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Interaction of oxidative stress, nitric oxide and peroxisome proliferator activated receptor γ in acute renal failure

Zivar Yousefipour [a], Adebayo Oyekan [a], Mohammad Newaz [b,*]

[a] Center for Cardiovascular Diseases, Texas Southern University, Houston, Texas, United States
[b] College of Pharmacy, Chicago State University, Chicago, Illinois, United States

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ABSTRACT

Oxidative stress has been reported to play a critical role in the pathology of acute renal failure (ARF). An interaction between different reactive species and/or their sources have been the focus of extensive studies. The exact sources of reactive species generated in biological systems under different disease states are always elusive because they are also a part of physiological processes. Exaggerated involvement of different oxidation pathways including NAD(P)H oxidase has been proposed in different models of ARF. An interaction between oxygen species and nitrogen species has drawn extensive attention because of the deleterious effects of peroxynitrite and their possible effects on antioxidant systems. Recent advances in molecular biology have allowed us to understand glomerular function more precisely, especially the organization and importance of the slit diaphragm. Identification of slit diaphragm proteins came as a breakthrough and a possibility of therapeutic manipulation in ARF is encouraging. Transcriptional regulation of the expression of slit diaphragm protein is of particular importance because their presence is crucial in the maintenance of glomerular function. This review highlights the involvement of oxidative stress in ARF, sources of these reactive species, a possible interaction between different reactive species, and involvement of PPARγ, a nuclear transcription factor in this process.

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1. Introduction

Acute renal failure (ARF), also known as acute kidney failure or acute kidney injury, is a rapid loss of renal function due to damage to the kidneys, resulting in retention of nitrogenous (urea and creatinine) and non-nitrogenous waste products that are normally excreted by the kidney. The condition often results from major trauma, illness, or surgery but is sometimes caused by a rapidly progressive, intrinsic renal disease. Causes of ARF can be classified as prerenal, intrinsic, or postrenal damage.

High rate of mortality has marked ARF as a significant health care concern. Since ARF is one of the complications of more serious problems such as cardiovascular diseases (CVD), many studies have attempted to clarify the mechanism of ARF in order to reduce the mortality. As the result of these studies, several mechanisms have been proposed and a role of oxidative stress has been among those.

* Corresponding author. College of Pharmacy, Chicago State University, 9501 S King Drive, Chicago, IL 60628, United States. Tel.: 773 821 2826; fax: 773 821 2595.
E-mail address: mnewaz@csu.edu (M. Newaz).

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Oxidative stress as the result of increased free radical (FR) generation has been implicated in the pathogenesis of many diseases including several renal diseases, such as ARF induced by glycerol, gentamicin, cisplatin, and cyclosporine. Numerous animal studies indicate that production of superoxide and nitric oxide are involved in prevention of normal kidney function. This article provides an overview of the causal interactions between oxidative stress and ARF.

2. Acute renal failure

Acute renal failure (ARF) has been defined as the rapid breakdown of renal function resulting in retention of high levels of uremic toxins. The condition occurs when the kidneys are unable to excrete the daily load of toxins in the urine. Reports indicate that approximately 1% of patients admitted to the hospital, 2 to 5% during the hospital stay, and 4 to 15% after cardiopulmonary bypass surgery develop ARF, but this condition can occur in all medical settings (Nolan & Anderson, 1998). In spite of extensive research in this area, the survival rate for ARF has not been improved mainly due to the age of the patients as well as more complicated conditions of the patients (Feest et al., 1993; Finn, 1993; Thadhani et al., 1996). The rate of reported mortality due to ARF can range from as low as 7% to as high as 80%, depending on the severity of the condition (Feest et al., 1993; Finn, 1993; Thadhani et al., 1996).

In ARF glomerular filtration rate (GRF) decreases, which causes reduction in excretion of nitrogenous waste and loss of balance of fluid and electrolytes. Based on the amount of urine that is excreted over a 24-hour period, patients with ARF are separated into oliguric (less than 500 mL of urine per day) or nonoliguric (more than 500 mL of urine per day) groups. Depending on the location of the problem within the renal system, three types of ARF can be identified: Prerenal, renal and postrenal. Prerenal conditions are characterized by inadequate blood circulation (perfusion) to the kidneys caused by dehydration, heart failure, sepsis, and severe blood loss. Postrenal conditions are caused by an acute obstruction that affects the normal flow of urine out of both kidneys and can be due to bladder outlet obstruction, kidney stones in ureters, neurogenic bladder, tubule obstruction, renal injury, or retroperitoneal fibrosis. Renal or intrinsic condition involves damage or injury within both kidneys, due to vascular disease, diseases of tubules and interstitium and acute tubular necrosis. The most common symptom of ARF is decrease in urine output, which increases the risk of developing complications that can cause seizures, bleeding, and coma. Advanced age and underlying diseases determine the risk of dying from ARF. Despite the increasing awareness of this syndrome, the advances in clinical care and extensive research of its pathophysiology, the mortality from ARF remains high and has not changed significantly during the last several decades (Lameire et al., 2006). A better understanding of the mechanism of ARF will be beneficial in reduction of ARF contributed mortality.

