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Cytokine Networks and Prostate Growth: Immune Mechanisms in the Development of BPH

Tom Robert

Department of Clinical Medicine and Dentistry, Kampala International University Uganda
Email: robert.tom@studwc.kiu.ac.ug

ABSTRACT

Benign prostatic hyperplasia (BPH) is a highly prevalent, age-associated condition characterized by nonmalignant enlargement of the prostate and a spectrum of lower urinary tract symptoms (LUTS). While androgen signaling and stromal–epithelial interactions are classic drivers of BPH, a large body of evidence now supports chronic, low-grade inflammation as a coequal engine of prostate growth and symptom progression. Inflammatory infiltrates in BPH tissue, typically macrophages, T cells, mast cells, and occasional neutrophils, create a cytokine-rich microenvironment that sustains epithelial and stromal proliferation, extracellular matrix (ECM) remodeling, angiogenesis, and smooth muscle hypercontractility. Cytokine networks rather than single mediators appear to determine biological outcomes: pro-growth and pro-fibrotic axes (eg, IL-6/STAT3, IL-8/CXCR signaling, TNF-α/NF-κB, IL-17–driven amplification loops, and TGF-β–mediated myofibroblast activation) intersect with counter-regulatory pathways (eg, IL-10, TGF-β immunoregulation, and specialized pro-resolving mediators) that may be insufficient or dysregulated in progressive disease. These immune signals interact with endocrine aging, metabolic syndrome, oxidative stress, local hypoxia, and recurrent infection or tissue injury, producing a "wound that does not heal" phenotype in the transitional zone. This review synthesizes current concepts on how cytokine networks shape the BPH microenvironment, linking immune cell recruitment and activation to prostate enlargement, fibrosis, and LUTS, and highlights emerging biomarker and therapeutic opportunities targeting inflammation-driven prostate growth.

Keywords: Benign prostatic hyperplasia; Cytokines; Chronic inflammation; Stromal-epithelial crosstalk; Fibrosis

INTRODUCTION

BPH arises primarily in the periurethral/transitional zone and reflects a shift in tissue homeostasis toward net growth driven by increased cellular proliferation, reduced apoptosis, altered differentiation, and progressive ECM deposition [1]. Clinically, BPH contributes to LUTS (frequency, urgency, nocturia, weak stream) via both a static component (increased tissue bulk narrowing the urethral lumen) and a dynamic component (smooth muscle tone regulated by autonomic and local signaling) [2]. Not all enlarged prostates cause severe symptoms, and LUTS severity does not perfectly correlate with prostate volume, suggesting multiple biological layers, including immune and neuromuscular factors [3]. Inflammation is common in BPH tissue and correlates with larger prostate volumes, faster growth, and worse symptoms in many cohorts [4]. Rather than being merely a bystander, inflammation can remodel prostate structure and function [5]. In particular, cytokine networks-interconnected pro- and anti-inflammatory mediators produced by immune cells, stromal fibroblasts, epithelial cells, and endothelium-act as a "communication grid" that integrates injury signals and instructs growth responses [6].

2. The BPH Immune Microenvironment: Cells and Triggers

2.1 Immune cell composition

Histologic BPH frequently contains focal or diffuse inflammatory infiltrates that reshape the local tissue environment and support prostate growth [7]. Macrophages are among the most abundant immune cells and can adopt pro-inflammatory or reparative phenotypes, releasing mediators such as TNF-α, IL-1β, IL-6, and TGF-β [8]. Through these signals, macrophages promote stromal proliferation, angiogenesis, and extracellular matrix remodeling, and they also contribute to fibrotic change that can worsen urethral compression. T lymphocytes are also prominent, including CD4+ and CD8+ populations as well as functionally distinct subsets such as Th17 and regulatory T cells[9]. These cells produce cytokines like IFN-γ, IL-17, and IL-10, influencing immune polarization and determining whether inflammation becomes persistent[10]. Mast cells add another layer by releasing histamine, tryptase, leukotrienes, and cytokines, which can enhance smooth muscle tone, amplify irritative symptoms, and sensitize local nerves. Neutrophils appear variably but can intensify tissue injury through reactive oxygen species and proteases while reinforcing recruitment loops via IL-8-related pathways[11]. In some cases, B cells and plasma cells are present and may sustain chronicity through antigen presentation and antibody production, especially when ongoing immune stimulation persists.

