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Role of Inflammation and Oxidative Stress in Benign Prostatic Hyperplasia Progression

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ABSTRACT

Benign prostatic hyperplasia (BPH) is a common urological condition affecting aging men, characterized by the non-malignant enlargement of the prostate gland, which often leads to lower urinary tract symptoms (LUTS). While hormonal changes and age are recognized as primary risk factors, the roles of chronic inflammation and oxidative stress in the progression of BPH have garnered significant attention. Chronic inflammation contributes to prostatic tissue remodeling and fibrosis, driven by inflammatory cytokines, immune cell infiltration, and pro-inflammatory signaling pathways. Oxidative stress, resulting from an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, exacerbates cellular damage, amplifying inflammatory responses and promoting tissue dysfunction. This article explores the molecular mechanisms underlying inflammation and oxidative stress in BPH progression, highlights their interconnection, and discusses potential therapeutic strategies targeting these processes to mitigate disease progression and associated symptoms.

Keywords: Benign prostatic hyperplasia (BPH), inflammation, oxidative stress, reactive oxygen species (ROS), cytokines, prostate enlargement

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a highly prevalent condition among men aged 50 years and above, significantly impacting their quality of life due to its associated lower urinary tract symptoms (LUTS)[1–3]. These symptoms, which include urinary frequency, urgency, nocturia, weak stream, and incomplete bladder emptying, can lead to considerable discomfort and disruption of daily activities. BPH represents a progressive condition characterized by non-malignant enlargement of the prostate gland, which often results in bladder outlet obstruction.

The etiology of BPH involves a multifactorial interplay of hormonal imbalances, aging-related changes, and genetic predisposition. Elevated levels of dihydrotestosterone (DHT), alterations in androgen signaling, and age-associated reductions in testosterone contribute significantly to prostatic hyperplasia[4–6]. Additionally, genetic factors have been implicated in modulating susceptibility to BPH, although their precise roles remain under investigation. Recent advances in research have highlighted the critical contributions of chronic inflammation and oxidative stress to the pathogenesis and progression of BPH[7, 8] Chronic inflammation is associated with infiltrating immune cells, pro-inflammatory cytokines, and local tissue damage, creating a microenvironment conducive to abnormal cellular activities. Oxidative stress, resulting from an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, exacerbates inflammatory responses and promotes cellular senescence, epithelial-mesenchymal transition (EMT), fibrosis, and aberrant tissue remodeling within the prostate gland[8–10]. These mechanisms collectively contribute to prostatic enlargement and the worsening of LUTS over time.

Given their pivotal roles in BPH pathogenesis, chronic inflammation and oxidative stress present promising therapeutic targets for halting or reversing disease progression. This article aims to delve into the intricate mechanisms by which inflammation and oxidative stress drive BPH progression, emphasizing their synergistic effects, interplay with other pathogenic factors, and potential for innovative therapeutic interventions.

Inflammation in BPH Progression

Chronic Inflammatory Microenvironment

Histological studies of prostate tissues from patients with benign prostatic hyperplasia (BPH) consistently reveal the presence of chronic inflammatory infiltrates, indicating a significant role of the immune system in the pathophysiology of the disease. These infiltrates are predominantly composed of immune cells such as macrophages, T-

lymphocytes, and mast cells, which are strategically localized around hyperplastic nodules and within stromal and epithelial compartments of the prostate[11]. The activated macrophages serve as key effectors, releasing a broad spectrum of pro-inflammatory mediators that sustain chronic inflammation. T-lymphocytes, particularly CD4+ helper T cells and CD8+ cytotoxic T cells, contribute to the immune response by secreting cytokines and directly interacting with stromal and epithelial cells. Mast cells, often found in close proximity to blood vessels, facilitate angiogenesis and tissue remodeling through the release of histamine, proteases, and other bioactive substances[11].

These immune cells collectively secrete cytokines such as interleukin-6 (IL-6), tumor necrosis factor-α (TNF-α), and interleukin-1β (IL-1β), which act as pivotal drivers of pro-inflammatory signaling pathways. IL-6, for instance, activates the Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathway, promoting stromal cell proliferation and epithelial hyperplasia. TNF-α and IL-1β further amplify the inflammatory milieu by activating nuclear factor-kappa B (NF-κB) signaling, which regulates the expression of genes involved in cell survival, proliferation, and the recruitment of additional immune cells[12]. This persistent inflammatory environment not only contributes to the development and progression of BPH but also creates a permissive microenvironment for fibrosis and tissue remodeling. Furthermore, these cytokine-mediated pathways may influence hormonal signaling, including androgen receptor activity, thereby perpetuating the cycle of inflammation and hyperplasia. Understanding these mechanisms offers potential therapeutic targets to mitigate inflammation and its associated complications in BPH patients[13].