3. Free radicals in biological systems

Free radical generation and resulting oxidative stress have been reported as one mechanism of development of ARF (Table 1) and organ injury. Free radicals are atoms, molecules, or ions with unpaired electrons that are usually very active. Radicals play an important role in many chemical processes as well as human physiology. Small amounts of reactive oxygen species (ROS) are crucial for several important biological functions such as signal transduction pathways, cellular response to anoxia, and induction of mitogenic responses (Valko et al., 2007). Superoxide and nitric oxide are known as important regulators of vascular tone. In biological systems, these small amounts of ROS are generated endogenously from different sources. Mitochondrial electron transport chain and enzymes such as NAD(P)H oxidases, xanthine oxidases, cyclooxygenases, cytochrome P450 enzymes, lipooxygenases, and uncoupled nitric oxide synthases (Mueller et al., 2005) are the main sources of ROS in biological systems. Although minute amounts of ROS are important and necessary for optimal functioning of the body, at high concentrations ROS have harmful effects and can cause organ damage. Among the classes of ROS, superoxide anion, which can interact with other molecules and produce secondary ROS (Valko et al., 2005), is considered the most damaging one. ROS are formed in many ways including interaction of ionizing radiation with biological molecules (Gouk et al., 2005), as byproducts of cellular respiration (Bailey et al., 2005), as well as enzymes such as NAD(P)H oxidases and myeloperoxidase (Babior, 1992, 2000; Koppenol et al., 1992; Winterbourn et al., 2000).

In the immune system, superoxide is used to destroy invading microorganisms and in phagocytes is used as an oxygen-dependent killing mechanism against invading pathogens (Sawyer, 2000). Superoxide is one of the byproducts of mitochondrial respiration (Complex I and Complex II) as well as xanthine oxidase (Sawyer, 2000). To keep the amount of superoxide in balance and prevent the damage caused by superoxide, organisms use isoforms of the superoxide scavenging enzyme, superoxide dismutase (SOD), which catalyzes neutralization of superoxide.

4. Oxygen free radicals and acute renal failure

In the absence of an effective scavenger system or in the case of high production of superoxide, oxygen free radicals can contribute to the pathogenesis of many diseases. There are several reports of induction of superoxide ions in different models of ARF. Schramm et al. (2008), using renal artery clamping for 40 min to induce ARF, reported an increased production of superoxide ions and showed treatment with L-Arg, SOD or

Table 1
Evidence of free radical involvement in different experimental models of ARF.

	Types of radicals	Reference
Gentamicin-induced ARF	↑$OH^{\bullet}$, ↑$O^{\bullet -}$; SAMC reduced both	Pedraza-Chaverrí et al., 2004
	↑O_2^-; M40403 (a superoxide dismutase mimetic) scavenged it	Cuzzocrea et al., 2002
	↑H_2O_2; catalase reduced ROS	Walker et al., 1999
	↑O_2^-, ↑H_2O_2, ↑$OH^{\bullet}$; DMSO, CAT and DFO reduced OH while SOD reduced O_2^-	Yang et al., 1995
Cisplatin-induced ARF	↑$ONOO^-$; S-allylmercaptocystein reduced it	Chirino and Pedraza-Chaverri, 2009
	↑$ONOO^-$; 1400 W caused reduction	Chirino et al., 2008
	↑$OH^{\bullet}$; DMTU and NAC scavenged it	Jiang et al., 2007
	↑$ONOO^-$; FeTPPS prevented renal damage	Chirino et al., 2004
	↑O_2^-, AH-SOD improved renal function	Nishikawa et al., 2001
	↑$OH^{\bullet}$, ↑H_2O_2; tiron, catalase, pyruvate, trolox and deferoxamine prevented damage	Baek, et al., 2003
Cyclosphorine-induced ARF	↑O_2^-; hydroxytyrosol (DOPET) prevented the production	Capasso et al., 2008
	↑H_2O_2, ↑O_2^-; Vit E removed ROS	Parra et al., 2003
	↑H_2O_2; dietary glycin prevented renal damage	Zhong et al., 1998
Glycerol-induced ARF	↑NO; chitosanoligosaccharide (COS) improved renal function	Yoon et al., 2008a, 2008b
	↑$OH^{\bullet}$; U74389G, prevented renal damage	Yousefipour, Hercule, Oyekan and Newaz, 2007, 2008
	↑H_2O_2; pyruvate scavenged H_2O_2	Salahudeen et al., 1991

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