understanding that ROS are not only nocive agents that damage plasma membranes, structural and functional proteins, and nucleic acids, but specific second messengers to transmit the intracellular signaling of multiple hormones and autacoids. Thus, ROS have been involved in the control of cell contraction, proliferation, migration, adherence, expression of membrane proteins, synthesis of extracellular matrix, initiation and regulation of inflammatory mechanisms, and several other physiological phenomena.

The objective of this forum issue on Renal Function is to summarize current research on the role of ROS in several physiological functions of the kidney, as well as the alterations in ROS production in several pathological situations or therapies, such as dialysis, and the consequences of these alterations in the body homeostasis. This issue is a compilation of original research articles and reviews from leading researchers in the field. Hanna *et al.* (3) provide an overview of the mechanisms responsible for angiotensin II-induced production of ROS in blood vessels, heart, and kidney, as well as the mechanisms by which ROS regulate the function of these cells. In addition, these authors review the role of increased ROS production in several cardiovascular pathologies, such as hypertension, restenosis, atherosclerosis, and renal diseases. Closely related to this topic, an original research article from Rodriguez-Puyol *et al.* (5) focuses on the mechanisms of angiotensin II-induced ROS production by activation of both NADH/NADPH oxidase and phospholipase A2. Wilmer *et al.* (7) describe how peroxisome proliferator-activated receptor-α ligands inhibit H_2O_2-mediated activation of transforming growth factor-β1 in human mesangial cells, a mechanism involved in the increased extracellular matrix synthesis by these cells that leads to glomerulosclerosis and chronic renal failure.

Basnakian *et al.* (1) review the role of ROS as mediators of renal tubular apoptosis associated with acute renal failure induced by toxins or drugs such as gentamicin, cisplatin, or glycerol. They also describe the mechanisms of ROS-induced cell apoptosis and address how ROS scavenging or inhibition of ROS production can prevent toxic-induced cell death. In relation to this topic an original research article from Morales *et al.* (4) reports that treatment with resveratrol is able to modulate the toxic effects of gentamicin in rat kidney, and proposes that this effect is mediated by its antioxidant properties. Valdivielso and Blantz (6) extensively review the role of nitric oxide in the pathogenesis of acute renal failure, with special attention to its protective effect as vasodilator or ROS scavenger and to its deleterious effects inducing cell apoptosis and necrosis and contributing to the production of other damaging radicals, such as peroxynitrites. Cachofeiro *et al.* (2) studied the mechanisms by which chronic inhibition of nitric oxide synthesis leads to chronic renal failure. They describe a major role for the activation of the adrenergic sympathetic system in these renal alterations. In the last review, Wratten *et al.* (8) summarize the mechanisms involved in the increased oxidative stress observed in patients with end-stage renal failure subjected to hemodialysis, as well as the relevance of oxidative stress in the inflammatory alterations and cardiovascular diseases often seen in these patients.

It is hoped that this forum issue will stimulate the interest of researchers of ROS and the renal function and diseases, as well as the potential therapeutic perspectives of antioxidant and ROS scavenger drugs in the treatment of renal pathologies.

ABBREVIATION

ROS, reactive oxygen species.

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