2.2 Triggers sustaining chronic inflammation

The inflammatory phenotype of BPH likely arises from multiple converging triggers [12]. Aging contributes to systemic low-grade inflammation that primes the prostate for chronic immune activation. Recurrent tissue injury, microtrauma, or ischemia can drive repeated damage-repair cycles that keep cytokine programs switched on [13]. Infection, prostatitis, or occult microbial signals can activate innate immune pathways through pattern-recognition receptors [14]. Metabolic syndrome and adipokine imbalance increase circulating inflammatory mediators and oxidative stress, amplifying local cytokine output [15]. Hormonal shifts in androgen and estrogen signaling may further modulate immune recruitment and responsiveness [16]. Hypoxia and oxidative stress activate transcriptional hubs such as NF-κB, STAT3, and AP-1, sustaining cytokine production and linking inflammation to hyperplasia and fibrosis.

3. Pro-fibrotic and Remodeling Cytokines: From Growth to Obstruction

3.1 TGF-β: fibrosis, stiffness, and myofibroblasts

TGF-β is a central driver of fibrotic remodeling in BPH and helps convert inflammation into long-term structural change [17]. Although TGF-β can suppress excessive immune activation in some contexts, its dominant effect in the prostatic stroma is to push fibroblasts toward an activated, matrix-producing state[18]. Under sustained TGF-β signaling, stromal fibroblasts differentiate into myofibroblasts that secrete large amounts of collagen and other extracellular matrix components, increasing tissue density and reducing compliance[19]. At the same time, TGF-β shifts the balance of matrix turnover by altering metalloproteinases and their inhibitors, favoring matrix accumulation rather than degradation. The resulting fibrosis is not simply a passive scar; it changes biomechanics and cell behavior[20]. Stiffer periurethral tissue can narrow the urethral lumen and can also amplify contractile responses of nearby smooth muscle, worsening the dynamic component of obstruction[21]. Fibrotic remodeling may also disrupt glandular architecture and local microvascular perfusion, reinforcing hypoxia and oxidative stress that further sustain cytokine production. In this way, TGF-β acts as a bridge between chronic inflammation and progressive, functionally important obstruction[22].

3.2 PDGF, FGF, VEGF, and growth factor–cytokine crosstalk

PDGF, FGF, and VEGF are frequently elevated in inflamed prostate tissue and operate alongside cytokines to support hyperplastic growth [23]. PDGF promotes stromal cell proliferation and recruitment of pericyte-like cells, strengthening the stromal compartment and facilitating nodular expansion [24]. FGF family members can stimulate both epithelial and stromal proliferation while enhancing survival pathways that favor net growth. VEGF promotes angiogenesis, increasing blood supply to expanding tissue and enabling continued remodeling [25]. These mediators are often induced by inflammatory cytokines such as TNF-α, IL-1β, and IL-6, and also by hypoxia-related pathways [26]. Newly formed vessels and activated endothelium, in turn, enhance leukocyte trafficking into the prostate, sustaining inflammation. This reciprocal interaction creates a self-reinforcing loop in which cytokine-driven inflammation promotes growth factor signaling, and growth factor–mediated angiogenesis and remodeling further support immune cell recruitment and persistent tissue expansion[27].

