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Dual-Targeted Antioxidant Therapies for Benign Prostatic Hyperplasia and Liver Dysfunction: Novel Strategies for Managing Urological and Hepatic Complications

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ABSTRACT

Benign Prostatic Hyperplasia (BPH) and liver dysfunction represent two prevalent health conditions in aging populations, often coexisting within the same patient due to shared metabolic and oxidative stress-related pathways. Oxidative stress plays a central role in the pathogenesis of both conditions, driving chronic inflammation, cellular damage, and fibrosis in the prostate and liver tissues. Conventional therapies for BPH primarily target androgenic and smooth muscle pathways but fail to address the systemic oxidative-inflammatory milieu exacerbated by hepatic impairment. This review explores the emerging concept of dual-targeted antioxidant therapies designed to simultaneously modulate oxidative stress and inflammation in both the prostate and liver. We examine the molecular underpinnings of the liver-prostate axis, discuss key phytochemicals and micronutrients with dual-organ protective properties, and highlight novel delivery systems such as nanoparticles and liposomes for enhanced bioavailability. The review further outlines therapeutic strategies integrating antioxidants with hepatoprotective and prostate-specific agents, providing a systems medicine approach to managing patients with concurrent urological and hepatic complications.

Keywords: Benign Prostatic Hyperplasia; Liver Dysfunction; Oxidative Stress; Dual-Targeted Therapy; Antioxidant Strategies

INTRODUCTION

Benign Prostatic Hyperplasia (BPH) is a highly prevalent non-cancerous condition characterized by the progressive enlargement of the prostate gland, primarily affecting aging men [1]. Clinically, BPH manifests as lower urinary tract symptoms (LUTS), including urinary frequency, nocturia, hesitancy, weak stream, and incomplete bladder emptying, which can significantly impair quality of life and increase healthcare costs [2]. Epidemiological studies suggest that over 50% of men above 50 years of age are affected, with prevalence rising to nearly 90% in men over 80 years [3]. Parallel to the increasing burden of BPH, liver dysfunction — particularly non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH) — has emerged as a global health challenge [4]. NAFLD affects approximately 25–30% of the global population and is closely linked to metabolic syndrome, obesity, insulin resistance, and systemic oxidative stress [5]. Historically, BPH and liver disorders have been managed as distinct clinical entities. However, recent insights into their overlapping pathophysiological mechanisms have unveiled a common denominator — oxidative stress and chronic low-grade inflammation. Oxidative stress occurs when the generation of reactive oxygen species (ROS) exceeds the capacity of the body's antioxidant defense systems, leading to molecular damage, lipid peroxidation, DNA fragmentation, and fibrosis in target tissues such as the prostate and liver [6]. Furthermore, the liver plays a pivotal role in maintaining systemic antioxidant homeostasis by metabolizing, detoxifying, and distributing endogenous and exogenous antioxidants [7]. In scenarios of hepatic dysfunction, these metabolic processes are impaired, compromising the bioavailability and therapeutic efficacy of antioxidants in peripheral organs, including the prostate [8]. Given this intricate interrelationship, dual-targeted antioxidant therapies that simultaneously modulate oxidative stress in both the liver

and prostate have gained significant attention. This review explores the molecular basis of the liver-prostate axis, identifies antioxidants with dual-organ protective effects, and discusses novel therapeutic strategies rooted in systems biology for integrated management of BPH and liver dysfunction.

Oxidative Stress in the Pathogenesis of BPH and Liver Dysfunction

Oxidative stress (OS) has emerged as a central molecular mechanism contributing to the initiation, progression, and complications of both benign prostatic hyperplasia (BPH) and liver dysfunction [6]. It plays a critical role in driving chronic inflammation, tissue remodeling, fibrosis, and metabolic disturbances in these conditions, which often coexist in aging males with underlying metabolic syndrome [9]. Understanding the role of oxidative stress in these pathologies is essential for designing dual-targeted therapeutic strategies. Oxidative stress is defined as a state of imbalance between the production of reactive oxygen species (ROS) and the capacity of antioxidant defense systems to neutralize them [10]. ROS, including superoxide anion (O_2^-), hydrogen peroxide (H_2O_2), and hydroxyl radicals (•OH), are generated as natural by-products of cellular metabolism, particularly in mitochondria [6]. However, when produced in excess, ROS damage cellular macromolecules, including DNA, proteins, and lipids, leading to cytotoxicity, apoptosis, and chronic tissue injury [11].

