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Gut-Prostate Axis in Benign Prostatic Hyperplasia: Role of Dietary Fiber, Probiotics, and Postbiotics

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ABSTRACT

Benign prostatic hyperplasia (BPH) is a prevalent, age-related condition characterized by non-malignant prostate enlargement and lower urinary tract symptoms (LUTS). Although traditionally linked to hormonal imbalance and inflammation, growing evidence points to the involvement of the gut microbiota and its metabolites in BPH pathophysiology through the emerging concept of the gut-prostate axis. The gut microbiome influences systemic inflammation, immune modulation, metabolic function, and hormonal signaling—all of which are relevant to BPH development and progression. Dietary components such as fiber, probiotics, and postbiotics can modulate the gut microbiome and may serve as novel therapeutic strategies. This review explores the bidirectional interaction between gut health and prostate function, emphasizing the role of microbiota-derived metabolites like short-chain fatty acids (SCFAs) in modulating prostatic inflammation and hormonal regulation. The review further evaluates current evidence on the use of dietary fiber, probiotic strains, and postbiotic compounds in BPH prevention and management, proposing a microbiome-centered approach to urological health. Finally, challenges and future perspectives for clinical translation are discussed.

Keywords: Gut-prostate axis, Benign prostatic hyperplasia, Probiotics, Dietary fiber, Postbiotics

INTRODUCTION

Benign prostatic hyperplasia (BPH) affects millions of men worldwide, with increasing incidence among individuals aged 50 and above [1]. It is characterized by progressive enlargement of the prostate gland, leading to bladder outlet obstruction and lower urinary tract symptoms (LUTS) [1]. These symptoms include urinary urgency, frequency, nocturia, and weak urinary stream, significantly impacting quality of life [2]. Traditional understanding of BPH pathogenesis has centered on androgenic stimulation, particularly the role of dihydrotestosterone (DHT), as well as age-related inflammation and oxidative stress [3]. However, recent studies have identified additional systemic contributors, notably the gut microbiota. The human gut contains trillions of microorganisms that exert widespread influence on immune regulation, inflammation, metabolism, and hormonal balance [4]. The concept of the "gut-prostate axis" suggests that dysbiosis in gut microbial composition may contribute to prostatic inflammation and hyperplasia via immune, metabolic, and neuroendocrine pathways [5]. Diet plays a central role in shaping gut microbiota. Components such as dietary fiber, probiotics, and postbiotics can beneficially modulate microbial communities and their metabolic outputs [6]. These microbiota-targeting strategies have been extensively studied in gastrointestinal, metabolic, and immune disorders but are only recently being explored in the context of BPH [7]. This review aims to synthesize current evidence on the gut-prostate axis, focusing on the role of fiber, probiotics, and postbiotics in BPH prevention and management.

The Gut-Prostate Axis: Mechanistic Overview

The concept of the gut-prostate axis has emerged from a growing understanding of how the gastrointestinal microbiome exerts systemic effects far beyond the intestinal tract [8]. Although anatomically distinct, the gut and prostate communicate through multiple interconnected pathways involving immune signaling, endocrine crosstalk,

metabolic regulation, and microbial metabolites [9]. This bidirectional relationship has significant implications for the development and progression of benign prostatic hyperplasia (BPH).