4. Anti-Inflammatory and Resolution Pathways: Why They May Fail

4.1 IL-10 and regulatory T cell restraint.

Anti-inflammatory control in the prostate depends heavily on IL-10 and regulatory T cells, which act as brakes on excessive immune activation [28]. IL-10 suppresses the production of key pro-inflammatory cytokines, reduces antigen-presenting cell activation, and limits collateral tissue injury that would otherwise perpetuate danger

signals. Regulatory T cells extend this restraint by suppressing effector T-cell responses and moderating macrophage activation, thereby reducing the intensity and duration of inflammatory cascades [29]. In progressive BPH, however, these regulatory mechanisms may become insufficient. One reason is that triggering inputs can be continuous or recurrent, including occult infection, metabolic inflammation, and repeated micro-injury, each repeatedly re-initiating cytokine programs faster than regulation can shut them down [30]. Another reason is functional imbalance: Tregs may be present in tissue but impaired in suppressive capacity due to local cytokine polarization, altered metabolism, or reduced responsiveness to regulatory cues. Finally, stromal-derived pro-growth and pro-fibrotic signaling can become partially autonomous [32]. Once fibroblasts and myofibroblasts are activated, they may continue producing extracellular matrix and growth factors even if overt inflammation decreases, creating a persistent phenotype where tissue remodeling outlasts immune flares.

These issues connect directly to the concept of resolution biology [33]. Resolution is not passive fading of inflammation; it is an active program involving clearance of apoptotic cells and debris, a coordinated shift in macrophage phenotype from pro-inflammatory states toward reparative states, and the generation of specialized pro-resolving mediators that terminate leukocyte recruitment while supporting tissue repair [34]. When resolution is incomplete, repair becomes chronic. The prostate can remain trapped in a wound-healing mode characterized by continued cytokine release, low-level immune cell influx, and progressive fibrosis [34]. This chronic repair state is particularly important in BPH because fibrosis and altered extracellular matrix architecture can amplify obstruction and symptoms, even without dramatic increases in gland volume.

Cytokine-driven stromal–epithelial crosstalk is the mechanism that converts immune signals into durable tissue growth [35]. Inflamed stromal cells produce IL-6, IL-8, TGF-β, and a range of growth factors that promote epithelial proliferation, survival, and remodeling. In parallel, stressed or damaged epithelial cells release cytokines and alarmins that recruit immune cells and activate fibroblasts, effectively broadcasting that repair is needed[36]. The extracellular matrix then acts as both scaffold and signaling platform: as stiffness rises with collagen deposition, mechanotransduction pathways sensitise stromal cells to cytokines and growth factors, lowering the threshold for further activation. This creates a feed-forward triad in which immune cells sustain cytokine supply, stromal cells translate cytokines into fibrosis and growth factor output, and epithelial cells reinforce immune recruitment and remodeling signals[37]. Over time, the result is nodular hyperplasia and periurethral remodeling that contributes to both static obstruction and altered smooth muscle dynamics.

Future progress will come from tools and study designs that capture this complexity rather than isolating single mediators. Spatial multi-omics can identify which cell niches produce specific cytokines and how these patterns evolve with severity and symptoms [38]. Longitudinal cohorts can link cytokine signatures to growth trajectories and treatment response, clarifying which inflammatory phenotypes drive progression. Mechanistic trials can test anti-inflammatory or anti-fibrotic add-ons in carefully selected subsets, improving benefit–risk balance. Finally, systems biology models can integrate feedback loops and thresholds to predict tipping points toward progression, guiding precision strategies that disrupt networks rather than chasing one cytokine at a time.

CONCLUSION

BPH is increasingly understood as an immune-modulated disorder of tissue remodeling rather than a purely hormone-driven hyperplastic process. Chronic inflammation in the transitional zone establishes cytokine networks that recruit and activate immune cells, stimulate stromal and epithelial proliferation, promote angiogenesis, and drive fibrotic ECM remodeling, together amplifying both prostate enlargement and symptom burden. Central axes such as IL-6/STAT3, TNF-α/IL-1β/NF-κB, IL-8 chemotactic signaling, IL-17 amplification loops, and TGF-β–mediated fibrosis cooperate to sustain a chronic wound-repair phenotype. Counter-regulatory mechanisms (IL-10, Tregs, resolution programs) may be present but insufficient to restore homeostasis in progressive disease. Recognizing BPH as a cytokine-network condition reframes clinical strategy: beyond reducing volume or tone, future management may incorporate inflammatory phenotyping, network-based biomarkers, and selective immunomodulatory or anti-fibrotic adjuncts aimed at slowing prostate growth and improving LUTS with precision and safety.

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