Cytokine-Mediated Prostatic Remodeling

Pro-inflammatory cytokines, such as interleukin-6 (IL-6), tumor necrosis factor-alpha (TNF-α), and interleukin-1 beta (IL-1β), play a central role in driving the pathological processes underlying prostate enlargement. These cytokines activate signaling pathways, particularly nuclear factor-κB (NF-κB) and signal transducer and activator of transcription 3 (STAT3), which are critical mediators of inflammation, cell proliferation, and survival[14].

When NF-κB is activated, it translocates to the nucleus, where it promotes the transcription of genes involved in cell cycle progression, anti-apoptotic proteins, and pro-inflammatory mediators. Similarly, STAT3, when phosphorylated, forms dimers that also translocate to the nucleus to induce the expression of genes that support cell proliferation and inhibit apoptosis. Together, these pathways create a pro-survival and pro-growth environment, enhancing the proliferation of both stromal and epithelial cells in the prostate[15]. Additionally, these cytokines stimulate the production of transforming growth factor-beta (TGF-β) and other profibrotic factors, which further drive fibrosis by inducing the activation of myofibroblasts and the deposition of extracellular matrix (ECM) proteins like collagen and fibronectin.[15] The accumulation of ECM disrupts the normal architecture of the prostate tissue, contributing to its enlargement and increased stiffness. This combination of uncontrolled cell growth, resistance to cell death, and ECM remodeling perpetuates a cycle of chronic inflammation and tissue remodeling, which is central to conditions like benign prostatic hyperplasia (BPH). Over time, these changes can exacerbate lower urinary tract symptoms (LUTS) due to mechanical compression of the urethra and impaired bladder function. Thus, targeting NF-κB and STAT3 signaling pathways or modulating cytokine activity presents a promising therapeutic strategy to manage prostate enlargement and associated symptoms[16, 17].

Immune Cell Recruitment and Activation

The recruitment of immune cells, such as macrophages, lymphocytes, and neutrophils, to the prostate gland creates a persistent inflammatory microenvironment. This sustained immune response generates a vicious cycle, as inflammatory mediators like cytokines (e.g., IL-6, TNF-α) and chemokines amplify immune cell infiltration and activation[18]. These immune cells, in turn, release additional pro-inflammatory molecules, reactive oxygen species (ROS), and proteolytic enzymes, perpetuating chronic inflammation. Over time, this inflammatory milieu induces tissue damage by promoting oxidative stress, disrupting epithelial integrity, and causing cellular apoptosis or necrosis. The resulting tissue injury activates compensatory repair mechanisms aimed at restoring tissue homeostasis. However, in the context of chronic inflammation, these repair processes become dysregulated. Fibroblasts, which are recruited and activated by growth factors such as TGF-β, undergo myofibroblast differentiation[19]. These activated fibroblasts deposit excessive amounts of extracellular matrix components, including collagen, leading to fibrotic remodeling of the prostate.

The progressive deposition of collagen and other matrix proteins results in prostatic fibrosis, which is characterized by increased tissue stiffness. This stiffening not only disrupts the normal architecture of the prostate but also exacerbates the mechanical stress on adjacent cells, further activating fibroblasts and perpetuating the cycle of inflammation and fibrosis. Prostatic fibrosis and tissue stiffening are hallmark features of benign prostatic hyperplasia (BPH) and contribute significantly to the clinical manifestations of the condition, such as lower urinary tract symptoms (LUTS) and bladder outlet obstruction (BOO)[20,

21]. Moreover, the altered microenvironment created by chronic inflammation and fibrosis may influence the progression of BPH by promoting epithelial and stromal cell proliferation, angiogenesis, and hormonal imbalances, further complicating disease management and treatment outcomes.

Oxidative Stress in BPH Progression

Sources of Oxidative Stress

Oxidative stress arises from an imbalance between the production of reactive oxygen species (ROS) and the body's ability to counteract or detoxify their harmful effects through antioxidant defenses. ROS are highly reactive molecules that include free radicals such as superoxide anion (O_2^-), hydroxyl radical (OH), and non-radical species like hydrogen peroxide (H_2O_2). These molecules are produced as natural byproducts of cellular metabolism, particularly during mitochondrial oxidative phosphorylation[22]. Mitochondrial dysfunction plays a pivotal role in excessive ROS production. In normal conditions, the electron transport chain (ETC) tightly couples the transfer of electrons to molecular oxygen, generating water. However, when mitochondrial function is impaired, electrons leak from the ETC and prematurely react with oxygen, leading to the formation of superoxide radicals[23].

