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Immune-Metabolic Dysregulation in Benign Prostatic Hyperplasia: Exploring Links with Diabetes and Systemic Inflammation

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ABSTRACT

Benign prostatic hyperplasia (BPH) is a highly prevalent condition in aging men, historically attributed to androgenic stimulation and age-related tissue remodeling. Emerging evidence, however, suggests that metabolic dysfunction and systemic inflammation-particularly those associated with type 2 diabetes (T2DM), obesity, and metabolic syndrome-play influential roles in modifying prostate biology, driving stromal–epithelial proliferation, and promoting lower urinary tract symptoms (LUTS). This review synthesizes current knowledge on immune-metabolic dysregulation in BPH, focusing on how hyperglycemia, insulin resistance, chronic low-grade inflammation, oxidative stress, and adipokine imbalance modulate immune activation and prostate tissue remodeling. We examine mechanistic pathways linking diabetes to prostate enlargement, including altered insulin/IGF-1 signaling, inflammatory macrophage and T-cell infiltration, mitochondrial dysfunction, and redox imbalance. Particular attention is given to how metabolic disease reshapes immune phenotypes, such as promoting Th17 responses, senescence-associated secretory phenotypes (SASP), and pro-fibrotic myofibroblast activation, driving progression from benign hyperplasia to symptomatic obstruction. Finally, we discuss therapeutic implications, highlighting opportunities for metabolic therapies, anti-inflammatory interventions, antioxidant strategies, and microbiome-targeted approaches to modify disease trajectory. Understanding immune–metabolic crosstalk in BPH opens important avenues for precision medicine, especially in populations experiencing rising diabetes prevalence and associated inflammatory burden.

Keywords: Benign prostatic hyperplasia, diabetes, immune-metabolism, systemic inflammation, oxidative stress

INTRODUCTION

Benign prostatic hyperplasia (BPH)-characterized by non-malignant enlargement of the prostate due to stromal and epithelial proliferation-is one of the most common urologic disorders among older men [1]. Although androgenic influence, aging, and genetic predisposition remain well-established factors, these elements alone fail to fully explain inter-individual variability in prostate size, symptom severity, and progression [2]. Growing epidemiological data link metabolic conditions, including type 2 diabetes mellitus (T2DM), obesity, dyslipidemia, and metabolic syndrome, to increased risk of BPH, greater prostate volume, and more severe LUTS [3]. These associations point toward a compelling hypothesis: that BPH is not purely a localized urological disorder but rather reflects systemic immune-metabolic dysregulation [4]. Diabetes and obesity induce chronic low-grade inflammation, promote oxidative stress, alter hormone metabolism, and reshape immune responses, all of which may impact prostate homeostasis [5]. Moreover, hyperglycemia, advanced glycation end-products (AGEs), adipokines, and insulin resistance exert direct biological effects on prostate stromal and epithelial cells.

In this context, BPH may be viewed as a metabolic-inflammatory disease, where systemic abnormalities intersect with local immune activity to accelerate hyperplasia, fibrosis, and urinary dysfunction [6]. Understanding these interactions provides critical insights into pathophysiology and offers new therapeutic opportunities.

2. Epidemiology: Linking Diabetes to BPH and LUTS

Numerous cohort and cross-sectional studies consistently demonstrate a strong epidemiologic association between diabetes and benign prostatic hyperplasia. Men with type 2 diabetes mellitus (T2DM) often present with larger prostate volumes, higher likelihood of moderate-to-severe lower urinary tract symptoms, and faster clinical progression compared with non-diabetic counterparts [7]. Diabetes is also associated with an increased risk of acute urinary retention and a diminished therapeutic response to alpha-adrenergic blockers, suggesting that underlying metabolic and inflammatory abnormalities may alter drug responsiveness at the level of smooth muscle tone or stromal remodeling [8]. Hyperinsulinemia, systemic inflammation, and chronic oxidative stress-core pathophysiologic features of metabolic disease-are consistently linked to these adverse outcomes [9]. Obesity, particularly central or visceral adiposity, further amplifies the risk of BPH and LUTS. Visceral fat functions as an active endocrine organ, producing high levels of inflammatory mediators, including IL-6, TNF-alpha, and C-reactive protein [10]. These biomarkers correlate with prostate enlargement, stromal inflammation, and symptom worsening in epidemiologic datasets [11]. Taken together, population-level evidence strongly supports the concept that metabolic dysfunction is an important and potentially modifiable driver of prostate disease. This growing body of research underscores the need to consider BPH not solely as an age-related urologic disorder but also as a manifestation of systemic metabolic and inflammatory disturbances.

