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# Mitochondrial Oxidative Stress and Prostatic Hyperplasia: The Role of Antioxidant-Rich Nutraceuticals

**Kato Sseguya I.**

**Faculty of Medicine Kampala International University Uganda**

## ABSTRACT

Benign prostatic hyperplasia (BPH) is a prevalent, non-cancerous condition that affects aging men and significantly impairs urinary function and quality of life. Recent advances in molecular pathology have implicated mitochondrial dysfunction and associated oxidative stress as central contributors to the initiation and progression of prostatic hyperplasia. Mitochondria, being the major sites of reactive oxygen species (ROS) production, are vulnerable to oxidative damage, which compromises cellular energy metabolism, redox signaling, and apoptosis regulation in prostatic tissues. The imbalance between mitochondrial ROS generation and antioxidant defenses exacerbates stromal and epithelial proliferation, inflammation, and fibrosis, key hallmarks of BPH. This review discusses the emerging role of mitochondrial oxidative stress in the pathophysiology of BPH and highlights the therapeutic promise of antioxidant-rich nutraceuticals. Phytochemicals such as polyphenols, flavonoids, carotenoids, and mitochondrial-targeted compounds like Coenzyme Q10 and alpha-lipoic acid are examined for their protective effects against mitochondrial ROS, promotion of biogenesis, and restoration of prostatic redox homeostasis. The integration of such nutraceuticals into BPH management offers a safe and potentially disease-modifying strategy, especially in early or progressive stages. Future research should focus on translational validation, bioavailability enhancement, and personalized nutraceutical approaches to mitigate mitochondrial dysfunction in prostate disease.

**Keywords:** Benign prostatic hyperplasia, Mitochondrial dysfunction, Oxidative stress, Antioxidant nutraceuticals, Prostatic inflammation

## INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common non-cancerous conditions affecting aging men, characterized by a progressive enlargement of the prostate gland due to the hyperplasia of epithelial and stromal cells [1]. The global prevalence of BPH rises steeply with age, affecting nearly 50 percent of men in their 50s and over 80 percent of men above the age of 80. The condition is associated with lower urinary tract symptoms (LUTS), such as increased urinary frequency, nocturia, incomplete bladder emptying, urinary hesitancy, and weak stream [2]. These symptoms significantly compromise patients' quality of life and pose an increasing socioeconomic burden due to the costs of clinical management, surgical interventions, and lost productivity. Historically, the pathophysiology of BPH has been attributed to hormonal imbalances, especially the role of androgens and the enzyme 5-alpha reductase, which converts testosterone into its more potent form, dihydrotestosterone (DHT) [3]. DHT binds to androgen receptors in prostate cells and drives proliferation [4]. However, recent molecular studies have illuminated a more complex picture in which chronic inflammation, immune dysregulation, metabolic disorders, and oxidative stress converge to sustain and accelerate prostatic enlargement. Among these, mitochondrial oxidative stress has emerged as a critical but underappreciated contributor to BPH pathogenesis [5].

Mitochondria are the central regulators of energy metabolism, cellular homeostasis, and apoptotic pathways [6]. They are also the primary sites of intracellular reactive oxygen species (ROS) production during oxidative phosphorylation [6]. Under physiological conditions, mitochondrial ROS serve important signaling functions and are counterbalanced by endogenous antioxidant systems [7]. However, during aging and in disease states such as BPH, there is increased mitochondrial dysfunction, resulting in excessive ROS generation that overwhelms antioxidant defenses and leads to oxidative damage of lipids, proteins, and DNA [8]. This redox imbalance promotes

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inflammation, fibrosis, aberrant cell proliferation, and resistance to apoptosis—core features of BPH histopathology [9]. The growing recognition of oxidative stress as a modifiable factor in BPH has spurred interest in natural, safe, and long-term management options. Antioxidant-rich nutraceuticals—bioactive compounds derived from food or plant sources—hold therapeutic promise in targeting mitochondrial ROS and restoring redox homeostasis in prostate tissue [10]. These compounds, including polyphenols, carotenoids, vitamins, and mitochondrial cofactors, offer multifaceted benefits such as scavenging free radicals, enhancing endogenous antioxidant enzyme activity, preserving mitochondrial integrity, and modulating signaling pathways implicated in cell proliferation and inflammation [11]. This review provides an in-depth examination of mitochondrial oxidative stress in the context of BPH and evaluates the evidence supporting the use of antioxidant nutraceuticals as a therapeutic strategy. By focusing on mechanistic insights and translational perspectives, we aim to inform integrative approaches to BPH management that address both symptom relief and disease progression.



