IAA Journal of Applied Sciences 14(1):49-54, 2026. www.iaajournals.org
©IAAJOURNALS ISSN: 2636-7246
https://doi.org/10.59298/IAAJAS/2026/1414954 IAAJAS: 141000

Redox Biology of Prostatic Enlargement: The Role of Antioxidants in BPH Progression and Therapy

Abonyi, Emmanuel Obiora

Department of Biochemistry, College of Medicine, Enugu State University of Science and Technology, Enugu State, Nigeria
Corresponding author: Abonyi, Emmanuel Obiora, abonyi.emmanuel@esut.edu.ng, +2347030853307

ABSTRACT

Benign prostatic hyperplasia (BPH) is a common age-related disease, which is a non-malignant enlargement of the prostate, lower urinary tract symptoms and progressive worsening of the quality of life. Even though it has historically been thought to be caused by hormonal imbalance and stromal-epithelial interactions, emerging evidence suggests that oxidative stress and redox dysregulation are key factors in driving prostate tissue remodeling. Mitochondrial dysfunction, chronic inflammation, metabolic dysfunctions, and environmental exposures lead to the production of reactive oxygen species (ROS), which facilitates DNA damage, cellular senescence, extracellular matrix expansion, and proliferative signaling, characteristic features of hyperplastic growth. The biological convergence of oxidative stress and chronic prostate inflammation is demonstrated by the integration of oxidative cues into inflammatory and fibrotic responses by redox-sensitive pathways, including NF- kB, Nrf2, MAPKs, and PI3K/Akt. Oxidative injury and BPH progression can be alleviated by antioxidants- endogenous systems and exogenous dietary or pharmacologic antioxidants-as a promising treatment option. This review summarizes existing information on the redox biology of BPH, outlines major molecular and cellular processes that relate oxidative imbalance to hyperplasia, and identifies the new evidence on the potential use of antioxidants in prevention and treatment. Gaining insights into the role of redox perturbations in the prostate aging process and pathology can provide a disease-modifying way of treating BPH.

Keywords: Prostate fibrosis, redox signaling, antioxidants, excess benign prostatic hyperplasia, oxidative stress.

INTRODUCTION

Benign prostatic hyperplasia is a disease on a large proportion of older men, and its prevalence rate increases at an alarming rate after 50 years. BPH is pathologically characterized by the hyperplasia of stromal and epithelial elements in the transitional zone of the prostate [1]. The clinical features encompass frequency of urine, nocturia, hesitancy, weak urinal stream, incomplete emptying of the bladder, and acute retention in some instances [2]. Previously deemed to be a hormonally driven condition, particularly dihydrotestosterone and stromal-epithelial interactions, BPH is currently considered a multifactorial disease with an effect caused by hormonal imbalance, low-grade chronic inflammation, metabolic dysfunction, and oxidative stress due to age [3]. Recent studies have put redox biology at the heart of the pathogenesis of BPH. As a person ages, the prostate is more prone to oxidative stress because of mitochondrial malfunctioning, diminished antioxidant defenses, and extended exposure to inflammatory stimuli [4]. Oxidative stress is not only an outcome of an inflammatory response but a first-time driver of tissue remodelling, fibrotic response, angiogenesis, and proliferative signaling [5]. This is because of the high sensitivity of the prostate to peroxidative damage mediated by ROS, which is specifically sensitive to metabolic conditions such as a high content of polyunsaturated lipids, intensive peroxisomal and mitochondrial activity [6]. Redox balance [7] may be therapeutically advantageous in the restoration of redox balance either through endogenous enzymatic systems or the exogenous nutraceutical and pharmacologic agents as antioxidants.

Nevertheless, their exact functions, processes, and clinical effects are the topics of ongoing research. The paper reviews the role of redox dysregulation in prostate enlargement and assesses the therapeutic benefit of using antioxidant measures to delay the progression of BPH [8].

2. Oxidative Stress Sources of Prostate Tissue.

2.1 Mitochondrial impairment and generation of ROS with age.

One of the most important causes of oxidative stress in the prostate is aging, especially since there is a gradual worsening of mitochondria [9]. In old age, men experience a decrease in mitochondrial efficiency with a resultant drop in electron transport chain fidelity and increasing electrons leaking to molecular oxygen prematurely to produce superoxide radicals [10]. This process is especially sensitive to the prostate stromal fibroblasts as well as epithelial basal cells due to their high metabolic rate and high sensitivity to oxidative damage. Mitochondrial DNA mutations that accompany aging also worsen oxidative phosphorylation and reduce ATP generation and enhance cell repair functions [11]. The outcome is a vicious cycle whereby mitochondrial damage facilitates excessive production of ROS and the buildup of ROS damages more mitochondrial proteins, lipids, and nucleic acids [12]. This long-term chronic redox imbalance leads to functional deterioration of prostate cells and a biochemical environment that supports hyperplastic development.