One of the primary links between gut and prostate health is chronic low-grade systemic inflammation [9]. Dysbiosis, or disruption of the gut microbial balance, leads to a reduction in beneficial commensals such as Lactobacillus and Bifidobacterium, and an overgrowth of pathobionts [10]. This microbial imbalance can compromise the intestinal epithelial barrier, resulting in increased permeability or "leaky gut [11]." A compromised barrier allows translocation of endotoxins such as lipopolysaccharides (LPS) from Gram-negative bacteria into the systemic circulation [12]. Once in the bloodstream, LPS activates toll-like receptor 4 (TLR4) signaling on immune and epithelial cells, inducing the release of pro-inflammatory cytokines such as IL-6, TNF-α, and IL-1β [13]. These cytokines are also elevated in BPH tissues and contribute to local stromal remodeling, glandular proliferation, and resistance to apoptosis [14]. Another mechanistic pathway involves microbial-derived metabolites, particularly short-chain fatty acids (SCFAs), including acetate, propionate, and butyrate [15]. These SCFAs are fermentation products of dietary fiber and play a critical role in maintaining mucosal immunity, regulating T-cell differentiation, and modulating nuclear factor-kappa B (NF-κB) signaling [16]. Butyrate, for instance, is known to inhibit histone deacetylases (HDACs), enhance T-regulatory cell differentiation, and suppress inflammatory cascades [17]. These effects are relevant to BPH, where NF-κB-mediated inflammation is a major driver of disease progression. In addition to immune pathways, the gut microbiota modulates hormonal metabolism, particularly through β-glucuronidase and sulfatase enzymes [18]. These enzymes influence the enterohepatic circulation of sex hormones such as testosterone and estradiol. Dysbiosis may disrupt this balance, resulting in altered systemic levels of dihydrotestosterone (DHT) and estrogen metabolites, both of which contribute to prostatic overgrowth [19]. Furthermore, gut microbiota influence the metabolism of bile acids, insulin sensitivity, and lipid homeostasis—factors that are closely associated with metabolic syndrome, a known risk factor for BPH [20]. Emerging evidence also points to the involvement of the gut nervous system in this axis. The enteric nervous system and its interaction with the autonomic nervous system can influence detrusor muscle activity, bladder function, and LUTS presentation [21]. Microbiota-derived neurotransmitters such as serotonin and gamma-aminobutyric acid (GABA) may influence these neural circuits, potentially impacting prostate-bladder dynamics [22].

Overall, the gut-prostate axis is a multifaceted regulatory network. Dysregulation of this axis contributes to a pro-inflammatory, pro-proliferative, and hormonally imbalanced environment that fosters the progression of BPH. Understanding the mechanistic underpinnings of this interaction opens new avenues for therapeutic interventions targeting the microbiome, including dietary fiber, probiotics, and postbiotics.

Role of Dietary Fiber in BPH Prevention

Dietary fiber represents one of the most accessible and effective nutritional tools for modulating the gut microbiome and systemic inflammation. Defined as non-digestible carbohydrates found in plant-based foods, fiber escapes digestion in the small intestine and is fermented by colonic bacteria, leading to the production of bioactive short-chain fatty acids (SCFAs) [23]. These metabolites, particularly butyrate, propionate, and acetate, are key mediators of the health-promoting effects of fiber and serve as a mechanistic bridge between dietary intake and prostate health [24]. Epidemiological studies have consistently shown that populations with high dietary fiber intake have lower rates of BPH and other chronic inflammatory diseases. For instance, large-scale cohort studies have observed that men consuming high-fiber diets—especially those rich in fruits, vegetables, legumes, and whole grains—experience fewer lower urinary tract symptoms (LUTS) and have lower prostate-specific antigen (PSA) levels [25]. These findings suggest that fiber may exert both preventive and therapeutic effects in BPH. Mechanistically, fiber-derived SCFAs exert multiple actions relevant to prostate inflammation and hyperplasia. Butyrate, in particular, has anti-inflammatory effects through inhibition of NF-κB signaling and suppression of cytokines such as IL-6 and TNF-α [26]. It also promotes regulatory T-cell (Treg) expansion, which is crucial for maintaining immune tolerance and preventing chronic inflammation [26]. Furthermore, SCFAs can modulate epithelial barrier function in the gut, reducing LPS leakage into systemic circulation and thus dampening distant inflammatory responses, including in the prostate [27].

Fiber intake also influences metabolic and hormonal parameters that indirectly affect prostate growth [28]. High-fiber diets are associated with improved insulin sensitivity, reduced circulating insulin-like growth factor 1 (IGF-1), and lower body mass index (BMI) [29]. These changes are significant because hyperinsulinemia and obesity are independent risk factors for BPH and LUTS. Additionally, fiber delays gastric emptying and enhances satiety, contributing to calorie control and weight management—factors that are essential in modulating systemic inflammation and androgen metabolism [30]. In animal models, diets enriched with soluble fiber have been shown to reduce prostate weight, inhibit androgen receptor expression, and decrease epithelial proliferation in testosterone-induced BPH [31]. These findings confirm the biological plausibility of fiber as a modulator of prostatic health through gut-mediated mechanisms.