## Mitochondrial Dysfunction and Oxidative Stress in BPH

Mitochondria are dynamic organelles responsible not only for generating cellular energy through oxidative phosphorylation but also for regulating redox balance, calcium signaling, and apoptotic pathways [12]. Within the mitochondria, electrons pass through the electron transport chain (ETC) to generate ATP, but a fraction of these electrons leak, particularly at complexes I and III, leading to the formation of reactive oxygen species (ROS) such as superoxide anions [13]. Under normal physiological conditions, these ROS are neutralized by antioxidant defenses including manganese superoxide dismutase (MnSOD), glutathione peroxidase (GPx), and catalase, maintaining cellular homeostasis [11]. However, aging and metabolic stress can impair mitochondrial function, leading to an overproduction of ROS and subsequent oxidative damage [14]. In the prostate gland, which is highly metabolically active and rich in polyunsaturated fatty acids, mitochondria are especially vulnerable [15]. Mitochondrial DNA (mtDNA), located near the inner mitochondrial membrane and lacking protective histones, is particularly susceptible to oxidative modifications [16]. Oxidative damage to mtDNA results in mutations and deletions that compromise the efficiency of the ETC, further exacerbating ROS generation and creating a vicious cycle of mitochondrial dysfunction and oxidative stress [17]. In the context of BPH, this persistent mitochondrial oxidative stress contributes to several pathogenic processes. Firstly, ROS can activate redox-sensitive transcription factors such as nuclear factor-kappa B (NF-κB), which upregulates pro-inflammatory cytokines including interleukin-6 (IL-6), interleukin-8 (IL-8), and tumor necrosis factor-alpha (TNF-α) [18]. These cytokines promote the recruitment and activation of immune cells within prostatic tissue, perpetuating chronic inflammation, which in turn contributes to cellular proliferation, tissue remodeling, and fibrosis [19]. Secondly, mitochondrial ROS influence key growth and survival pathways such as the phosphoinositide 3-kinase (PI3K)/Akt and mitogen-activated protein kinase (MAPK)/ERK signaling cascades [20, 21, 22, 23, 24, 25]. Activation of these pathways results in increased expression of cyclins and other cell cycle regulators, supporting the hyperplastic expansion of epithelial and stromal compartments in the prostate [26, 27, 28, 29, 30]. Concurrently, oxidative stress disrupts apoptotic signaling, allowing abnormal cells to evade programmed cell death, which further contributes to tissue overgrowth [31, 32, 33, 34, 35, 36].

Studies have demonstrated elevated levels of oxidative stress markers in BPH tissue, including lipid peroxidation products like malondialdehyde (MDA), oxidative DNA damage markers such as 8-hydroxy-2'-deoxyguanosine (8-OHdG), and reduced levels of antioxidant enzymes [37,38,39]. Additionally, mitochondrial ultrastructural abnormalities, such as swollen cristae and vacuolization, have been observed in prostate tissue of BPH patients, confirming mitochondrial involvement in disease pathology [40, 41, 42, 43, 44, 45]. These findings position mitochondrial dysfunction and oxidative stress as not just consequences but central drivers of BPH development and progression. Therefore, strategies that restore mitochondrial integrity and rebalance redox signaling, such as the use of targeted antioxidant nutraceuticals, represent a rational therapeutic approach. These interventions may not only alleviate LUTS but also slow or reverse the molecular processes underpinning prostatic enlargement [46, 47, 48].

## Antioxidant-Rich Nutraceuticals: Mechanisms and Therapeutic Relevance

The concept of using nutraceuticals natural compounds derived from dietary or botanical sources for managing oxidative stress-related diseases is increasingly gaining traction. In the context of benign prostatic hyperplasia (BPH), nutraceuticals rich in antioxidants offer a promising therapeutic avenue by modulating mitochondrial redox status and interrupting pathological pathways driven by oxidative stress, chronic inflammation, and abnormal cell growth [49, 50]. Antioxidant nutraceuticals exert their beneficial effects through several mechanisms. First, they directly scavenge reactive oxygen species (ROS), neutralizing superoxide, hydrogen peroxide, and hydroxyl radicals generated by dysfunctional mitochondria [51, 52, 53, 54]. Second, they enhance the expression and activity of endogenous antioxidant enzymes such as superoxide dismutase (SOD), catalase, and glutathione peroxidase (GPx), restoring redox equilibrium [55, 56, 57, 58, 59]. Third, certain nutraceuticals activate mitochondrial biogenesis via the peroxisome proliferator-activated receptor gamma coactivator-1 alpha (PGC-1α) pathway, improving energy

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metabolism and reducing ROS leakage [60]. Lastly, they regulate key signaling cascades including NF-κB, MAPK, and PI3K/Akt, which are involved in inflammation, cell proliferation, and apoptosis resistance [61, 62, 63].

**Polyphenols**

Polyphenols are among the most extensively studied antioxidant nutraceuticals. Resveratrol, a stilbene found in grapes and red wine, has been shown to improve mitochondrial function by activating sirtuin 1 (SIRT1) and PGC-1α, promoting mitochondrial biogenesis and reducing ROS production [28]. In BPH models, resveratrol suppresses NF-κB-mediated inflammation and downregulates androgen receptor signaling, thereby reducing prostate volume and histological hyperplasia [29]. Curcumin, the yellow pigment of turmeric, has pleiotropic antioxidant, anti-inflammatory, and anti-proliferative effects. It scavenges ROS, inhibits cyclooxygenase-2 (COX-2), and enhances Nrf2 signaling, which governs the expression of multiple antioxidant genes [30]. In animal models of testosterone-induced BPH, curcumin has been shown to reduce prostate weight, normalize epithelial architecture, and decrease oxidative stress markers [31]. Epigallocatechin gallate (EGCG), a catechin in green tea, modulates mitochondrial membrane potential and prevents cytochrome c release, a key event in ROS-mediated apoptosis resistance [32]. EGCG also inhibits 5-alpha reductase, the enzyme that converts testosterone to dihydrotestosterone (DHT), thus modulating androgenic stimulation in the prostate [33].