2.2 ROS amplifier in chronic inflammation.

Chronic inflammation is a powerful and well-known cause of enhanced oxidative stress in prostate tissue [13]. Macrophages, neutrophils, and activated T lymphocytes that compose immune cell infiltrates produce large amounts of ROS and reactive nitrogen species by the activity of enzymes, including NADPH oxidases, myeloperoxidase, and inducible nitric oxide synthase [14]. In numerous BPH samples, enduring inflammatory lesions are accompanied by epithelial and stromal cell proliferation, which may indicate that oxidative species that are generated under the impact of inflammation are some signaling mediators that stimulate tissue remodeling [15]. Inflammatory cytokines promote redox-sensitive processes, fibroblast activation, and strengthen stromal-epithelial crosstalk [16]. The resultant oxidative microenvironment triggers cell growth and ECM deposition, which stimulates hyperplastic transformation [17].

2.3 Redox imbalance and metabolic dysfunction

Oxidative stress in the prostate is greatly aggravated by metabolic diseases such as obesity, insulin resistance, and type 2 diabetes [18]. Hyperglycemia augments the production of ROS by glucose auto-oxidation, augmented polyol flux, as well as deposition of advanced glycation end-products, which attach to RAGE receptors and trigger pro-inflammatory, pro-oxidant pathways [19]. Lipid peroxidation is also increased, and intrinsic antioxidant defenses are weakened by dyslipidemia and chronic low-grade inflammation [20]. Such metabolic abnormalities increase prostate susceptibility to oxidative damage and could be one reason that men with metabolic syndrome have larger prostate volumes with more severe lower urinary tract symptoms.

2.4 Environmental and lifestyle factors.

There are other oxidative burdens introduced by environmental exposures and lifestyle choices that have an impact on prostate biology. Increased ROS formation is caused by cigarette smoke, alcohol consumption, environmental toxins and chronic genitourinary infections [21]. High saturated fat and low antioxidant-containing fruit, vegetable, and micronutrient diets are damaging redox balance in cells and are associated with high levels of oxidative stress markers [22]. Combined together with natural aging, inflammation and metabolic dysfunction, these factors increase oxidative stress and accelerate the development of benign prostatic enlargement.

3. Redox Imbalance Molecular Consequences of Redox Imbalance in the Prostate.

3.1 Genomic instability and oxidative DNA damage.

Oxidative stress has significantly far-reaching effects concerning genomic integrity in the prostate. Reactive oxygen species are attracted easily to nucleic acids, producing mutagenic damage in the form of 8-hydroxy-2'-deoxyguanosine, oxidized purines and pyrimidines, and single and double-strand breakages [23]. In spite of various repair mechanisms used by the cells, such as base excision repair and nucleotide excision repair, there is persistence of oxidative damage when the systems face constant bombardment and lesions persist in accumulating [24]. Those cells that fail to repair DNA may commit to senescence, a condition where irreversible growth arrest and continued activation of inflammatory signals characterize senescence [25]. Senescent prostate epithelial and stromal cells have a senescence-associated secretory phenotype, which is a secretory phenotype characterized by the release of cytokines, chemokines, growth factors, and proteases. This secretory program enhances local inflammation, increases additional oxidative damage, and changes the behavior of surrounding cells, resulting in a microenvironment more conducive to hyperplastic growth and stromal expansion [26]. With time, genomic instability and chronic SASP signaling may converge, which leads to prostate remodelling and exposure to abnormal proliferative responses.

3.2 Lipid peroxidation and dysfunction of the membrane.

Polyunsaturated fatty acids are also found in high levels in the prostate; the cellular membranes of the prostate are particularly susceptible to lipid peroxidation [27]. The oxidation of these lipids results in the production of reactive

aldehydes, including 4- hydroxynonenal and malondialdehyde, that form covalent adducts with proteins, nucleic acids, and phospholipids. These adducts destabilize membranes, interfere with receptor activity and function, and affect the operation of ion channels, eventually disrupting cellular signaling and metabolic processing [28]. Lipid peroxidation products can also be considered the secondary messengers that stimulate the stress-responsive and profibrotic pathways. Their buildup is connected to the accelerated fibroblast activity, the elevated smooth muscle tone, and the stimulation of the proliferative signals that expedite stromal and epithelial hyperplasia [29]. Lipid peroxidation, therefore, is an important marker of oxidative stress and a direct mediator of structural and biochemical events that are evident in benign prostatic enlargement.

