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The Intersection of Benign Prostatic Hyperplasia and Reproductive Hormones: Oxidative Stress, Inflammation, and Immunomodulatory Pathways

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ABSTRACT

Benign prostatic hyperplasia (BPH) is one of the most prevalent urological disorders in aging men, characterized by prostate gland enlargement, lower urinary tract symptoms, and impaired quality of life. While androgen-driven growth has traditionally been emphasized in its pathogenesis, emerging evidence highlights the intricate interplay between reproductive hormones, oxidative stress, inflammation, and immunomodulation. Hormonal imbalances involving androgens, estrogens, and gonadotropins regulate stromal-epithelial interactions, driving cellular proliferation and remodeling. Concurrently, oxidative stress induces lipid peroxidation, DNA damage, and cellular senescence within the prostate microenvironment. Chronic low-grade inflammation further amplifies disease progression, recruiting immune cells and perpetuating a cycle of cytokine release, growth factor activation, and fibrotic remodeling. Immunomodulatory pathways, including T-cell polarization and innate immune responses, have been increasingly implicated in the chronicity of BPH. This review synthesizes current evidence on the hormonal, oxidative, and immune-mediated drivers of BPH, discusses therapeutic implications, and highlights future directions for integrative interventions targeting both endocrine and immunological pathways.

Keywords: Benign prostatic hyperplasia, reproductive hormones, oxidative stress, inflammation, immunomodulation

INTRODUCTION

Benign prostatic hyperplasia (BPH) is one of the most common nonmalignant urological disorders in aging men and represents a leading cause of lower urinary tract symptoms (LUTS) [1]. The condition increases in prevalence with advancing age, affecting nearly half of men over 50 years and up to 90 percent of men by the eighth decade of life. The clinical manifestations of BPH include both obstructive and irritative urinary symptoms such as urinary hesitancy, weak stream, incomplete bladder emptying, nocturia, and increased frequency [2]. These symptoms can severely impair quality of life, limiting social and occupational activities, and in advanced cases, may lead to complications including recurrent urinary tract infections, bladder stones, and even renal dysfunction due to chronic obstruction. For decades, the development of BPH was viewed primarily through the lens of androgen action, particularly the role of dihydrotestosterone (DHT) [3]. DHT, derived from testosterone by the enzyme 5α-reductase, exerts potent effects on prostatic epithelial and stromal cells, promoting cellular proliferation and glandular growth [4]. While androgen signaling is undoubtedly central to prostate physiology, emerging research demonstrates that BPH is not a simple consequence of androgen excess [5]. Instead, it is now understood as a multifactorial condition arising from the intersection of hormonal imbalances, oxidative stress, chronic inflammation, and immune dysregulation. These factors act synergistically to create a microenvironment that

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sustains progressive enlargement and tissue remodeling. Aging further complicates this process. With age, the testosterone-to-estrogen ratio shifts, with relative increases in estrogenic influence on the prostate [6]. At the same time, systemic oxidative stress increases due to diminished antioxidant defenses and heightened reactive oxygen species (ROS) production. ROS contribute to DNA damage, mitochondrial dysfunction, and cellular senescence, which in turn drive abnormal proliferation and tissue remodeling [7]. Chronic low-grade inflammation, often fueled by immune cell infiltration and persistent cytokine release, amplifies these processes and establishes a self-perpetuating cycle of growth and repair [7]. The recognition of BPH as a condition shaped by interconnected endocrine, oxidative, and immunological mechanisms has important implications for therapy [8]. Conventional treatments such as alpha-adrenergic blockers and 5α-reductase inhibitors remain mainstays of management, offering symptom relief and partial modulation of androgen pathways [9]. However, these therapies do not fully address the broader pathogenic landscape that includes oxidative and inflammatory drivers of disease. Future strategies must move beyond isolated pathways to incorporate the complexity of BPH pathophysiology. This review therefore, examines the role of reproductive hormones, oxidative stress, inflammation, and immunomodulatory mechanisms in the development and progression of BPH. By focusing on these intersecting processes, it seeks to provide a more comprehensive understanding of disease mechanisms and highlight emerging avenues for integrated therapeutic interventions.