**Carotenoids**

Carotenoids are lipophilic antioxidants that localize to membranes and protect against lipid peroxidation [34]. Lycopene, a red carotenoid found in tomatoes, accumulates in prostate tissue and has been shown to reduce oxidative stress and inhibit insulin-like growth factor (IGF) signaling [35]. Clinical trials suggest that lycopene supplementation may improve lower urinary tract symptoms (LUTS) and reduce prostate-specific antigen (PSA) levels [36].

**Coenzyme Q10 and Alpha-Lipoic Acid**

Coenzyme Q10 (ubiquinone) plays a central role in the mitochondrial electron transport chain and also functions as a potent antioxidant. In oxidative stress conditions, CoQ10 reduces lipid peroxidation and improves mitochondrial efficiency [37]. Alpha-lipoic acid (ALA) is another mitochondrial cofactor that regenerates other antioxidants such as glutathione, vitamin C, and vitamin E [38]. ALA has been used successfully in diabetic neuropathy and shows potential in addressing oxidative damage in prostatic tissue.

**N-Acetylcysteine (NAC)**

NAC serves as a precursor to glutathione, the primary intracellular thiol antioxidant. By replenishing glutathione stores, NAC supports detoxification and protects against oxidative DNA and protein damage [39]. In rodent BPH models, NAC reduces prostate weight, inflammatory cytokine levels, and tissue fibrosis, suggesting its value as an adjunctive antioxidant strategy [40].

These nutraceuticals, alone or in combination, provide a comprehensive mechanism-based strategy for reducing mitochondrial oxidative stress and attenuating the progression of BPH. However, their clinical effectiveness is influenced by factors such as bioavailability, dosage, duration of use, and individual variability, which are discussed in the next section.

**Clinical Implications, Bioavailability, and Future Directions**

Despite the extensive preclinical data supporting the role of antioxidant nutraceuticals in counteracting mitochondrial dysfunction and prostatic hyperplasia, clinical translation remains complex. One major challenge is the poor bioavailability of many phytochemicals due to low solubility, rapid metabolism, and limited gastrointestinal absorption. This limits their therapeutic concentration in prostate tissue and dampens clinical outcomes. For instance, curcumin exhibits potent antioxidant and anti-inflammatory properties in vitro, but its clinical application has been hampered by poor absorption and rapid systemic elimination [41]. To address this, formulation innovations such as nanoparticles, liposomes, phytosomes, and solid lipid carriers have been developed. These delivery systems enhance absorption, prolong circulation time, and facilitate tissue-specific targeting. For example, curcumin phytosomes have demonstrated superior bioavailability and improved efficacy in inflammatory and metabolic disorders [42]. Similarly, combining bioactive compounds with absorption enhancers improves pharmacokinetics. Piperine, derived from black pepper, inhibits hepatic and intestinal glucuronidation, significantly enhancing the bioavailability of curcumin and resveratrol [43]. Lipid-based carriers for lycopene and CoQ10 also show improved plasma levels and tissue accumulation in clinical studies. In terms of clinical evidence, although randomized controlled trials (RCTs) investigating individual nutraceuticals in BPH are relatively limited, findings are promising. A double-blind RCT using lycopene in men with BPH reported reduced prostate volume and improved LUTS over six months [44]. Another trial evaluating green tea extract demonstrated improvement in IPSS scores and quality of life indices [45]. Despite positive findings, differences in patient selection, extract standardization, outcome measures, and trial duration limit generalizability. Additionally, there is a need to evaluate interactions between nutraceuticals and conventional therapies such as alpha-blockers or 5-alpha reductase inhibitors. While some combinations may offer additive or synergistic effects, others could alter drug metabolism

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and efficacy. Therefore, integrative treatment plans should be guided by evidence-based protocols and personalized based on patient-specific risk factors, comorbidities, and pharmacogenetic profiles.

**Future research should prioritize**

- Development of standardized, bioavailable nutraceutical formulations
- Large-scale, multicenter trials with long-term endpoints
- Validation of mitochondrial biomarkers (e.g., mtDNA damage, mitochondrial membrane potential) as therapeutic targets
- Exploration of microbiome-mediated metabolism and its influence on nutraceutical efficacy
- Creation of clinical algorithms integrating nutraceuticals into standard BPH management pathways



## CONCLUSION

Antioxidant-rich nutraceuticals represent a novel and promising frontier in BPH management. By targeting mitochondrial oxidative stress, a central driver of prostatic hyperplasia. These agents offer the potential not only for symptom control but also for modifying disease trajectory. While challenges remain in formulation and clinical validation, ongoing research and technological advances are rapidly expanding the therapeutic utility of nutraceuticals in urological health.

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