In addition to mitochondria, other cellular sources contribute to ROS production. Nicotinamide adenine dinucleotide phosphate (NADPH) oxidases (NOX enzymes) are specialized enzymes whose primary function is to generate ROS. These enzymes are activated in response to various stimuli, including cytokines, growth factors, and cellular stress, contributing to oxidative stress in pathological conditions. Similarly, xanthine oxidase, an enzyme involved in purine metabolism, generates superoxide and hydrogen peroxide as byproducts, particularly under hypoxic or ischemic conditions. Environmental and lifestyle factors exacerbate ROS generation, overwhelming the antioxidant defense system[23]. A poor diet, especially one rich in processed foods, high sugars, and trans fats, can increase ROS levels due to pro-inflammatory responses and metabolic disturbances. Smoking introduces a wide range of exogenous free radicals and pro-oxidant chemicals, further amplifying oxidative stress. Metabolic syndrome, characterized by central obesity, insulin resistance, hyperglycemia, dyslipidemia, and hypertension, is another critical contributor. In metabolic syndrome, chronic low-grade inflammation and hyperglycemia promote the activation of ROS-generating enzymes, mitochondrial dysfunction, and glycation reactions, creating a vicious cycle of oxidative stress[24, 25]. The cumulative effects of these factors can lead to cellular damage, including lipid peroxidation, protein oxidation, and DNA damage, ultimately contributing to the pathogenesis of numerous diseases such as cardiovascular disorders, neurodegenerative diseases, cancer, and diabetes. Understanding these mechanisms underscores the importance of maintaining mitochondrial health, minimizing exposure to environmental risk factors, and bolstering antioxidant defenses through diet and lifestyle interventions.

ROS-Induced Cellular Damage

Reactive oxygen species (ROS) directly damage critical cellular components, including lipids, proteins, and DNA, compromising cellular integrity and functionality. This oxidative damage contributes to the onset of cellular senescence, a state of irreversible cell cycle arrest, and apoptosis, a programmed cell death mechanism. Beyond direct molecular damage, ROS-induced oxidative stress exacerbates inflammatory responses. It achieves this by activating redox-sensitive transcription factors such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) and activator protein-1 (AP-1)[26]. These transcription factors play pivotal roles in regulating the expression of genes involved in the inflammatory response, including pro-inflammatory cytokines and chemokines. Consequently, this heightened inflammatory state perpetuates a vicious cycle of cellular damage and dysfunction, further contributing to pathological conditions such as aging, neurodegeneration, and chronic inflammatory diseases[27].

Interaction with Inflammation

Oxidative stress and inflammation are closely linked biological processes that often coexist and amplify one another in pathological conditions. Reactive oxygen species (ROS), which are generated during oxidative stress, play a pivotal role in activating inflammatory pathways such as the nuclear factor-kappa B (NF-κB) signaling cascade. Activation of NF-κB leads to the upregulation of pro-inflammatory cytokines, chemokines, and enzymes like cyclooxygenase-2 (COX-2) and inducible nitric oxide synthase (iNOS). These inflammatory mediators further recruit immune cells to the site of damage, which in turn produce additional ROS, perpetuating oxidative stress[28].

Conversely, chronic inflammation creates a microenvironment rich in pro-inflammatory cytokines (e.g., tumor necrosis factor-alpha [TNF-α], interleukin-6 [IL-6]) and other reactive intermediates that stimulate excessive ROS production. This overproduction overwhelms the cellular antioxidant defense systems, including enzymes like superoxide dismutase (SOD), catalase, and glutathione peroxidase, leading to an accumulation of oxidative damage. In the context of prostate tissue, this bidirectional relationship forms a feed-forward loop that perpetuates tissue damage and dysfunction[28]. ROS-induced oxidative damage affects lipids, proteins, and DNA, leading to cellular senescence, apoptosis, or malignant transformation. In parallel, the chronic inflammatory milieu contributes to the disruption

of normal prostate tissue architecture and function. This vicious cycle is particularly relevant in conditions like benign prostatic hyperplasia (BPH) and prostate cancer, where oxidative stress and inflammation are key drivers of disease progression. Understanding this interplay provides a foundation for therapeutic strategies targeting either oxidative stress or inflammation—or both—to break the cycle and mitigate prostate tissue damage.