Oxidative Stress in BPH Pathogenesis

In the prostate gland, oxidative stress contributes significantly to the hyperplastic processes characteristic of BPH [12]. Aging-associated decline in antioxidant defense mechanisms predisposes prostatic tissues to oxidative damage [13]. ROS activate several redox-sensitive transcription factors, including nuclear factor-kappa B (NF-κB) and activator protein-1 (AP-1), which drive the expression of pro-inflammatory cytokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and cyclooxygenase-2 (COX-2) [14]. These cytokines create a chronic inflammatory microenvironment within the prostate, promoting stromal and epithelial cell proliferation, extracellular matrix deposition, and fibrosis [15]. Additionally, ROS facilitate the activation of transforming growth factor-beta (TGF-β), a potent pro-fibrotic cytokine that induces fibroblast activation and collagen synthesis, contributing to prostatic tissue remodeling and urethral obstruction [16]. Moreover, oxidative stress induces lipid peroxidation of cell membranes, mitochondrial dysfunction, and DNA damage within prostate cells, leading to increased cellular turnover and hyperplasia [17]. These pathological processes underpin the progression of BPH and the development of lower urinary tract symptoms (LUTS).

Oxidative Stress in Liver Dysfunction

Similarly, oxidative stress plays a pivotal role in the pathogenesis of liver disorders, particularly non-alcoholic fatty liver disease (NAFLD) and its progressive form, non-alcoholic steatohepatitis (NASH) [18]. In the liver, excess ROS production contributes to hepatocyte injury, lipid peroxidation, mitochondrial dysfunction, and activation of hepatic stellate cells, leading to fibrosis [19]. The initial stages of NAFLD involve hepatic lipid accumulation due to insulin resistance and dysregulated lipid metabolism [20]. The "two-hit hypothesis" of NAFLD pathogenesis proposes that lipid accumulation sensitizes hepatocytes to oxidative injury (the second hit), leading to inflammation, apoptosis, and fibrosis [18]. In NASH, oxidative stress exacerbates inflammatory signaling through the activation of NF-κB, TGF-β, and other pathways, promoting hepatic necroinflammation and fibrosis progression [21]. Markers of oxidative stress, such as malondialdehyde (MDA), 8-hydroxy-2′-deoxyguanosine (8-OHdG), and reduced glutathione (GSH), are commonly elevated in patients with liver disease [22].

Systemic Impact of Oxidative Stress: The Liver-Prostate Link

The liver-prostate axis is increasingly recognized as a critical systemic pathway linking hepatic dysfunction to BPH progression through oxidative stress and inflammation [23]. Hepatic injury results in the release of inflammatory cytokines, ROS, and acute-phase proteins, such as C-reactive protein (CRP), into the circulation [24]. These systemic factors can aggravate oxidative stress within the prostate, exacerbating prostatic hyperplasia, inflammation, and fibrosis. Additionally, liver dysfunction often leads to metabolic disturbances, including insulin resistance, hyperinsulinemia, and dyslipidemia, which are well-established risk factors for BPH [25]. Hyperinsulinemia enhances insulin-like growth factor-1 (IGF-1) activity, stimulating prostatic cell proliferation [26]. Dysregulated lipid metabolism further impacts androgen and estrogen levels, contributing to hormonal imbalances that drive BPH pathology [27].

Rationale for Targeting Oxidative Stress in Both Organs

Given the shared role of oxidative stress in the pathogenesis of both BPH and liver dysfunction, targeting ROS generation and enhancing antioxidant defenses offers a rational therapeutic approach for patients with dual organ involvement [28]. Antioxidant therapies have the potential to:

- Suppress inflammatory cytokine production [29.
- Inhibit fibrotic pathways [30].

- Reduce oxidative DNA damage and lipid peroxidation [31].
- Improve mitochondrial function and metabolic homeostasis [29,32].

Therapeutic strategies aimed at restoring redox balance in both the liver and prostate may not only attenuate disease progression but also improve quality of life and reduce complications associated with urological and hepatic disorders. Future therapies should focus on dual-targeted antioxidants that exert protective effects in both organs, as well as integrated management approaches that address systemic metabolic disturbances, inflammation, and gut-liver-prostate axis dysregulation.

Novel Strategies for Dual-Targeted Therapy

Emerging therapeutic strategies seek to optimize the efficacy of antioxidants while addressing the unique challenges posed by the liver-prostate axis.

Nanotechnology-Based Delivery Systems

Poor bioavailability and rapid hepatic metabolism limit the effectiveness of several antioxidants, such as curcumin and resveratrol [33,34]. Nanoparticles, liposomes, micelles, and polymer-based carriers have been developed to enhance the solubility, stability, and targeted delivery of these compounds to both the liver and prostate [35]. Such delivery systems improve cellular uptake, prolong circulation time, and enable site-specific release, maximizing therapeutic benefits while minimizing systemic side effects.

