

High blood pressure: How does it affect male fertility potential?

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Abstract: *Reactive Oxygen Species (ROS) are ubiquitous reactive derivatives of O₂ metabolism found in the environment and in all biological systems. Within the cardiovascular system, ROS play a crucial physiological role in maintaining cardiac and vascular integrity and a pathophysiological role in cardiovascular dysfunction associated with several clinical conditions, including hypertension. It is known that oxidative stress affects the testicular function by disruption of germinal cell epithelial division and differentiation along with the induction of germ cell apoptosis*

Key words: *Reactive oxygen species, cardiovascular dysfunction, testicular function*

Introduction

Reactive Oxygen Species (ROS) are ubiquitous reactive derivatives of O₂ metabolism found in the environment and in all biological systems. ROS are implicated in many intracellular signaling pathways leading to changes in gene transcription and protein synthesis and consequently in cell function.

ROS and cardiovascular system

Within the cardiovascular system, ROS play a crucial physiological role in maintaining cardiac and vascular integrity and a pathophysiological role in cardiovascular dysfunction associated with several clinical conditions, including hypertension [1, 2]. The most important ROS detectable within the vasculature include the superoxide anion ($\bullet\text{O}_2^-$), hydrogen peroxide (H_2O_2), hydroxyl radical ($\bullet\text{OH}$), and the reactive nitrogen species peroxynitrite (ONOO^-), which have been regarded as a nasty, life-threatening, and destructive oxygen-derived toxicant. In healthy conditions, ROS are produced in a controlled manner at low concentrations and function as signalling molecules regulating vascular contraction-relaxation and cell growth [3].

Physiologically, ROS generation is tightly regulated by endogenous cellular antioxidants, which include superoxide dismutase (SOD), catalase, thioredoxin, glutathione, and antioxidant vitamins. In physiological conditions, the rate of ROS generation is counterbalanced by the rate of elimination. In contrast, under pathological conditions, such as hypertension, ROS are produced in concentrations that cannot be controlled by the usual protective antioxidant mechanisms

employed by the cells, leading to a state of oxidative stress [2]. Indeed, when produced in excess, $\bullet\text{O}_2$ – reacts with nitric oxide (NO) to produce a dramatic concentration of the toxic ONOO– which promotes a variety of negative effects on cellular function. These include alteration of transcription factors, kinases, protein synthesis, and redox-sensitive genes, which in turn influence endothelial function, increase vascular contractility, vascular smooth muscle cell growth and apoptosis, monocyte migration, lipid peroxidation, inflammation, and increased deposition of ECM proteins, all major processes deeply involved in the pathogenesis and progression of vascular damage in cardiovascular disease [4,5].

ROS and testicular function

It is known that oxidative stress affects the testicular function by disruption of germinal cell epithelial division and differentiation along with the induction of germ cell apoptosis [6, 7].

The mechanisms underlying the apoptosis induction by oxidative stress are not clear. However, they are shown to be due to the involvement of cytokine-induced stress kinase and E-selectin expression in the testicular vascular endothelium [7, 8, 9].

Induction of apoptosis leads to testicular neutrophil recruitment and increases the generation of intra-testicular reactive oxygen species (ROS). ROS in turn, cause peroxidative damage to cell membranes and also activate germ cell apoptosis [10, 11, 12]. The rate of phagocytosis by Sertoli cells is also enhanced by increased germ cell apoptosis so as to clear the dying and damaged germ cells [13, 14].

The ROS produces toxic effects at 3 different levels. Firstly ROS activates apoptotic mechanism on gamete cells [7, 8, 9]. Secondly suppress the cell division and differentiation directly [6]. Thirdly, activates the phagocytic mechanism in Sertoli cells so that damaged and apoptotic cells are phagocytosed [13, 14].

Conclusion

The studies indicate that oxidative stress affects the cardiovascular system as well as male reproductive system adversely.

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