Interplay Between Inflammation and Oxidative Stress

The interplay between inflammation and oxidative stress establishes a vicious cycle that significantly contributes to the progression of benign prostatic hyperplasia (BPH). In this context, reactive oxygen species (ROS) act as critical signaling molecules, driving the activation of transcription factors such as nuclear factor kappa B (NF-κB) and signal transducer and activator of transcription 3 (STAT3)[29]. This activation upregulates the expression of pro-inflammatory mediators, including interleukins (e.g., IL-6 and IL-8), tumor necrosis factor-alpha (TNF-α), and cyclooxygenase-2 (COX-2), which sustain a chronic inflammatory state within the prostate tissue. Simultaneously, this pro-inflammatory milieu amplifies oxidative stress through increased production of ROS and a concomitant reduction in antioxidant defense mechanisms. Cytokines like TNF-α and IL-1β further exacerbate oxidative stress by stimulating the activity of NADPH oxidase and other ROS-generating enzymes, leading to an accumulation of oxidative damage. The excessive ROS not only damage cellular components such as lipids, proteins, and DNA but also promote tissue remodeling by activating matrix metalloproteinases (MMPs) and other factors that degrade the extracellular matrix[30]. This sustained oxidative and inflammatory environment contributes to epithelial and stromal cell proliferation, fibrosis, and angiogenesis, all of which are hallmark features of BPH progression. Moreover, ROS-induced activation of STAT3 and NF-κB reinforces the production of inflammatory cytokines, creating a feedback loop that perpetuates tissue damage and cellular dysfunction. Together, these processes establish a microenvironment that favors hyperplasia and obstructive symptoms characteristic of BPH, highlighting the importance of targeting oxidative stress and inflammation in therapeutic strategies.

Therapeutic Implications

Anti-Inflammatory Therapies

Targeting inflammation through the use of non-steroidal anti-inflammatory drugs (NSAIDs), cytokine inhibitors, and immune modulators holds promise for mitigating BPH progression. For instance, IL-6 and TNF-α inhibitors have shown potential in reducing prostatic inflammation and fibrosis.

Antioxidant Strategies

Antioxidants such as vitamin E, selenium, and polyphenols can counteract oxidative stress, preserving cellular function and reducing inflammation. Emerging therapies targeting oxidative stress pathways, including NADPH oxidase inhibitors, are being explored in preclinical studies.

Combination Approaches

Combining anti-inflammatory and antioxidant strategies presents a promising therapeutic avenue for managing benign prostatic hyperplasia (BPH) by targeting its dual contributors: inflammation and oxidative stress. Chronic inflammation within the prostate gland is a well-documented driver of tissue remodeling, stromal proliferation, and glandular hyperplasia, all of which contribute to the progression of BPH. Similarly, oxidative stress, resulting from an imbalance between reactive oxygen species (ROS) production and the antioxidant defense system, exacerbates cellular damage, promotes inflammatory cascades, and accelerates prostatic tissue growth. By integrating anti-inflammatory agents that suppress pro-inflammatory pathways (e.g., COX-2 inhibitors, cytokine blockers) with antioxidants that neutralize ROS and reduce oxidative damage (e.g., polyphenols, vitamins C and E), combination therapies can simultaneously address these interconnected pathological mechanisms. This dual approach has the potential to alleviate lower urinary tract symptoms (LUTS) more effectively by reducing prostate volume, improving bladder function, and mitigating inflammation-induced urinary obstruction.

Moreover, such combination therapies could slow the progression of BPH more efficiently than monotherapies by targeting multiple pathways involved in disease development. For example, natural compounds like curcumin and resveratrol exhibit both anti-inflammatory and antioxidant properties, making them ideal candidates for combination regimens. Future research focusing on the synergistic effects of these strategies, optimal dosing, and long-term safety profiles could pave the way for novel, multi-targeted treatments that improve patient outcomes and quality of life.

CONCLUSION

Inflammation and oxidative stress play central roles in the pathogenesis and progression of BPH, contributing to tissue remodeling, fibrosis, and prostate enlargement. Understanding the molecular mechanisms underlying these processes provides a basis for developing targeted therapeutic interventions. By addressing the interconnected pathways of inflammation and oxidative stress, novel treatments can improve outcomes for men suffering from BPH and its associated symptoms.

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