Despite its promise, the type and amount of fiber consumed are critical determinants of its efficacy. Soluble fibers, such as inulin, pectin, and beta-glucans, are particularly effective in promoting SCFA production, whereas insoluble fibers primarily aid bowel regularity [32]. Personalized dietary interventions based on fiber type, gut microbiota composition, and patient metabolic profile may enhance clinical outcomes. [33] Dietary fiber acts as a pivotal modulator of the gut-prostate axis. Through its influence on microbial composition, metabolite production, immune function, and metabolic balance, fiber-rich diets may offer a non-invasive, scalable, and cost-effective strategy for BPH prevention and management. Future interventional studies should focus on quantifying fiber-mediated microbial and inflammatory biomarkers in men at risk for or suffering from BPH.

Probiotics in Prostatic Health

Probiotics are live microorganisms that confer health benefits when administered in adequate amounts. Specific strains such as Lactobacillus and Bifidobacterium are known to improve gut barrier integrity, reduce LPS translocation, and modulate immune responses [34]. In preclinical studies, probiotic supplementation has been shown to lower inflammatory cytokine levels and inhibit prostatic epithelial proliferation [35].

Some clinical studies have observed improvement in urinary symptoms and inflammatory markers in patients with BPH following probiotic use, although evidence remains preliminary. Probiotics may also indirectly influence hormone metabolism by altering microbial enzymatic activity in the gut, reducing the enterohepatic recycling of androgens and estrogens [36]. In addition, probiotics have shown potential in reducing oxidative stress and improving insulin sensitivity—two important pathophysiological factors in BPH progression [37]. However, the therapeutic effects appear to be strain-specific, and more rigorous clinical trials are needed to identify the most effective formulations.

Emerging Role of Postbiotics

Postbiotics are defined as non-viable bacterial products or metabolic byproducts derived from probiotics that exert biological activity. These include SCFAs, bacteriocins, peptidoglycans, and cell wall fragments [38]. Postbiotics provide a safe and stable alternative to live probiotics, especially for immunocompromised individuals [38]. Butyrate, the most studied postbiotic, exhibits strong anti-inflammatory and epigenetic effects relevant to BPH. It enhances T regulatory cell function, inhibits pro-inflammatory cytokine expression, and improves epithelial barrier function [39]. In murine models, butyrate supplementation led to reduced prostate enlargement and lower expression of IL-6 and TNF-α [40]. Other postbiotics such as indole derivatives and polyamines have been shown to suppress NF-κB and MAPK pathways, modulate macrophage polarization, and reduce fibrotic remodeling [40]. These effects suggest that postbiotics may provide a novel therapeutic approach in microbiome-centered BPH management.

Future Directions

The gut-prostate axis represents a novel and promising area of research in urology. Modulating the gut microbiota through dietary fiber, probiotics, and postbiotics holds potential not only for reducing prostatic inflammation but also for altering the metabolic and hormonal milieu associated with BPH. Integrating microbiome-targeted therapies with conventional pharmacological treatments may enhance therapeutic outcomes and offer personalized management strategies. However, challenges remain in translating preclinical findings to clinical practice. Key research priorities include:

- Identifying microbial signatures predictive of BPH risk and treatment response
- Conducting randomized controlled trials with standardized probiotic and postbiotic formulations
- Developing fiber-based interventions tailored to individual microbiota profiles
- Investigating long-term safety and microbiome stability

CONCLUSION

Nutritional modulation of the gut microbiome is an emerging frontier in BPH prevention and therapy. Targeting the gut-prostate axis through evidence-based dietary strategies may offer a safe, holistic, and innovative adjunct to traditional urological care.

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