2. Pathophysiology of Benign Prostatic Hyperplasia

The pathophysiology of BPH is complex and multifaceted, reflecting the dynamic interplay of endocrine regulation, local tissue interactions, oxidative imbalance, and immune system activity [1,5,6,7]. At the anatomical level, BPH is characterized by progressive enlargement of the transitional zone of the prostate, which surrounds the urethra [1]. This expansion leads to mechanical obstruction and contributes to bladder outlet obstruction, the primary cause of LUTS in affected men [10]. A central feature of BPH is cellular proliferation [11]. Both epithelial and stromal compartments exhibit hyperplasia, leading to nodular growth and disruption of normal tissue architecture [12]. Stromal cells, particularly fibroblasts and smooth muscle cells, interact with epithelial cells through paracrine signaling [13]. Growth factors such as fibroblast growth factor (FGF) and transforming growth factor-beta (TGF-β) are upregulated, driving proliferation and extracellular matrix deposition [14].

Fibrotic remodeling is another hallmark of BPH. Excessive deposition of extracellular matrix proteins, including collagen, increases tissue stiffness and alters prostate structure [15,16]. Fibrosis not only supports hyperplastic expansion but also contributes to urethral compression and impaired bladder function [17]. Angiogenesis further sustains the growth of hyperplastic tissue by ensuring oxygen and nutrient delivery [18]. However, the newly formed blood vessels are often structurally abnormal and inefficient, creating regions of local hypoxia [19]. Hypoxic stress in turn stimulates oxidative stress and inflammatory signaling, perpetuating pathological remodeling [20].

Chronic inflammation is consistently observed in histological studies of BPH tissue [21]. Infiltration by macrophages, lymphocytes, and mast cells generates a proinflammatory microenvironment [22]. These immune cells release cytokines such as interleukin-6, interleukin-8, and tumor necrosis factor-alpha, which stimulate stromal proliferation, angiogenesis, and matrix remodeling [22]. Activation of transcription factors such as nuclear factor-kappa B (NF-κB) sustains inflammatory signaling and amplifies cellular responses [23]. Oxidative stress further exacerbates these pathological changes. Reactive oxygen species induce DNA damage, lipid peroxidation, and protein oxidation, impairing cellular function and promoting senescence [7]. Importantly, oxidative stress also acts as a signaling mechanism, activating pathways that overlap with inflammatory and fibrotic responses [24]. This creates a direct link between redox imbalance, immune activation, and tissue remodeling. Taken together, the pathophysiology of BPH is best understood as a convergence of processes. Hormonal dysregulation provides the initiating stimulus, oxidative stress accelerates damage and genomic instability, and chronic inflammation sustains a cycle of repair and proliferation. These mechanisms operate in concert to produce prostate enlargement, urethral compression and the symptomatic burden associated with BPH

3. Role of Reproductive Hormones in BPH

Reproductive hormones are fundamental to the growth, maintenance, and pathological enlargement of the prostate. Among them, androgens, estrogens, gonadotropins, and prolactin work in interconnected ways to regulate cell proliferation, differentiation, and communication between stromal and epithelial compartments [25]. With advancing age, imbalances in these hormones create conditions that favor the onset and progression of benign prostatic hyperplasia (BPH) [6]. Androgens are central to prostate physiology. Testosterone, synthesized primarily in the testes, enters prostate cells where it is converted to dihydrotestosterone (DHT) through the action of 5α-reductase [26]. DHT binds to androgen receptors (AR) in both stromal and epithelial cells with higher affinity than

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testosterone, triggering transcription of genes responsible for proliferation, secretory activity, and tissue remodeling [27]. In BPH, excessive AR activation leads to uncontrolled cell growth and architectural distortion of the gland [28]. This effect is reinforced through cross-talk between androgen signaling and growth factors such as fibroblast growth factor and insulin-like growth factor, amplifying the hyperplastic process.

Estrogens also influence prostate dynamics, particularly in older men, where a shift in the testosterone-to-estrogen ratio occurs [6]. Estrogen receptor alpha (ERα) activation stimulates stromal proliferation, fibrotic remodeling, and inflammatory responses that favor disease progression [29]. In contrast, estrogen receptor beta (ERβ) counterbalances these effects by inducing apoptosis and inhibiting proliferation, but its protective influence may diminish with age [30]. Dysregulated estrogen signaling, therefore, creates a hormonal imbalance that tilts toward growth promotion and inflammation.

Beyond androgens and estrogens, hormones such as luteinizing hormone (LH), follicle-stimulating hormone (FSH), and prolactin have emerged as important modulators of prostate activity. LH and FSH, classically involved in gonadal regulation, may alter prostate tissue sensitivity to androgens [31]. Prolactin enhances androgen receptor responsiveness and influences local cytokine release, thereby reinforcing proliferative and inflammatory changes [32]. Elevated prolactin levels have been correlated with a greater risk of prostate enlargement, further underscoring its role in disease pathogenesis [33].