3.3 Redox-dependent signaling pathways activation.

The oxidative stress can regulate various intracellular signaling pathways that control the processes of inflammation, proliferation and tissue remodeling [30]. The NF-kB is at the center stage because its activation triggers cytokine, adhesion molecule and survival gene transcription that promotes immune recruitment and stromal proliferation [31]. Oxidative cues are combined with mitogen-activated protein kinases, such as ERK, JNK, and p38 in order to regulate cell growth, apoptosis, and adaptation to stress. AP-1 is involved in the extracellular matrix reorganization and expression of inflammatory genes, whereas the PI3K/Akt pathway increases cell survival and hyperplastic growth in oxidative conditions [32]. Also, the TGF-b/SMAD signaling is highly affected by ROS, which promotes fibroblast differentiation into contractile myofibroblasts and stimulates fibrosis [33]. Redox imbalance is one of the molecular engines of pathogenic remodeling in BPH through activation of these pathways in the long run.

3.4 Extracellular matrix remodelling, fibrosis.

Fibrosis is a characteristic of progressive benign prostatic hyperplasia, and it is gaining more and more weight as a predictor of symptom severity and resistance to medical treatment. The high level of ROS induces fibroblast growth, increases of myofibroblast activation, and stimulates the production of extracellular matrix proteins (collagen types I and III, fibronectin, and elastin) [34]. Simultaneously, oxidative stress triggers matrix metalloproteinases, which break down already existing matrix structures to form a dynamic yet unregulated remodeling [35]. The net result is the formation of thick, chaotic, and hard extracellular matrix that decreases tissue compliance and forms part of the mechanical obstruction of the lower urinary tract. Constant oxidative stress also enhances the effect of TGF-b, continuing the fibrotic loop [36]. With time, the redox imbalance leads to structural changes resulting in severe enlargement of the prostate and less responsiveness to the conventional therapies.

4. Therapeutic Implications and Future Perspectives.

The entirety of redox biology in benign prostate hyperplasia provides several areas of therapy application. The recognition of patient subgroups with high levels of oxidative stress or certain redox imbalances could allow the consideration of stratified treatment strategies in order to fully use the existing efficacy and avoid unnecessary exposure to pharmacologic agents [37]. Activation of the main redox-sensitive signaling pathways, including the Nrf2 activation, is of particular interest as this transcription factor coordinates the innate antioxidant defenses and cytoprotective mechanisms. Redox balance, antiproliferative effects of ROS, and fibrosis in the prostate could be restored by pharmacologic/nutraceutical Nrf2 modulators [38]. The combination of antioxidants and conventional BPH medications, such as alpha-adrenergic blockers or 5-alpha-reductase inhibitors, can be used to improve the control of symptoms and possibly reduce the rate of progression of the disease by managing the underlying oxidative processes instead of only improving the obstruction of urine [39]. Individualized therapy would be possible through the development and utilization of oxidative stress biomarkers to enable monitoring of response to treatment and expression of antioxidant regimens in a person. Complementary approaches to lifestyle and diet, including raising the intake of foods abundant in antioxidants, caloric deprivation, and exercise, are effective interventions that lower the systemic oxidative load and enhance metabolic well-being [40]. The future studies should involve integration of redox profiling, mitochondrial functional assessment, and individual antioxidant therapy during clinical trials to find out the optimal dosing, synergies, and longer-term results, finally reducing experimental results to effective disease-modifying approaches that can be applied in managing BPH.

CONCLUSION

Oxidative stress is a key mediator of BPH pathogenesis linking aging, inflammation, metabolic dysfunction and tissue remodelling. Many of the structural changes and functional changes in prostate enlargement are mediated by ROS-mediated damage and redox-sensitive signalling. Antioxidants provide an attractive, comprehensive method of regulating hyperplasia, fibrosis, and inflammatory stimulation. Despite the lack of clinical evidence, redox biology advances offer a solid scientific basis for interventions based on antioxidants. With the advancement in research, combining redox-targeted therapies with the currently available pharmacologic and lifestyle interventions could provide a more holistic and disease-altering approach in the management of BPH.

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CITE AS: Abonyi, Emmanuel Obiora (2026). Redox Biology of Prostatic Enlargement: The Role of Antioxidants in BPH Progression and Therapy. IAA Journal of Applied Sciences 14(1):49-54. https://doi.org/10.59298/IAAJAS/2026/1414954