Combination Therapies

Combining antioxidants with other pharmacological agents provides a synergistic approach to managing dual organ dysfunction. For example, integrating hepatoprotective agents like silymarin with prostate-specific drugs such as alpha-blockers or 5-alpha-reductase inhibitors may offer additive or even synergistic benefits [36]. This strategy addresses both the hepatic and urological components of the disease, improving patient outcomes.

Gut Microbiota Modulation

The gut microbiota plays a critical role in modulating systemic oxidative stress, inflammation, and metabolic homeostasis [37]. Dysbiosis has been linked to both NAFLD and BPH [38,39]. Probiotics, prebiotics, and synbiotics can restore gut microbial balance, reducing the translocation of endotoxins, enhancing hepatic antioxidant defenses, and modulating hormonal pathways that influence prostate health [40]. Future research should explore the gut-liver-prostate axis as a novel therapeutic target.

Future Directions

Future research should prioritize the advancement of dual-targeted antioxidant therapies that can simultaneously manage benign prostatic hyperplasia (BPH) and liver dysfunction, given their interconnected pathophysiology. Well-designed clinical trials evaluating the safety, efficacy, and optimal dosing of antioxidant regimens in patients with coexisting BPH and hepatic disorders are urgently needed. These trials should incorporate comprehensive assessments of liver function markers (ALT, AST, GGT, liver fibrosis scores) alongside prostate-specific outcomes (prostate volume, symptom scores, PSA levels), ensuring a holistic evaluation of treatment responses. Another important future direction lies in the application of systems pharmacology modeling to predict patient-specific responses to dual-targeted therapies. By integrating computational tools, pharmacokinetic-pharmacodynamic data, and multi-omics profiling (genomics, proteomics, metabolomics), researchers can simulate and optimize antioxidant treatment regimens tailored to individual patient characteristics, including their liver metabolic capacity and prostate pathology. Moreover, the identification and validation of liver-prostate axis-specific biomarkers will enable precision medicine approaches in the management of these comorbidities. Potential biomarkers could include circulating oxidative stress markers (MDA, 8-OHdG), inflammatory cytokines, bile acid profiles, and gut microbiota-derived metabolites, providing clinicians with tools for early diagnosis, risk stratification, and therapeutic monitoring. Dual-targeted antioxidant therapies represent a novel and promising paradigm in the integrated management of benign prostatic hyperplasia (BPH) and liver dysfunction. The intricate interplay between the liver and prostate, mediated by oxidative stress, chronic inflammation, hormonal dysregulation, and metabolic disturbances, necessitates a holistic therapeutic approach that addresses both organ systems simultaneously. Conventional monotherapies often overlook the systemic nature of BPH pathogenesis, particularly in patients with underlying hepatic impairment. By leveraging antioxidants with dual hepatoprostatic benefits—such as lycopene, curcumin, resveratrol, and silymarin—clinicians can modulate redox homeostasis, suppress inflammatory pathways, and ameliorate tissue remodeling in both the liver and prostate. Emerging therapeutic strategies, including nanotechnology-based delivery systems, combination pharmacotherapy, and gut microbiota modulation, further enhance the potential of antioxidant interventions by improving bioavailability, targeting specific metabolic pathways, and restoring systemic homeostasis. The integration of these strategies into clinical practice offers an opportunity to improve therapeutic outcomes, minimize drug-related side effects, and address the multifaceted nature of urological and hepatic comorbidities. Moving forward, personalized medicine approaches guided by

systems pharmacology modeling, biomarker profiling, and individualized treatment planning will be essential to optimize the use of dual-targeted antioxidants. Additionally, recognizing the role of the gut-liver-prostate axis opens new avenues for microbiome-targeted interventions, which may complement antioxidant therapy and further improve patient outcomes.

CONCLUSION

Finally, exploring the gut-liver-prostate axis represents an innovative frontier in the integrated management of BPH and liver dysfunction. Interventions targeting gut microbiota, such as probiotics, prebiotics, synbiotics, and dietary modifications, have the potential to modulate systemic inflammation, oxidative stress, and metabolic balance, thereby enhancing antioxidant therapeutic efficacy. Future research should focus on elucidating the specific gut microbial signatures associated with BPH and liver diseases, paving the way for microbiome-guided antioxidant therapies. Dual-targeted antioxidant therapies hold significant promise in revolutionizing the management of patients with BPH and liver dysfunction. Future research and clinical translation of these strategies will contribute to the development of integrated, patient-centered care models aimed at enhancing quality of life and reducing the burden of urological and hepatic complications in aging populations.

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