4. Oxidative Stress in BPH

Oxidative stress is another major factor in BPH pathophysiology [7]. It occurs when the production of reactive oxygen species (ROS) overwhelms the body's antioxidant defense mechanisms [34]. The prostate is particularly vulnerable because of its high metabolic activity and age-related declines in enzymes such as superoxide dismutase, catalase, and glutathione peroxidase [35]. ROS induce multiple forms of cellular damage [7]. DNA damage is a primary consequence, with strand breaks, mutations, and mitochondrial dysfunction contributing to genomic instability and cellular senescence. Lipid peroxidation further disrupts membrane integrity and signaling, producing reactive aldehydes such as malondialdehyde that exacerbate fibrosis and inflammatory signaling [36]. In addition, protein oxidation alters the structure and function of regulatory proteins, impairing apoptosis, enzymatic activity, and receptor function [37].

Beyond direct damage, oxidative stress serves as a potent signaling mechanism. ROS activate transcription factors such as NF-κB and AP-1, which stimulate proinflammatory cytokine release and attract immune cells into the prostate [38]. This creates a feedback loop where oxidative stress promotes inflammation, and inflammation, in turn, generates more ROS. Thus, oxidative stress is both a direct cause of cellular injury and an indirect amplifier of inflammation and fibrosis [39]. By linking aging, hormonal imbalance, and immune activation, oxidative stress emerges as a central driver of the chronic pathological cycle that sustains BPH progression.

5. Inflammation and Immunomodulatory Pathways

Chronic inflammation is increasingly recognized as a central hallmark of benign prostatic hyperplasia (BPH), serving as a link between hormonal imbalance, oxidative stress, and tissue remodeling [40]. Histological studies consistently demonstrate immune cell infiltration and elevated levels of proinflammatory mediators in hyperplastic prostates [41]. Cytokines and chemokines such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-8 (IL-8) drive stromal proliferation, angiogenesis, and extracellular matrix deposition [42]. These molecules also activate fibroblasts, reinforcing fibrotic remodeling. Immune cell infiltration further sustains disease progression, with T-cells, B-cells, macrophages, and mast cells contributing to chronic injury [43]. A predominance of Th1 and Th17 subsets promotes inflammation, while reduced regulatory T-cell activity weakens immune tolerance [44]. Macrophages add to this process by releasing proinflammatory mediators or profibrotic growth factors, depending on their polarization state. Innate immune mechanisms also participate. Pattern recognition receptors such as Toll-like receptors detect cellular stress and activate transcription factors like nuclear factor-kappa B (NF-κB), which amplify cytokine release and perpetuate cross-talk between innate and adaptive immunity [45]. Together, these immunomodulatory processes establish a self-perpetuating cycle of inflammation, fibrosis, and hyperplasia that sustains BPH progression.

6. Therapeutic Perspectives

Recognition of the hormonal–oxidative–immune axis in BPH has broadened therapeutic strategies beyond conventional symptom relief. Hormonal modulation remains a cornerstone, with 5α-reductase inhibitors lowering dihydrotestosterone and selective estrogen receptor modulators fine-tuning estrogenic effects [46]. Targeting oxidative stress is a promising adjunct approach. Natural compounds such as lycopene, resveratrol, and curcumin, as well as pharmacological antioxidants, are under investigation for reducing reactive oxygen species and their

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downstream consequences [47]. Anti-inflammatory options, including nonsteroidal anti-inflammatory drugs and phytotherapeutics like saw palmetto, have shown variable benefit [48]. More advanced interventions, such as cytokine-targeting biologics or T-cell regulatory therapies, represent emerging avenues of immunomodulation [49]. Ultimately, the most effective strategies may combine hormonal, antioxidant, and immune-targeted therapies to address the multifactorial drivers of BPH and improve long-term outcomes for affected men.

CONCLUSION

Benign prostatic hyperplasia is a complex disorder shaped not only by androgenic stimulation but also by oxidative stress, chronic inflammation, and immunomodulatory pathways. Reproductive hormones orchestrate a microenvironment in which oxidative and immune-mediated processes perpetuate hyperplasia. A deeper understanding of these interactions is crucial for developing integrated therapeutic strategies. Future research should focus on identifying novel biomarkers linking hormonal imbalance with oxidative and immune responses and testing multi-targeted therapies that can prevent progression and improve patient quality of life.

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