3. Immune–Metabolic Mechanisms Linking Diabetes to BPH

3.1 Chronic systemic inflammation (metaflammation)

Obesity and T2DM give rise to a state of persistent low-grade inflammation often termed metaflammation [12]. This inflammatory state is propelled by infiltration of adipose tissue with macrophages, increased circulating cytokines such as IL-1β, IL-6, and TNF-alpha, and elevated chemokines and acute-phase proteins [13]. Activation of the NLRP3 inflammasome within metabolic tissues further amplifies systemic inflammatory signaling[14]. This circulating inflammatory burden can influence distant organs, including the prostate. Inflammatory mediators readily penetrate the prostatic microvasculature, where they promote stromal cell activation, upregulation of adhesion molecules, and recruitment of additional immune cells[15]. Sustained exposure leads to macrophage and T-cell infiltration, excessive cytokine signaling, enhanced fibroblast activity, and progressive extracellular matrix deposition, all of which contribute to hyperplasia and LUTS progression[16].

3.2 Hyperinsulinemia and IGF-1 axis dysregulation

Insulin resistance drives compensatory hyperinsulinemia, which has direct growth-promoting effects on the prostate because both epithelial and stromal cells express high levels of insulin and IGF-1 receptors [17]. Elevated insulin can stimulate cellular proliferation, reduce apoptosis, and activate key pro-growth pathways including PI3K/Akt/mTOR [18]. Insulin and IGF-1 also support increased production of local growth factors and exert pro-inflammatory effects by enhancing cytokine secretion and promoting fibroblast activation [19]. Dysregulation of the insulin–IGF-1 axis, therefore, links metabolic dysfunction to both proliferative and inflammatory aspects of BPH pathogenesis.

3.3 Hyperglycemia and advanced glycation end-products (AGEs)

Chronic hyperglycemia accelerates the formation of AGEs, which accumulate in the prostate and bind to RAGE receptors expressed by epithelial cells, stromal fibroblasts, and infiltrating immune cells [20]. AGE–RAGE engagement activates NF-κB signaling, increases ROS generation, and triggers further cytokine release, thereby integrating metabolic stress with inflammatory activation [21]. This pathway also promotes fibroblast-to-myofibroblast differentiation, facilitating prostatic fibrosis, which contributes directly to urinary flow obstruction even in the absence of significant increases in glandular size.

3.4 Dysregulated adipokines

Adipose tissue dysfunction in diabetes alters circulating levels of adipokines, which modulate immunity and tissue remodeling [22]. Elevated leptin promotes inflammation, angiogenesis, and cellular proliferation within the prostate. Increased resistin supports macrophage activation and cytokine production. In contrast, adiponectin, normally anti-inflammatory and protective, is reduced in obesity and T2DM, diminishing its ability to counteract fibrotic and inflammatory signaling [23]. The combined effect of high leptin and low adiponectin creates a pro-inflammatory endocrine environment that accelerates prostate remodeling and contributes to the progression of BPH.

4. Immune Cell Dynamics in Metabolically Driven BPH

4.1 Macrophage infiltration and polarization

Metabolic dysfunction alters the immunologic landscape of the prostate, and macrophages are among the earliest responders to hyperglycemia-associated tissue stress [24]. Elevated glucose levels, together with chronic systemic inflammation, enhance monocyte recruitment through increased expression of adhesion molecules and chemokines within the prostatic microvasculature [25]. Once resident in the tissue, macrophages in men with diabetes frequently exhibit a mixed or intermediate polarization state, characterized by simultaneous production of classic

M1 cytokines such as IL-1β and TNF-α alongside M2-associated mediators including TGF-β [26]. This hybrid phenotype has significant pathogenic implications. Pro-inflammatory outputs amplify local immune activation and epithelial stress responses, while TGF-β promotes fibroblast activation, extracellular matrix production, and stromal stiffness[27]. The combined effect is a tissue environment conducive to both proliferative expansion and fibrotic remodeling, two hallmarks of progressive BPH in metabolically compromised individuals.

4.2 T-cell dysregulation

T-cell alterations also play a critical role in linking metabolic disease to prostate enlargement [28]. Diabetes is associated with a shift in circulating and tissue-resident T-cell subsets toward pro-inflammatory phenotypes, with increased Th1 and Th17 cell frequencies and reduced regulatory T-cell (Treg) populations [29]. Elevated levels of IL-17 and IFN-γ in the diabetic prostate promote stromal proliferation, enhance chemokine expression, and sustain macrophage infiltration. The relative deficiency or dysfunction of Tregs compromises local immune tolerance, allowing inflammatory signaling to persist rather than resolve[30]. Persistent T-cell–mediated inflammation contributes not only to glandular growth but also to heightened symptom severity, as cytokine-driven stromal alterations increase tissue density and impair smooth muscle relaxation.

4.3 Stromal cell activation and immunologic remodeling

Prostate stromal fibroblasts are highly sensitive to immune and metabolic cues, and diabetic conditions markedly intensify their activation [31]. Exposure to elevated cytokine levels drives fibroblasts to produce growth factors such as FGFs and TGF-β, large quantities of extracellular matrix proteins, and matrix metalloproteinases that reorganize the stromal architecture. Concurrently, increased reactive oxygen species production by activated fibroblasts reinforces oxidative stress and promotes myofibroblast differentiation[32]. These changes establish a self-amplifying paracrine loop that stimulates epithelial proliferation, accelerates fibrosis, and contributes to glandular enlargement and lower urinary tract obstruction.

5. Therapeutic Implications

5.1 Metabolic control as a strategy for BPH management

Given the strong links between metabolic dysfunction, inflammation, and prostate enlargement, improving metabolic health represents a promising adjunctive approach to BPH management [33]. Enhancing insulin sensitivity, reducing visceral adiposity, and achieving tighter glycemic control can collectively dampen inflammatory and proliferative signaling within the prostate. Lifestyle strategies such as weight loss, increased physical activity, and dietary modification remain foundational, but pharmacologic agents, including metformin and GLP-1 receptor agonists, offer additional benefits [34]. These therapies can reduce systemic inflammatory markers, improve lipid and glucose homeostasis, and diminish activation of pathways such as PI3K/Akt that contribute to stromal and epithelial proliferation. Although not traditionally considered urologic drugs, their indirect effects on prostate biology may meaningfully influence disease trajectory.

5.2 Anti-inflammatory therapies

Targeting inflammation more directly is another therapeutic avenue under exploration. Selective inhibitors of cytokines involved in prostate remodeling, as well as COX-2 inhibitors and other immunomodulatory agents, may reduce leukocyte infiltration and cytokine-driven stromal activation [35]. While preliminary findings are encouraging, robust long-term clinical trials are needed to determine safety, efficacy, and appropriate patient selection.

5.3 Redox-modulating and mitochondrial therapies

Because oxidative stress is a central driver of metabolism-linked BPH, therapies that enhance mitochondrial efficiency or activate Nrf2-dependent antioxidant pathways may help mitigate ROS accumulation [36]. Agents that improve mitochondrial biogenesis, enhance glutathione production, or reduce NADPH oxidase activity hold particular promise in slowing oxidative injury and tissue remodeling.

5.4 Anti-fibrotic interventions

Fibrosis is increasingly recognized as a major contributor to urinary obstruction. Strategies aimed at reducing TGF-β signaling, limiting extracellular matrix deposition, or preventing myofibroblast differentiation could benefit patients with fibrotic-dominant BPH phenotypes [37]. Anti-fibrotic therapies used in other chronic diseases may provide translational models.

5.5 Microbiome-targeted therapies

Emerging evidence suggests that gut dysbiosis and metabolic endotoxemia contribute to systemic inflammation [38]. Probiotics, prebiotics, and dietary interventions designed to lower endotoxin burden and improve microbial composition may reduce inflammatory priming of the prostate, offering a novel adjunct to traditional BPH therapies.

CONCLUSION

BPH is increasingly recognized as a disorder shaped by systemic metabolic and inflammatory pressures, rather than merely age-related hormonal shifts. Diabetes and obesity create a pro-inflammatory, oxidative, and

pro-fibrotic environment that accelerates prostate enlargement and worsens urinary symptoms. Immune-metabolic dysregulation, including macrophage and T-cell activation, insulin and AGE signaling, oxidative stress, and stromal–epithelial crosstalk, forms the mechanistic foundation for this relationship. Understanding these pathways offers critical opportunities to design personalized interventions that address both systemic metabolic health and localized prostate pathology. As diabetes prevalence rises globally, incorporating metabolic assessment and management into BPH care will become increasingly essential for effective long-term disease control.

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